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National Toxicology Program
U.S. Department of Health and Human Services

NTP Technical Report on the Toxicology and Carcinogenesis Studies in Sprague Dawley (Hsd:Sprague Dawley® SD®) Rats Exposed to Whole-body Radio Frequency Radiation at a Frequency (900 MHz) and Modulations (GSM and CDMA) Used by Cell Phones

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NTP Technical Report on the Toxicology and Carcinogenesis Studies in Sprague Dawley (Hsd:Sprague Dawley® SD®) Rats Exposed to Whole-body Radio Frequency Radiation at a Frequency (900 Mhz) and Modulations (GSM and CDMA) Used by Cell Phones

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Foreword

The National Toxicology Program (NTP) is an interagency program within the Public Health Service (PHS) of the Department of Health and Human Services (HHS) and is headquartered at the National Institute of Environmental Health Sciences of the National Institutes of Health (NIEHS/NIH). Three agencies contribute resources to the program: NIEHS/NIH, the National Institute for Occupational Safety and Health of the Centers for Disease Control and Prevention (NIOSH/CDC), and the National Center for Toxicological Research of the Food and Drug Administration (NCTR/FDA). Established in 1978, NTP is charged with coordinating toxicological testing activities, strengthening the science base in toxicology, developing and validating improved testing methods, and providing information about potentially toxic substances to health regulatory and research agencies, scientific and medical communities, and the public.

The Technical Report series began in 1976 with carcinogenesis studies conducted by the National Cancer Institute. In 1981, this bioassay program was transferred to NTP. The studies described in the Technical Report series are designed and conducted to characterize and evaluate the toxicologic potential, including carcinogenic activity, of selected substances in laboratory animals (usually two species, rats and mice). Substances selected for NTP toxicity and carcinogenicity studies are chosen primarily on the basis of human exposure, level of production, and chemical structure. The interpretive conclusions presented in NTP Technical Reports are based only on the results of these NTP studies. Extrapolation of these results to other species, including characterization of hazards and risks to humans, requires analyses beyond the intent of these reports. Selection per se is not an indicator of a substance's carcinogenic potential.

NTP conducts its studies in compliance with its laboratory health and safety guidelines and FDA Good Laboratory Practice Regulations and must meet or exceed all applicable federal, state, and local health and safety regulations. Animal care and use are in accordance with the Public Health Service Policy on Humane Care and Use of Animals. Studies are subjected to retrospective quality assurance audits before being presented for public review.

The NTP Technical Reports are available free of charge on the NTP website and cataloged in PubMed, a free resource developed and maintained by the National Library of Medicine (part of the National Institutes of Health). Data for these studies are included in NTP's Chemical Effects in Biological Systems database.

For questions about the reports and studies, please email NTP or call 984-287-3211.

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Explanation of Levels of Evidence of Carcinogenic Activity

The National Toxicology Program describes the results of individual experiments on a test agent and notes the strength of the evidence for conclusions regarding each study. Negative results, in which the study animals do not have a greater incidence of neoplasia than control animals, do not necessarily mean that a test agent is not a carcinogen, inasmuch as the experiments are conducted under a limited set of conditions. Positive results demonstrate that a test agent is carcinogenic for laboratory animals under the conditions of the study and indicate that exposure to the test agent has the potential for hazard to humans. Other organizations, such as the International Agency for Research on Cancer, assign a strength of evidence for conclusions based on an examination of all available evidence, including animal studies such as those conducted by NTP, epidemiologic studies, and estimates of exposure. Thus, the actual determination of risk to humans from test agents found to be carcinogenic in laboratory animals requires a wider analysis that extends beyond the purview of these studies.

Five categories of evidence of carcinogenic activity are used in the Technical Report series to summarize the strength of evidence observed in each experiment: two categories for positive results **(clear evidence and some evidence)**; one category for uncertain findings **(equivocal evidence)**; one category for no observable effects **(no evidence)**; and one category for experiments that cannot be evaluated because of major flaws **(inadequate study)**. These categories of interpretative conclusions were first adopted in June 1983 and then revised on March 1986 for use in the Technical Report series to incorporate more specifically the concept of actual weight of evidence of carcinogenic activity. For each separate experiment (male rats, female rats, male mice, female mice), one of the following five categories is selected to describe the findings. These categories refer to the strength of the experimental evidence and not to potency or mechanism.

- **Clear evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a dose-related (i) increase of malignant neoplasms, (ii) increase of a combination of malignant and benign neoplasms, or (iii) marked increase of benign neoplasms if there is an indication from this or other studies of the ability of such tumors to progress to malignancy.
- **Some evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a test agent-related increased incidence of neoplasms (malignant, benign, or combined) in which the strength of the response is less than that required for clear evidence.
- **Equivocal evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing a marginal increase of neoplasms that may be test agent related.
- **No evidence** of carcinogenic activity is demonstrated by studies that are interpreted as showing no test agent-related increases in malignant or benign neoplasms
- **Inadequate study** of carcinogenic activity is demonstrated by studies that, because of major qualitative or quantitative limitations, cannot be interpreted as valid for showing either the presence or absence of carcinogenic activity.

For studies showing multiple test agent-related neoplastic effects that if considered individually would be assigned to different levels of evidence categories, the following convention has been

adopted to convey completely the study results. In a study with clear evidence of carcinogenic activity at some tissue sites, other responses that alone might be deemed some evidence are indicated as “were also related” to test agent exposure. In studies with clear or some evidence of carcinogenic activity, other responses that alone might be termed equivocal evidence are indicated as “may have been” related to test agent exposure.

When a conclusion statement for a particular experiment is selected, consideration must be given to key factors that would extend the actual boundary of an individual category of evidence. Such consideration should allow for incorporation of scientific experience and current understanding of long-term carcinogenesis studies in laboratory animals, especially for those evaluations that may be on the borderline between two adjacent levels. These considerations should include:

- adequacy of the experimental design and conduct;
- occurrence of common versus uncommon neoplasia;
- progression (or lack thereof) from benign to malignant neoplasia as well as from preneoplastic to neoplastic lesions;
- some benign neoplasms have the capacity to regress but others (of the same morphologic type) progress. At present, it is impossible to identify the difference. Therefore, where progression is known to be a possibility, the most prudent course is to assume that benign neoplasms of those types have the potential to become malignant;
- combining benign and malignant tumor incidence known or thought to represent stages of progression in the same organ or tissue;
- latency in tumor induction;
- multiplicity in site-specific neoplasia;
- metastases;
- supporting information from proliferative lesions (hyperplasia) in the same site of neoplasia or other experiments (same lesion in another sex or species);
- presence or absence of dose relationships;
- statistical significance of the observed tumor increase;
- concurrent control tumor incidence as well as the historical control rate and variability for a specific neoplasm;
- survival-adjusted analyses and false positive or false negative concerns;
- structure-activity correlations; and
- in some cases, genetic toxicology.

Peer Review

The members of the Peer Review Panel who evaluated the draft *NTP Technical Report on the Toxicology and Carcinogenesis Studies in Sprague Dawley (Hsd:Sprague Dawley® SD®) Rats Exposed to Whole-body Radio Frequency Radiation at a Frequency (900 Mhz) and Modulations (GSM and CDMA) Used by Cell Phones* on March 26–28, 2018, are listed below. Panel members served as independent scientists, not as representatives of any institution, company, or governmental agency. In this capacity, reviewers had five major responsibilities in reviewing the NTP studies:

- to ascertain that all relevant literature data have been adequately cited and interpreted,
- to determine if the design and conditions of the NTP studies were appropriate,
- to ensure that the Technical Report presents the experimental results and conclusions fully and clearly,
- to judge the significance of the experimental results by scientific criteria, and
- to assess the evaluation of the evidence of carcinogenic activity and other observed toxic responses.

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Abstract

The predominant source of human exposure to radio frequency radiation (RFR) occurs through usage of cellular phone handsets. The Food and Drug Administration nominated cell phone RFR emission for toxicology and carcinogenicity testing in 1999. At that time, animal experiments were deemed crucial because meaningful human exposure health data from epidemiological studies were not available. Male and female Hsd:Sprague Dawley® SD® rats were exposed to time-averaged whole-body specific absorption rates of Global System for Mobile Communications (GSM)- or Code Division Multiple Access (CDMA)-modulated cell phone RFR at 900 MHz in utero, during lactation, and after weaning for 28 days or 2 years. Genetic toxicology studies were conducted in rat peripheral blood erythrocytes and leukocytes, brain cells, and liver cells.

GSM

Twenty-eight-day Study

Beginning on gestation day (GD) 6, groups of 20 time-mated F_0 female rats were housed in specially designed reverberation chambers and received whole-body exposures to GSM-modulated cell phone RFR at power levels of 0 (sham control), 3, 6 or 9 W/kg for 5 to 7 days per week, continuing throughout gestation and lactation. Exposure was up to 18 hours and 20 minutes per day, 5 or 7 days per week, with continuous cycling of 10 minutes on and 10 minutes off during the exposure periods. The sham control animals were housed in reverberation chambers identical to those used for exposed groups but were not exposed to cell phone RFR; a shared group of unexposed rats of each sex served as sham controls for both cell phone RFR modulations. At weaning, 10 males and 10 females per group were selected across ten litters for continuation. Weaning occurred on the day the last litter reached postnatal day (PND) 21, marking the beginning of the 28-day study. Male and female F_1 offspring continued to receive whole-body exposures to GSM-modulated cell phone RFR at the same power levels and under the same exposure paradigm, 5 to 7 days per week for up to 28 days. Prior to exposures, 10 F_0 females per group and four male and four female F_1 litters per group had temperature microchips implanted subcutaneously to monitor individual animal temperatures.

In F_0 females, there were no exposure-related effects on survival or littering rates. There were significantly decreased maternal body weight gains in the 9 W/kg group during gestation (GD 6 through 21). During lactation, there were significantly decreased mean body weights and mean body weight gains at most time points. Mean body temperatures in the 9 W/kg group were significantly greater than those of the sham controls throughout most gestation and lactation. There were also sporadic increased mean body temperatures in the 3 and 6 W/kg groups.

In F_1 offspring, there were no exposure-related effects on total and live litter size during lactation although there was a significantly increased number of dead pups per litter and decreased survival ratio in the 9 W/kg group from PND 1 to 4. There were also significant decreases in body weights of males and females exposed to 9 W/kg during lactation (PND 1 through 21). All offspring survived to the end of the study and body weights of 9 W/kg males were lower than those of the sham controls throughout the study. Mean body temperatures were generally similar between the exposed groups and the sham controls. There were no exposure-related effects on organ weights in either sex. There were increased incidences of chronic progressive nephropathy in the kidney of exposed female groups, but the incidences were not significant, and the severity was minimal in all cases.

Two-year Study

Beginning on GD 5, groups of 56 time-mated F_0 female rats were housed in specially designed reverberation chambers and received whole-body exposures to GSM-modulated cell phone RFR at power levels of 0 (sham control), 1.5, 3, or 6 W/kg for 7 days per week, continuing throughout gestation and lactation. Exposure was up to 18 hours and 20 minutes per day with continuous cycling of 10 minutes on and 10 minutes off during the exposure periods. There were seven exposure groups per sex, including a shared sham control and three exposure groups for each modulation. At weaning, three males and three females per litter from 35 litters were randomly selected per exposure group for continuation. Weaning occurred on the day the last litter reached PND 21, marking the beginning of the 2-year studies. Groups of 105 male and 105 female F_1 offspring continued to receive whole-body exposures to GSM-modulated cell phone RFR at the same power levels and under the same exposure paradigm, 7 days per week for up to 104 weeks. After 14 weeks of exposure, 10 rats per group were randomly selected for interim histopathologic evaluation and five were designated for genetic toxicity evaluation.

In F_0 females, there no exposure-related effects on pregnancy status, maternal survival, or the percentage of animals that littered. During gestation, mean body weight gains of 6 W/kg females were significantly lower than those of the sham controls from GD 15 through 18 and during the overall gestation period (GD 6 through 21). During lactation, the mean body weights of 3 and 6 W/kg females were significantly lower than those of the sham controls for the period of PND 4 through 21.

In F_1 offspring, there was no effect on litter size, pup mortality or survival ratio. During lactation, mean pup weights were significantly lower at most timepoints in the 3 W/kg groups and at all timepoints in the 6 W/kg groups. At the end of 2 years, survival of all exposed male groups was significantly greater than that of the sham control group due to the effect of chronic progressive nephropathy in the kidney of sham control males. Survival of exposed female groups was similar to that of the sham controls. The mean body weights of all exposed males and females were similar to those of the sham control groups. There were no exposure-related clinical observations.

At the 14-week interim evaluation, there were no changes in clinical pathology parameters or organ weights that were considered to be related to exposure. There were no GSM exposure-related effects on reproductive organ weights or sperm parameters in males. The estrous cycle in females was not evaluated due to poor slide quality. In the heart, there were increased incidences of right ventricle cardiomyopathy and cardiomyopathy (all sites) in the 3 and 6 W/kg groups. Only the incidence of cardiomyopathy (all sites) in the 3 W/kg males was significantly greater than that of the sham controls.

In the heart at the end of the 2-year studies, malignant schwannoma was observed in all exposed male groups and the 3 W/kg female group, but none occurred in the sham controls. Endocardial Schwann cell hyperplasia also occurred in a single 1.5 W/kg male and two 6 W/kg males. There were also significantly increased incidences of right ventricle cardiomyopathy in 3 and 6 W/kg males and females.

In the brain of males, there were increased incidences of malignant glioma and glial cell hyperplasia in all exposed groups, but none in the sham controls. There was also increased incidences of benign or malignant granular cell tumors in all exposed groups.

There were significantly increased incidences of benign pheochromocytoma and benign, malignant, or complex pheochromocytoma (combined) of the adrenal medulla in males exposed to 1.5 or 3 W/kg. In the adrenal medulla of females exposed to 6 W/kg, there were significantly increased incidences of hyperplasia.

In the prostate gland of male rats, there were increased incidences of adenoma or adenoma or carcinoma (combined) in 3 W/kg males and epithelium hyperplasia in all exposed male groups. In the pituitary gland (pars distalis), there were increased incidences of adenoma in all exposed male groups. There were also increased incidences of adenoma or carcinoma (combined) of the pancreatic islets in all exposed groups of male rats, but only the incidence in the 1.5 W/kg group was significant.

In female rats, there were significantly increased incidences of C-cell hyperplasia of the thyroid gland in all exposed groups, and significantly increased incidences of hyperplasia of the adrenal cortex in the 3 and 6 W/kg groups.

CDMA

Twenty-eight-day Study

Beginning on gestation day (GD) 6, groups of 20 time-mated F_0 female rats were housed in specially designed reverberation chambers and received whole-body exposures to CDMA-modulated cell phone RFR at power levels of 0 (sham control), 3, 6 or 9 W/kg for 5 to 7 days per week, continuing throughout gestation and lactation. Exposure was up to 18 hours and 20 minutes per day, 5 or 7 days per week, with continuous cycling of 10 minutes on and 10 minutes off during the exposure periods. The sham control animals were housed in reverberation chambers identical to those used for exposed groups but were not exposed to cell phone RFR; a shared group of unexposed rats of each sex served as sham controls for both cell phone RFR modulations. At weaning, 10 males and 10 females per group were selected across ten litters for continuation. Weaning occurred on the day the last litter reached postnatal day (PND) 21, marking the beginning of the 28-day study. Male and female F_1 offspring continued to receive whole-body exposures to CDMA-modulated cell phone RFR at the same power levels and under the same exposure paradigm, 5 to 7 days per week for up to 28 days. Prior to exposures, 10 F_0 females per group and four male and four female F_1 litters per group had temperature microchips implanted subcutaneously to monitor individual animal temperatures.

In F_0 females, there were no exposure-related effects on survival or littering rates. There were significantly decreased mean body weight gains in the 9 W/kg group from GD 15 through 18 and for the gestation period as a whole (GD 6 through 21). During lactation in the 9 W/kg group, there were significantly decreased mean body weights on PNDs 7 through 21 and a significant decrease in mean body weight gain over the whole period (PND 1 through 21). Mean body temperatures during gestation and lactation were significantly increased when compared to the sham controls at several time points in the 9 W/kg group and sporadically in the 6 W/kg group.

In F_1 offspring, there were no exposure-related effects on total and live litter size during lactation although there was a slightly greater number of dead pups in exposed groups from PND 1 to 4 and in the 6 and 9 W/kg groups from PND 4 to 21. There were significantly decreased mean body weights in 6 and 9 W/kg males and 9 W/kg females during lactation. All offspring survived to the end of the study. Only the mean body weights of 9 W/kg males were significantly lower than those of the sham controls throughout the study. Mean body temperatures in exposed groups were similar to those of the sham controls throughout the study. There were no exposure-related

effects on organ weights in either sex. There was a significantly increased incidence of chronic progressive nephropathy in the kidney of 6 W/kg females, but the severity was minimal in all cases.

Two-year Study

Beginning on GD 5, groups of 56 time-mated F_0 female rats were housed in specially designed reverberation chambers and received whole-body exposures to CDMA-modulated cell phone RFR at power levels of 0 (sham control), 1.5, 3, or 6 W/kg for 7 days per week, continuing throughout gestation and lactation. Exposure was up to 18 hours and 20 minutes per day with continuous cycling of 10 minutes on and 10 minutes off during the exposure periods. There were seven exposure groups per sex, including a shared sham control and three exposure groups for each modulation. At weaning, three males and three females per litter from 35 litters were randomly selected per exposure group for continuation. Weaning occurred on the day the last litter reached PND 21, marking the beginning of the 2-year studies. Groups of 105 male and 105 female F_1 offspring continued to receive whole-body exposures to CDMA-modulated cell phone RFR at the same power levels and under the same exposure paradigm, 7 days per week for up to 104 weeks. After 14 weeks of exposure, 10 rats per group were randomly selected for interim histopathologic evaluation and five were designated for genetic toxicity evaluation.

In F_0 females, there no exposure-related effects on pregnancy status, maternal survival, or the percentage of animals that littered. During gestation, the mean body weights and mean body weight gains of exposed groups were similar to those of the sham controls. During lactation, mean body weights were significantly lower than those of the sham controls at most time points in the 6 W/kg group, at several time points in the 1.5 and 3 W/kg groups, and the mean body weight gains for the period as a whole (PND 1 through 21) were significantly lower in the 3 and 6 W/kg groups.

In F_1 offspring, there were no effects on litter size on PND 1. On PND 7 through 21, there were significant decreases in live litter size in the 6 W/kg group when compared to the sham controls. Throughout lactation, the male and female pup mean body weights in the 6 W/kg groups were significantly lower than those of the sham controls. At the end of 2 years, survival in all exposed male group was greater than that of the sham control group due to the effects of chronic progressive nephropathy in the kidney of the sham control males. In females, there was a small, but statistically significant increase in survival in the 6 W/kg group. Although there were some differences in mean body weights in exposed male groups, at the end of the study, the mean body weights of exposed male and female groups were similar to those of the sham controls. There were no exposure-related clinical observations.

At the 14-week interim evaluation, there were changes in clinical pathology or organ weights that were considered to be related to exposure. There were no CDMA exposure-related effects on reproductive organ weights or sperm parameters in males. The estrous cycle in females was not evaluated due to poor slide quality. In the heart, there were increased incidences of right ventricle cardiomyopathy in all exposed male groups, but the severities were minimal in all cases. There were marginally increased incidences of cardiomyopathy (all sites) in the 3 and 6 W/kg females.

At the end of the 2-year study, malignant schwannoma of the heart occurred in all exposed male groups and the incidence in the 6 W/kg group was significantly increased; this neoplasm did not occur in the sham controls. There was also an increased incidence of endocardial Schwann cell

hyperplasia in 6 W/kg males. In females, malignant schwannoma occurred in two animals each in the 1.5 and 6 W/kg groups.

In the brain, malignant glioma occurred in 6 W/kg males and 1.5 W/kg females; none occurred in the sham control groups. Glial cell hyperplasia also occurred in 1.5 and 6 W/kg males and 3 and 6 W/kg females.

In males, there was a significantly increased incidence of pituitary gland (pars distalis) adenoma in the 3 W/kg group, and increased incidences of hepatocellular adenoma or carcinoma (combined) in the liver of all exposed groups.

In the adrenal medulla of females, there were increased incidences of benign, malignant, or complex pheochromocytoma (combined) in all exposed groups, but only the incidence in the 1.5 W/kg group was significantly increased compared to the sham controls.

In the prostate gland of male rats, there were increased incidences of epithelial hyperplasia in all exposed groups, but only the incidence in the 6 W/kg group was significantly increased compared to the sham control group.

Genetic Toxicology

Comet Assay

As part of the 14-week interim evaluation, samples of frontal cortex, hippocampus, cerebellum, liver, and blood leukocytes were evaluated for DNA damage using the comet assay (two sexes, two cell phone RFR modulations, and five tissues per animal). Samples of peripheral blood from these same animals were also evaluated for chromosome damage in the micronucleus assay. Results in the comet assay are based on the 100-cell scoring approach that was standard at the time of the studies; data obtained using a second, 150-cell scoring approach recommended in a recently adopted international guideline for the in vivo comet assay, are noted for the few instances where results differed between the two methods. A significant increase in DNA damage (% tail DNA) was observed in hippocampus cells of male rats exposed to the CDMA modulation. Although the levels of DNA damage in hippocampus cells were also increased in an exposure-related fashion using the 150-cell scoring approach, the increases were not statistically significant. An exposure-related increase in DNA damage seen in the cells of the frontal cortex of male rats exposed to the CDMA modulation was judged to be equivocal based on a significant trend test. Although results from scoring 100 cells were negative for male rat blood leukocytes exposed to either CDMA or GSM modulations, the results (both CDMA and GSM) were judged to be equivocal when evaluated using the 150-cell scoring method. No statistically significant increases in DNA damage were observed in any of the female rat samples scored with the 100-cell approach; with the 150-cell approach, results in peripheral blood leukocytes of female rats (CDMA) were judged to be equivocal.

Micronucleus Assay

No significant increases in micronucleated red blood cells or changes in the percentage of immature erythrocytes among total erythrocytes were observed in peripheral blood of rats of either sex exposed to either modulation of cell phone RFR.

Conclusions

GSM-modulated RFR

Under the conditions of this 2-year whole-body exposure study, there was *clear evidence of carcinogenic activity* (see Explanation of Levels of Evidence of Carcinogenic Activity; see a

summary of the Peer Review Panel comments and the public discussion on this Technical Report in Appendix L) of GSM-modulated cell phone RFR at 900 MHz in male Hsd:Sprague Dawley® SD® rats based on the incidences of malignant schwannoma of the heart. The incidences of malignant glioma of the brain and benign, malignant, or complex pheochromocytoma (combined) of the adrenal medulla were also related to RFR exposure. The incidences of benign or malignant granular cell tumors of the brain, adenoma or carcinoma (combined) of the prostate gland, adenoma of the pars distalis of the pituitary gland, and pancreatic islet cell adenoma or carcinoma (combined) may have been related to RFR exposure. There was *equivocal evidence of carcinogenic activity* of GSM-modulated cell phone RFR at 900 MHz in female Hsd:Sprague Dawley® SD® rats based on the incidences of schwannomas of the heart.

Increases in non-neoplastic lesions of the heart, brain, and prostate gland in male rats, and of the heart, thyroid gland, and adrenal gland in female rats occurred with exposures to GSM-modulated RFR at 900 MHz.

CDMA-modulated RFR

Under the conditions of this 2-year whole-body exposure study, there was *clear evidence of carcinogenic activity* (see Explanation of Levels of Evidence of Carcinogenic Activity; see a summary of the Peer Review Panel comments and the public discussion on this Technical Report in Appendix L) of CDMA-modulated cell phone RFR at 900 MHz in male Hsd:Sprague Dawley® SD® rats based on the incidences of malignant schwannoma of the heart. The incidences of malignant glioma of the brain were also related to RFR exposure. The incidences of adenoma of the pars distalis of the pituitary gland and adenoma or carcinoma (combined) of the liver may have been related to RFR exposure. There was *equivocal evidence of carcinogenic activity* of CDMA-modulated cell phone RFR at 900 MHz in female Hsd:Sprague Dawley® SD® rats based on the incidences of malignant schwannoma of the heart, malignant glioma of the brain, and benign, malignant, or complex pheochromocytoma (combined) of the adrenal medulla.

Increases in non-neoplastic lesions of the heart, brain, and prostate gland in male rats, and of the brain in female rats occurred with exposures to CDMA-modulated RFR at 900 MHz.

Synonyms: cell phone radio frequency radiation; mobile phone radio frequency radiation

Summary of the Two-year Carcinogenesis and Genetic Toxicology Studies of GSM- and CDMA-modulated Cell Phone RFR Exposure in Rats

	GSM-modulated Cell Phone RFR Male Rats	GSM-modulated Cell Phone RFR Female Rats	CDMA-modulated Cell Phone RFR Male Rats	CDMA-modulated Cell Phone RFR Female Rats
Whole-body GSM- or CDMA-modulated cell phone RFR exposure	0, 1.5, 3, or 6 W/kg	0, 1.5, 3, or 6 W/kg	0, 1.5, 3, or 6 W/kg	0, 1.5, 3, or 6 W/kg
Survival rates	25/90, 45/90, 50/90, 60/90	48/90, 53/90, 48/90, 57/90	25/90, 43/90, 56/90, 43/90	48/90, 46/90, 50/90, 61/90
Body weights	Exposed groups similar to the sham control group	Exposed groups similar to the sham control group	Exposed groups similar to the sham control group	Exposed groups similar to the sham control group
Non-neoplastic effects	Heart: ventricle right, cardiomyopathy (54/90, 62/90, 72/90, 74/90); Schwann cell hyperplasia (0/90, 1/90, 0/90, 2/90) Brain: glial cell, hyperplasia (0/90, 2/90, 3/90, 1/90) Prostate gland: epithelium, hyperplasia (5/90, 13/90, 11/90, 11/90)	Heart: ventricle right, cardiomyopathy (4/90, 9/90, 14/90, 15/90) Thyroid gland: C-cell, hyperplasia (28/90, 49/88, 45/90, 43/88) Adrenal medulla: hyperplasia (13/86, 19/90, 14/90, 25/86)	Heart: Schwann cell hyperplasia (0/90, 0/90, 0/90, 3/90) Brain: glial cell, hyperplasia (0/90, 2/90, 0/90, 2/90) Prostate gland: epithelium, hyperplasia (5/90, 11/90, 9/90, 15/85)	Brain: glial cell, hyperplasia (0/90, 0/90, 1/90, 1/90)
Neoplastic effects	Heart:schwannoma malignant (0/90, 2/90, 1/90, 5/90) Brain: glioma malignant (0/90, 3/90, 3/90, 2/90) Adrenal medulla: benign, malignant, or complex pheochromocytoma (11/88, 24/90, 28/89, 14/87)	None	Heart: schwannoma malignant (0/90, 2/90, 3/90, 6/90) Brain: glioma malignant (0/90, 0/90, 0/90, 3/90)	None

	GSM-modulated Cell Phone RFR Male Rats	GSM-modulated Cell Phone RFR Female Rats	CDMA-modulated Cell Phone RFR Male Rats	CDMA-modulated Cell Phone RFR Female Rats
Equivocal findings	Brain: meninges, granular cell tumor benign or malignant (1/90, 3/90, 4/90, 3/90) Prostate gland: adenoma or carcinoma (2/90, 2/90, 7/90, 3/90) Pituitary gland: pars distalis, adenoma (17/89, 28/90, 26/90, 26/90) Islets, pancreatic: adenoma or carcinoma (13/90, 27/89, 19/86, 16/85)	Heart: schwannoma malignant (0/90, 0/90, 2/90, 0/90)	Pituitary gland: pars distalis, adenoma (17/89, 25/90, 34/90, 13/90) Liver: hepatocellular adenoma or carcinoma (combined) (0/90, 2/90, 4/89, 1/88)	Heart: schwannoma malignant (0/90, 2/90, 0/90, 2/90) Brain: glioma malignant (0/90, 3/90, 0/90, 0/90) Adrenal medulla: benign, malignant, or complex pheochromocytoma (1/86, 9/89, 5/87, 4/88)
Level of evidence of carcinogenic activity	Clear evidence	Equivocal evidence	Clear evidence	Equivocal evidence
Genetic toxicology				
DNA damage:				
GSM-modulated	Negative in frontal cortex, hippocampus, cerebellum, liver, and leukocytes (males and females)			
CDMA-modulated	Positive in hippocampus (males); equivocal in frontal cortex (males); negative in hippocampus and frontal cortex (females), cerebellum, liver, and leukocytes (males and females)			
Micronucleated erythrocytes in peripheral blood in vivo:				
GSM-modulated	Negative in males and females			
CDMA-modulated	Negative in males and females			

Introduction

All consumer cell phone devices function through the transmission of radio waves on a cellular network. The cellular network itself is composed of a collection of individual “cells” that include a fixed-location transceiver (a device that transmits and receives radio signals), also referred to as a cell tower. The collection of adjacent smaller “cells” in the cellular network enables cell phones and towers to use low-power transmitters, thereby allowing for the same frequencies to be reused in non-adjacent cells without interference. Together the individual “cells” comprise the cellular network that provides coverage over a large geographical area. In the United States two major nationwide cellular technologies in use are CDMA (Code Division Multiple Access) and GSM (Global System for Mobile Communications). While technologies are rapidly evolving to meet consumers’ increased demand for better coverage, increased call quality, faster data transfer rates, and increased accessibility, in the context of this report, the terms CDMA and GSM group together multiple, sometimes successive, technologies that are implemented by the service providers that maintain the service networks. In the United States, Sprint® and Verizon® networks use CDMA; AT&T® and T-Mobile® use GSM.

For both the GSM and CDMA technologies, transmissions occur at specific radio frequencies, which are allocated and regulated by the Federal Communications Commission (FCC). While the transmission of radio signals (radiofrequency radiation) can occur at the same frequencies for both technologies, they differ in the method by which information is incorporated and transmitted within frequency bands. In telecommunications, these are referred to as signal modulations. Because this process differs for CDMA and GSM, cell phones are not interchangeable between the two network technologies and will only function on one or the other.

The constantly evolving cellular technologies are commonly referred to by their successive generations (G). The first generation (1G) devices were analogue phones, as opposed to the digital phones of today. Digital voice systems of the second generation (2G) replaced the analogue system of 1G. At the time that these studies were being designed, 2G technology was the primary technology in use and 3G technologies were emerging. Therefore, the current studies were conducted using modulated signals that replicated the 2G and 3G technology in use at the time. Over the course of the studies, however, more advanced 4G technologies were developed. Currently, all of these technologies (2G, 3G, and 4G) are still actively in use for mobile communication applications. 2G and 3G are still the basis for voice calling applications, while 3G and 4G technologies were primarily developed to offer faster access to the internet. Some of the 3G technology is based on 2G technology. While 2G technology is being phased out in the United States, this technology will remain in use in other places throughout the world. More advanced and efficient technologies that are currently in development and not yet deployed, termed 5G, will utilize higher frequencies than existing technologies.

Radio Frequency Radiation (RFR) Measurement and Applications

RFR is a form of nonionizing electromagnetic energy that consists of propagating electromagnetic waves of oscillating electric (E-) and magnetic (H-) fields that move together at the speed of light. RF waves are characterized by their wavelength (the distance covered by one complete cycle of the electromagnetic wave) and their frequency (the number of electromagnetic

waves passing a given point in 1 second). The frequency of an RF signal is expressed in terms of Hertz (Hz), where one Hz is equivalent to one cycle per second. RF radiation refers to the region of the electromagnetic spectrum from 3 kilohertz (3 kHz) to 300 gigahertz (300 GHz) (Figure 1). As opposed to ionizing radiation, which contains enough energy when passing through matter to break chemical bonds or remove an electron from an atom or molecule to produce charged ions, nonionizing radiation has at most sufficient energy for excitation of an electron to a higher energy state.

The intensity of an RF field can be expressed by its electric and magnetic components and is measured in volts per meter (V/m) for electric fields and amperes per meter (A/m) for magnetic fields. Another measure of RFR is the power density, which is defined as the power per unit area and is expressed in watts per square meter (W/m2). The quantity used to describe the amount of RFR energy absorbed by the body is referred to as the specific absorption rate (SAR), which is expressed in watts per kilogram (W/kg). SAR is a function of the geometry and the dielectric loss properties of biological tissues absorbing the energy (which results from the interaction of electro-magnetic radiation with constituents at the cellular and molecular level), the square of the strength of the induced E-field, and the mass density of the exposed tissue. The SAR value is derived by averaging the absorbed energy over a specific volume (typically 1 gram, 10 grams, or the whole body for regulatory purposes).

Different applications utilize different frequency bands within the RF portion of the electromagnetic spectrum. RF frequencies for radio and television are in the 145 kHz to 850 MHz range. Wireless communications and networking typically utilize frequencies between 800 MHz and 6 GHz. Cell phone networks that are currently in use (2G, 3G, and 4G) utilize frequencies in the range of 600 MHz to 5.7 GHz. In the United States, wireless telecommunications networks and devices operate in bands at frequencies of nominally 800 MHz, 850 MHz, or 1,900 MHz for 2G; 850 MHz, 1,700 MHz, 1,900 MHz, or 2,100 MHz for 3G; and 600 MHz, 700 MHz, 800 MHz, 850 MHz, 1,700 MHz, 1,900 MHz, 2,100 MHz, 2,300 MHz, 2,500 MHz, 5,200 MHz, or 5,700 MHz for 4G. The next generation, i.e., the 5th generation of wireless communications, will also utilize the RFR spectrum above 6 GHz. Other terms are also used in the literature for part of the RFR spectrum, e.g., microwaves for frequencies above 1 GHz, millimeter waves for frequencies above 30 GHz.

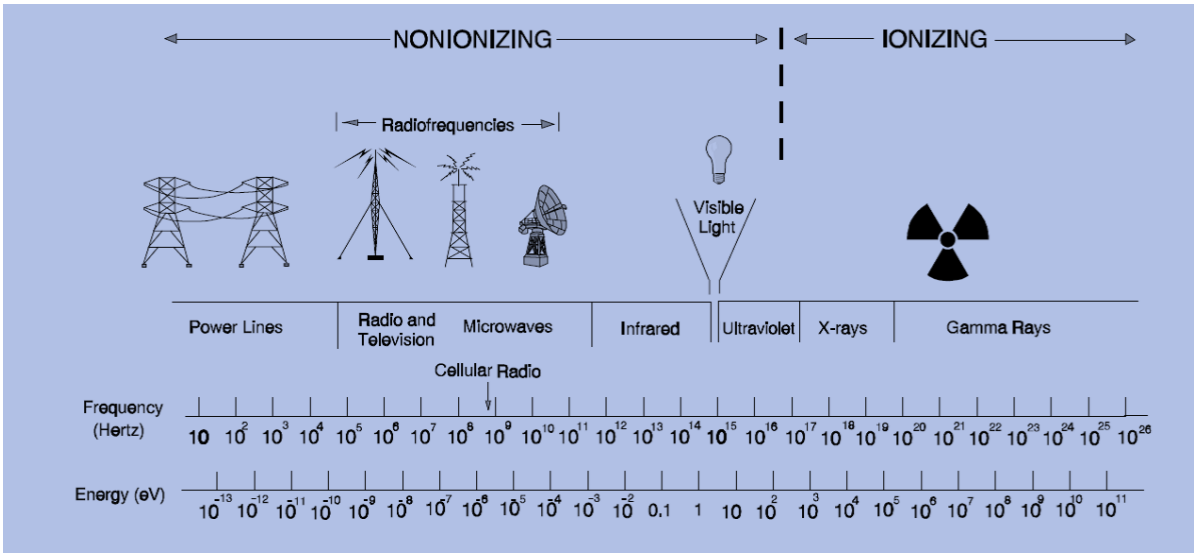

Figure 1. Electromagnetic Spectrum[1]

Cell Phones and RFR

Cell phones and other commonly used wireless communication devices are essentially two-way radios that contain both a receiver and a transmitter. When a user makes a call, voice sound is converted into digital information. The information is imposed on to RFR and transmitted to the nearest base station, commonly referred to as a cell tower, that receives and transmits RF signals and forms a bridge to the rest of the communications infrastructure. The base station receives and transmits radio signals in its area or "cell." As the user moves around, the radio signal can be relayed within the communications network from one "cell" of coverage to another, maintaining call connection. The call is routed through the communications network either through a landline phone or another wireless phone again using radio signals. To conserve energy and minimize interference, mobile phones automatically regulate the RFR signal strength, and hence the emitted field, to the lowest power level possible for a connection to be made. However, in a poor transmission environment (caused by, e.g., a distant base station, presence of obstacles between the base station and the mobile phone, or interference from adjacent cells), there is a higher output power and emission from the mobile phone in order to make a connection. Therefore, the better the connection, the lower the power output of the wireless device.

Cell Phone RFR Signal Modulation

In wireless telecommunications, modulation is the process of conveying digital or analog signals or information (the message) by varying one or more parameters of another signal (the carrier), typically at a much higher frequency. The modulated carrier contains complete information about the message signal and the original message can be recovered by suitable signal processing of the signal when received at a remote location (base station). One of the main goals of the modulation used in mass wireless communications systems is to transfer as much data as possible in the least amount of spectrum. Over the years, multiple modulation techniques have emerged to achieve and improve spectral efficiency, either when considering a single user in isolation or multiple users simultaneously using the same spectrum.

The first generation (1G) of wireless technology introduced in the 1980s, used analog frequency modulation for voice calls. This technology was replaced by second-generation (2G) networks that were digital, provided encryption, were significantly more efficient, and introduced data services [i.e., text messages, picture messages, and Multimedia Message Service (MMS)] in addition to voice calls. The 2G networks became commercially available in 1992 and used three common multiple access technologies for accommodating multiple simultaneous users:

- Frequency Division Multiple Access (FDMA): The available spectrum is split into a number of distinct parts (channels) each large enough to accommodate a single user or call without overlap, all users utilize their channel 100% of the time for the duration of the call or message. The channels are normally of equal bandwidth.
- Time Division Multiple Access (TDMA): The available spectrum is allocated to a single channel, each user or call assigned a certain portion of time.
- Code Division Multiple Access (CDMA): The available spectrum is allocated to a single channel, each user or call is assigned a unique sequence code to spread the message over the available spectrum. All users use the whole of the spectrum all of the time. At the receiver, the same unique sequence code is used to recover the desired signal from the sum of all the user calls.

2G systems used a combination of FDMA/TDMA for GSM or various versions of CDMA, for example, cdmaOne (IS-95). While the 2G technology continues to operate, subsequent third and fourth generations of network technologies were introduced in 1998 (3G), 2006 (4G), and 2011 [4G-Long Term Evolution (LTE)]. These technologies were developed to support increased data demands for multimedia access with increased bandwidth and transfer rates to accommodate internet-based broadband applications, including video conferencing, streaming video, sending and receiving faxes, and downloading e-mail messages with attachments. With the introduction of 3G technology, “smartphones” were developed. With these devices, the newer technologies were overlaid with 2G to support multiple access modes (2G, 3G, and 4G)[2]. Although the 2G technologies will be phased out over time and replaced by newer technologies, the current wireless communication networks continue to utilize 2G for voice and text.

All 3G systems utilize CDMA/WCDMA technology and fall into two groups complying with the 3rd Generation Partnership Project (3GPP) or 3GGP2 family of standards. Universal Mobile Telecommunications Service (UMTS), Wideband Code Division Multiple Access (WCDMA), and Time Division-Synchronous Code Division Multiple Access (TD-SCDMA) are 3GPP variants, CDMA2000 (which is based on 2G cdmaOne) is 3GPP2. 4G systems use Orthogonal Frequency Division Multiplexing (OFDM) within the E-UTRAS (LTE-Advanced) or Worldwide Interoperability for Microwave Access (WiMAX) standards.

Modulation Schemes (GSM and CDMA)

The Global System for Mobile Communications (originally Groupe Spécial Mobile; GSM) was developed to establish a digital standard for compatibility throughout Europe. GSM is a circuit-switched system that uses both FDMA and TDMA technologies. The frequency division mechanism divides the GSM band into 200 kHz-wide channels. The time division mechanism enables up to eight different time slots (voice channels) per frequency channel wherein a single cell phone transmits in only one out of eight available time slots during a voice communication. This introduces a pulsed signal shape with a pulse repetition rate of 217 Hz. Such a TDMA

frame has a length of 4.6 milliseconds (ms) (Figure 2), and 26 TDMA frames make up a multiframe with a 120 ms duration (Figure 3). During a multiframe, a mobile phone transmits in 25 out of 26 possible time slots. This TDMA frame structure causes significant low frequency amplitude modulation components to be superimposed on the RF carrier at 8.3 and 217 Hz. Furthermore, as a direct consequence of the TDMA the peak power, and instantaneous SARs are 8.3 × higher than the average power and SAR, note that the average power is the metric of importance for SAR determination within the context of the current safety standards.

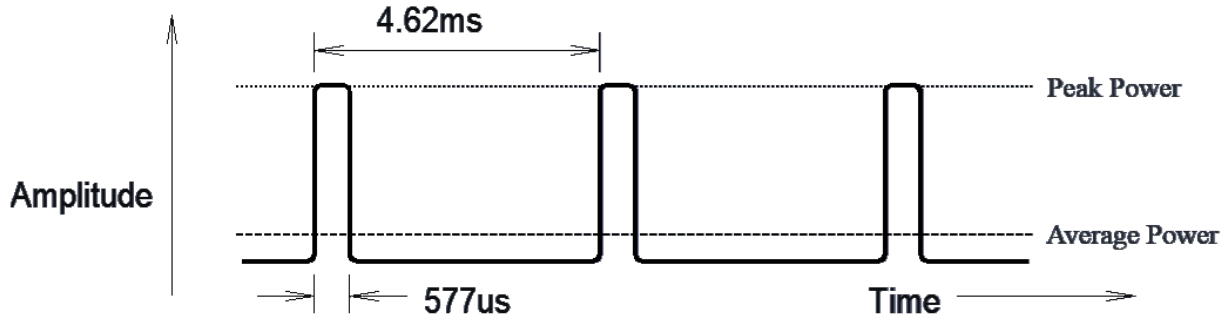

Figure 2. GSM Frame Showing Peak and Average Transmit Powers

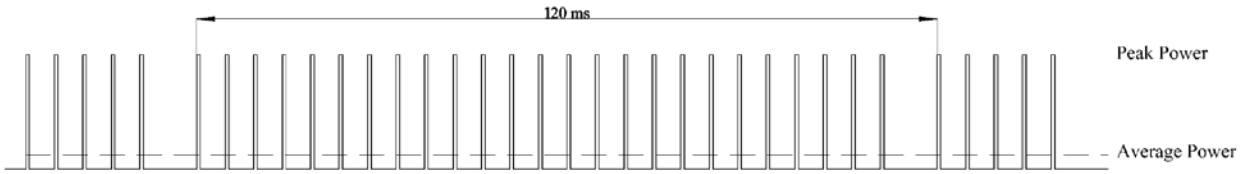

Figure 3. GSM Multiframe Showing Missing 26th Frame

With GSM, the duplexing between uplink (when the handset transmits to the base station) and downlink (when the base station transmits to the handset) is implemented in the frequency and time domain. Constant frequency spacing is maintained between up and downlink frequencies; in the United States the uplink is 1,850 to 1,910 MHz, and the downlink 1,930 to 1,990 MHz. The uplink and downlink frequencies are chosen according to the cell (area that is covered by a base station) into which the mobile is registered. In order to minimize interference between neighboring cells, a frequency reuse policy is applied. In this approach, when a mobile phone moves from one cell into an adjacent cell, frequencies used for data uplink and downlink change in association with this movement (i.e., transmission frequencies change at handover from one cell to another).

CDMA technology uses a form of coded transmission known as Direct Sequence Spread Spectrum (DSSS) in which data are multiplied by a much faster pseudo random code before being modulated on to the carrier. The effect of the multiplication is to spread the message across the whole frequency bands available for use at a given time in a given cell, but with very specific characteristics. CDMA signal access technology is based on code division separation of mobile stations as well as base stations. This implies differences of the signal structure compared to GSM. For example, in IS-95 in the forwardlink (downlink), a set of 64 Walsh codes (which are deterministic and orthogonal) are applied to spread/separate the individual channels in the

downlink of a cell. After the orthogonal spreading, a short (16-bit) Pseudo Noise code is applied to further spread the signal and identify the cell. Hence, a separation of neighboring cells in the frequency domain is no longer necessary, and there is no need for the mobile station to change its transmission frequency during the transition from one cell into another. As with GSM systems, the duplexing between the forward and reverse links is implemented in the frequency domain. In CDMA systems, an efficient power control is crucial. Because all mobile stations transmit and interfere in the same frequency channel, each mobile device decreases the signal to noise ratio of all the other mobile devices. Hence, the output power of a mobile phone should be kept at a minimum that guarantees good transmission quality.

IS-95, also known as cdmaOne, was developed by Qualcomm (San Diego, CA) as the first 2G CDMA-based digital cellular technology. The term IS-95 generally applies to a protocol revision (P_REV = 1) that was adopted as a standard (TIA-EIA-95) by the Telecommunications Industry Association (TIA) in 1995. Over time, subsequent iterations of the IS-95 protocol such as IS-95A, TSB-74, and IS-95B were developed, each with incremental improvements over the previous protocols. Later, more advanced versions of the CDMA technology have evolved to include IS-2000, which incorporated much higher transfer rates than the previous 2G versions. For a further explanation of these technologies and how the NTP exposure system was designed to reproduce similar GSM and CDMA cell phone RFR exposures please see the video presentation[a] (day 1 at 54 minutes) by Dr. Myles Capstick[3].

Sources, Use, and Human Exposure

The predominant source of exposure to RFR for the majority of the population is through use of telecommunications and mobile internet access applications for wireless devices, and the highest human exposure to cell phone RFR occurs through the use of cellular phone handsets and other wireless devices such as tablets and laptop computers held in close proximity to the human body. Aside from telecommunications, there are other man-made applications of RFR, which include microwave ovens, radar, industrial heating and sealing, medical diagnostics [Magnetic Resonance Imaging (MRI)] and therapy (surgical diathermy and ablation), and remote tracking or detection of objects [anti-theft, Radio Frequency Identification (RFID)]. There are also natural sources of RFR such as atmospheric electrical discharges (lightning) and solar and cosmic radiation. RFR exposures from natural sources are much smaller and tend to be spread over a much wider range of frequencies compared to exposures to fields from man-made radiation sources[4].

The use of cell phones has become widespread over the last two decades, and concern has been expressed regarding the potential health risks associated with use specifically by children. According to a Pew Research poll[5], approximately 95% of adult Americans own a cell phone. As of December 2015, the number of active wireless subscriber connections was 377.9 million, which exceeded the population of the United States[6]. According to the same survey, 49.3% of households in the United States utilize only a wireless phone, and not a landline.

There has been a great deal of focus on the possibility of increased risk of brain cancer because of the traditional use of these devices in close proximity (0 to 2 cm) to the head. In general (apart from the case when very close to the antenna), the level of RFR exposure from a cell phone is

[a] https://doi.org/10.22427/NTP-VIDEO-41.

inversely proportional to the square of the distance of the body from the device's antenna, resulting in the highest SAR levels in the parts of the body nearest to the antenna.

Accurate and detailed measurements of RFR exposure in humans are difficult to estimate because the output power of wireless devices constantly varies depending on several factors. Overall, the network carrier adjusts the output power of each connected device to the lowest level that is still compatible with a good quality signal. This adaptive power control occurs continuously and is achieved by a logarithmic downscaling of the time-averaged power from the maximum of 0.125 or 0.25 W to a level as low as 1 mW. When in use, the output power (and subsequent exposure to cell phone RFR) from the device is increased compared to that in "standby" mode. Therefore, exposures are related to the amount of active time a user spends on the device. The output power of a device changes based on the signal received at the base station. Decreases in signal strength result in higher output powers. Therefore, there are increases in the output power as the distance between the device and the base station increases, if there are physical obstacles between the device and the base station, reflections off buildings or other structures, and during handovers from one cell to another in the case of GSM. The proximity of the device to the body and the type, number, and position of antennas in the device are other important factors affecting the amount of exposure to RFR.

Potential exposure to RFR used in cell phones also occurs from the cell phone towers that form the network. While modern towers emit substantially more power than devices, exposures from base station antennas are considerably lower to users than from the handheld device. Typically, base station antennas are placed at heights of 50 to 200 feet, in order to adequately cover a cell. The antennas direct RF energy toward the horizon, with some downward tilt. As with all forms of radiation (ionizing and nonionizing), the RF energy level decreases rapidly as the distance from the antenna increases. As a result, the level of exposure to RFR at ground level is very low compared to the level close to the antenna.

Some base station antennas are installed on rooftops and at the top of lamp poles that are in close proximity or adjacent to office space and residential buildings. Occupational exposure can occur during maintenance of base stations. As a result, the FCC established guidelines for occupational exposures. Safety guidelines and regulatory compliance are discussed below.

The levels of RFR inside buildings with base station antennas mounted on the roof or on the side of the building are typically much lower than the level outside, depending on the construction materials of the building. Wood or cement block reduces the exposure to RFR by a factor of about 10. Due to the directional nature of the signals, the energy level behind an antenna is orders of magnitude lower than in front of the antenna.

Safety Guidelines for Exposure

The FCC and U.S. Food and Drug Administration (FDA) are jointly responsible for the regulation of wireless communication devices.

Federal Communications Commission

The FCC is required by its responsibilities under the National Environmental Policy Act of 1969 to evaluate the impact of emissions from FCC-regulated transmitters on the quality of the human environment[7]. As a result, the FCC regulates both the wireless devices as well as the base

stations. Since 1996, the FCC has required that all wireless communication devices (transmitting in the 100 kHz to 6 GHz frequency range) sold in the United States comply with its minimum guidelines for safety and maximum RFR absorption standards based on SAR. The FCC requires a formal approval process for all devices sold in the United States. FCC approval is contingent on the demonstration that the device does not exceed the maximum allowable SAR level when the device is operating at its maximum power. The SAR limit adopted by the FCC for exposure in the general population is 0.08 W/kg, as averaged over the whole body (wbSAR), and a peak spatial-average SAR (psSAR) of 1.6 W/kg, averaged over any 1 gram of tissue[8] when averaged over 6 minutes. Exceptions are made for the extremities (hands, wrists, feet, ankles, and pinnae), where the psSAR limit is 4 W/kg, averaged over any 10 grams of tissue for an exposure period of no longer than 30 minutes. For occupational exposures, the wbSAR limit is 0.4 W/kg and the psSAR limit is 8 W/kg, averaged over any 1 gram of tissue. For the hands, wrists, feet, ankles, and pinnae, the psSAR limit for occupational exposure is 20 W/kg, averaged over any 10 grams of tissue for an exposure period not to exceed 6 minutes.

The FCC rules and guidelines for cell phone RFR exposure are based upon standards initially developed by the Institute of Electrical and Electronics Engineers (IEEE) and the National Council on Radiation Protection and Measurements (NCRP). These standards for RF exposure in workers and the general population are based on protection against adverse effects that might occur due to increases in tissue or body temperature in excess of 1°C (wbSAR of approximately 4 W/kg) or less (after applying safety factors). Because RF-energy absorption and any induced effects are dependent on the frequency of incident-field parameters and the composition of exposed tissues, it has been suggested that quantifying SARs in small averaging regions is more relevant for evaluations of human health effects.

Food and Drug Administration

The FDA does not currently regulate the use of wireless communications devices or the devices themselves. The FDA also does not require safety evaluations for radiation-emitting wireless communication devices. It does maintain the authority to take regulatory action if it is demonstrated that exposure to the emitted cell phone RFR from these devices is hazardous to the user.

Absorption of RFR

RFR interacts with the human body via inductive or capacitive coupling or a combination of both. The absorption of the coupled RFR is dependent on the frequency of the signal and the dielectric properties of the exposed tissue. It generates oscillating currents in the tissue, which in turn give rise to induced E-fields. The energy is transferred into molecular motion of polar molecules like water, a strongly dipolar molecule and major component of biological tissues. Resonant oscillations in polar subgroups of cellular macromolecules are damped by collisions with surrounding water molecules that disperse the energy of the RF signal into random molecular motion. Tissue heating occurs as the energy is transferred to the surrounding aqueous environment as heat[4].

Toxicity

A comprehensive review of the toxicity of RFR in in vitro models, laboratory animals, and humans was conducted and published in the International Agency for Research on Cancer (IARC) Monograph series[4].

Thermal Effects

Given the ability of RFR to heat tissues, the toxic effects of RFR are often considered due to thermal effects. The most well-established and biologically plausible mechanism for RFR-induced effects is through tissue heating. At sufficiently high levels of RFR exposure, the absorption of energy could overwhelm an organism's ability to thermoregulate and maintain an acceptable body temperature. Typical human exposures to RFR occur at intensities that are not anticipated to cause significant tissue heating if handsets are used according to the manufacturers' recommendations for use, and assuming the phones are not emitting more RFR than permitted by FCC regulations.

Nonthermal RFR effects refer to biological changes that occur with body temperature increases that are below 1°C. Changes of temperature up to 1°C are considered in the range of thermal noise[4]. There is an ongoing debate regarding whether nonthermal biological effects can occur as a result of exposures to low-intensity RFR. It has been suggested that there is no plausible nonthermal mechanism by which exposure to low-intensity RFR could induce significant biological effects[9-11]. However, there are numerous reports of specific biological effects associated with RFR exposures at levels considered below those expected to result in a measurable amount of tissue heating. Other than tissue heating, the mechanisms of interaction between RFR and biological systems have not been well characterized, but several mechanisms have been proposed, including the generation of reactive oxygen species, induction of ferromagnetic resonance, and the alteration of ligand binding to hydrophobic sites in receptor proteins[4]. Additionally, low levels of exposure to RFR may result in small temperature changes in localized areas of exposed tissues that cause conformational changes in temperature-sensitive proteins and induce the expression of heat-shock or stress-response proteins.

Experimental Animals

Toxic effects have been reported in RFR-exposed laboratory animals and in vitro systems[4; 12]. Many studies investigating the potential toxicity of RFR have focused on genotoxicity and related effects and are reviewed in the Genetic Toxicity section. However, studies have been conducted to evaluate a variety of other aspects of toxicity, particularly those potentially related to cancer development or surveillance, including specific studies on gene and protein expression, immunotoxicity, and permeability of the blood-brain barrier. The results of these studies have not led to a clear understanding of the interactions of RFR with biological systems, but it's important to note that many of these studies were conducted with RFR of differing parameters (frequency, power density, continuous wave versus amplitude-modulated signals, etc.).

Several effects on the humoral and cell-mediated responses of the immune system have been reported at various frequencies of RFR in rats and mice. These include effects on the activity of NK cells, plaque-forming cell response to sheep erythrocytes, production of tumor necrosis factor (TNF) in peritoneal macrophages and splenic T-cells, mitogenic response in T lymphocytes, phagocytic activity of neutrophils, leukocyte profile, and thymic and splenic

cellularity[13-18]. However, many of these effects were observed in studies conducted with RFR at frequencies greater than 10 GHz. Other studies have demonstrated no exposure-related effects on the immune system[17; 19-23].

A few studies have investigated the impact of RFR at frequencies between 800 and 1,900 MHz on gene and protein expression. Several studies have demonstrated that RFR can alter the expression of certain genes in the brain[24-26], while others have failed to find changes in gene expression[27-29]. The expression of various proteins has also been investigated in rats and mice. These studies have primarily yielded negative results for the specific proteins being evaluated in the rat brain[24; 25; 30-32]. Similarly, no effects of RFR on protein expression have been reported in the testis[33] or in the skin[34-36]. Liu et al.[37] reported adverse effects on sperm following exposure for 2 hours/day to 900 MHz RFR at 0.66 W/kg for 50 days. Changes in the expression of bone morphogenic protein and bone morphogenic protein receptors have been reported in the kidney of newborn rats[38]. A study by Eşmekaya et al.[39] also demonstrated increased expression and activity for caspase 3 and caspase 9 in the thyroid gland of Wistar rats. Ohtani et al.[40] observed induction of expression of some heat shock protein genes in the cerebral cortex and cerebellum of rats exposed to 2.14 GHz of WCDMA RF at 4W/kg, but not in rats exposed for 3 hours, or for 3 or 6 hours to 0.4 W/kg.

Exposure to RFR induces changes in markers for oxidative stress in multiple tissues, including the brain[30; 41-44], heart[45], kidney[46; 47], eye[48], liver[49; 50], endometrium[51; 52], and testis and epididymis[53]. Yakymenko et al.[54] reviewed oxidative mechanisms reported in a number of in vitro and in vivo experiments with “low intensity” RFR. A few studies have also demonstrated RFR-mediated effects on differentiation and apoptosis in the endometrium[51; 52] and brain[32; 55]. Changes have also been noted in the permeability of the blood-brain barrier in some studies[56-58]. However, other studies conducted under similar experimental conditions failed to demonstrate any effect of RFR exposure on the permeability of the blood-brain barrier[59-62].

Humans

Numerous epidemiology studies have investigated the association between exposure to RFR and health effects in humans. However, many of these studies examined small groups exposed to RFR signals with different characteristics (frequencies, modulations, intensities, etc.) such as microwaves, extremely low frequency (ELF) fields, and radar rather than the specific frequency bands and modulated RFR signals used in wireless communication.

There is limited research investigating the general toxicity of RFR in humans because most of the focus has been on the potential for carcinogenic effects. There are reports of exposed individuals that complain of acute, subjective effects following exposure to RFR, including headaches, fatigue, skin itching, and sensations of heat[63-68]. These have primarily been reported in people that consider themselves electrosensitive. It has been suggested that there are likely other causes, not RFR, for these subjective symptoms[69]. Variable results have been observed in the electroencephalogram (EEG) of volunteers exposed to RFR during sleep. Some studies indicate that exposure to RFR induces changes in sleep latency and sleep EEG[70-80]. Glucose metabolism in the brain, a marker for metabolic activity, is increased in the region of the brain closest to the antenna[81]. While these results demonstrate exposure-related effects, the toxicologic significance of these findings is unclear.

Carcinogenicity

A comprehensive review of the carcinogenicity of RFR in laboratory animals and humans was conducted and published in the IARC Monograph series[4]. Additional reviews of animal cancer studies have been published by Lin[82], and of human studies by Repacholi et al.[83] and Yang et al.[84].

Experimental Animals

Studies published to date have not demonstrated consistently increased incidences of tumors at any site associated with exposure to RFR in rodents[82]. No increases in tumor incidences were observed in B6C3F1 mice exposed to GSM-modulated RFR for 24 months[85], F344 rats exposed to CDMA-modulated RFR for 24 months[86], or Wistar rats exposed to GSM-modulated RFR for 24 months[87]. In studies conducted in transgenic and tumor-prone mouse strains, exposure to RFR has not been consistently associated with an increased incidence of tumors at any site[88-92]. While these studies have advanced the knowledge of the potential toxicity of RFR, critical limitations in the design of many of these studies severely limit the utility of the information to adequately evaluate the carcinogenicity of RFR. These limitations include studies with very short daily exposure durations (≤ 2 hours per day) in heavily restrained animals or with levels of RFR exposures too low to adequately assess carcinogenic potential. The focus of many of these studies conducted in genetically altered and tumor-susceptible mice was not to evaluate the overall carcinogenicity of RFR, but to investigate the effects in the specific predisposed tissues in that model.

Based on the constraints in the designs of the existing studies, it is difficult to definitively conclude that these negative results adequately establish that RFR is not carcinogenic. To adequately evaluate the potential chronic toxicity and carcinogenicity of RFR, further studies with enhanced study designs and improved exposure paradigms were needed.

Humans

As a result of the IARC review conducted in 2011[93], RF electromagnetic fields were classified as possibly carcinogenic to humans (Group 2B). This classification was based on limited evidence of carcinogenicity in humans based on positive associations between exposure to RFR from wireless phones and increased risk for gliomas and acoustic neuromas, specifically in users with the greatest amount of cell phone usage. The IARC Working Group acknowledged that the findings were affected by potential selection and information bias, weakness of associations, and inconsistencies between study results[93].

While several other studies were considered, the IARC evaluation was based primarily on reports from the INTERPHONE Study, the largest research effort conducted to date examining the potential association between exposure to RFR and cancer in humans. INTERPHONE was an IARC-coordinated research effort that included a series of studies conducted with a common core protocol at 16 study centers in 13 countries: Australia, Canada, Denmark, Finland, France, Germany, Israel, Italy, Japan, New Zealand, Norway, Sweden, and the United Kingdom[94]. The studies were specifically designed to investigate the association between RFR and tumors of the brain (glioma and meningioma), acoustic nerve (schwannoma), and parotid gland. The final report for the INTERPHONE studies was published in 2011[93].

The results of these studies seemingly demonstrated an elevated risk of glioma and acoustic neuroma in the group in the highest decile for exposure (cumulative phone call time). However, the INTERPHONE study group concluded that recall and selection biases and implausible values for usage reported by the participants in the study may explain the increased risk[95; 96].

Other studies have compared time trends in cell phone usage and the incidences of different types of cancers to investigate indirect evidence of an association between RFR used in cell phones and cancer. These studies were conducted across several different countries[97], and in a group of European countries[98-102], the United States[103-105], Japan[106], New Zealand[107], and Israel[108]. Overall, the evaluations suggest that there was no significant change in the trends of cancer incidences. Any increases in cancer rates that were observed in these studies were attributed to enhanced detection capabilities for cancer that were the result of advances in diagnostic medical equipment, like computerized tomography (CT) scans and MRI.

Several cohort studies have been conducted, but also failed to establish a clear association between cell phone RFR and the development of any of the investigated cancer types[109-111]. Additional studies have demonstrated that there was no association between cell phone usage and pituitary gland tumors[112; 113], testicular tumors[110; 114], parotid gland tumors[115; 116], uveal melanoma in the eye[110; 117], and cutaneous melanoma[118]. Some studies have demonstrated that there was no association between cell phone usage and leukemia[109; 110] and non-Hodgkin's lymphoma[119], whereas others have reported increased risk of non-Hodgkin's lymphoma[120] and leukemia[121].

Since the 2011 IARC Working Group evaluation, few additional epidemiological studies have examined mobile phone use and risk of cancer. A case-control study of children and adolescents from four European countries did not find an association between overall mobile phone use with brain cancer[122]. A pooled analysis of multiple Swedish case-control studies by Hardell, Carlberg and colleagues found a significant increased risk of glioma and acoustic neuroma, particularly among analog phone, ipsilateral, and long-term or high frequency mobile phone users[123-126]. No increased risk of meningioma was found with overall mobile phone use[123; 124; 127]. Other case-control studies did not report an increased risk of glioma[128; 129] or meningioma[130] with regular mobile phone use; however, Coureau et al.[128] did find a significant increased risk of glioma and meningioma with heavy mobile phone users. A prospective cohort study of UK women did not find an association with glioma, meningioma, or acoustic neuroma[131; 132].

Numerous systematic reviews of the epidemiology literature database have been conducted in addition to the 2011 IARC evaluation, with conflicting conclusions. Available systematic reviews have found an association between cell phone use and increased risk of brain tumors[124; 133], while other reviews did not find an association with brain tumors[83; 134]. These contrasting results have been considered possibly due, in part, to differences in study eligibility criteria, the number of studies included, when the review was conducted, and how studies were evaluated[135].

Genetic Toxicity

Extensive reviews of the literature on the genotoxicity of various frequencies and modulations of RFR, covering experimental systems ranging broadly from cell-free DNA preparations to cells of exposed animals and humans, have concluded that evidence for cell phone RFR-associated genotoxicity is inconsistent and weak[83; 136-138]. Interpretations of the genotoxicity studies and the

ability to draw definitive conclusions based on weight-of-evidence from the large number of studies that have been reported have been hampered by inadequacies in experimental design, especially related to exposure standards and radiation-measuring procedures[136]. Although the majority of studies report a lack of effect, the several reports of a positive response are concentrated among experiments assessing chromosomal or DNA damage in mammalian cell systems in vitro and in vivo. Some key studies reporting RFR-associated genotoxicity in human cell lines, including DNA damage and chromosomal effects, could not be replicated[139; 140]. A critical complicating factor in the study of the genotoxic effects of cell phone RFR is that under certain conditions, RFR is sufficiently energetic to heat cells and tissues, and not all studies have considered this factor in their design. Heating of cells in vivo and in vitro has produced positive results in tests for genotoxicity, such as the comet assay and micronucleus assay[141-143]. The mode of action whereby heat induces these effects may be through induction of protein denaturation and aggregation, which can interfere with chromatin structure and slow the kinetics of DNA repair or interfere with mitosis by disrupting microtubule function[144; 145]. Thus, heat-induced increases in DNA migration seen in the comet assay may reflect slowed repair of endogenous lesions, and similarly, activity in the micronucleus assay may be due to aneugenic rather than clastogenic events[141-143]. Therefore, it is important to control thermal conditions when studying measures of genotoxicity following exposure to cell phone RFR.

Study Rationale

The FDA nominated cell phone RFR emissions of wireless communication devices for toxicology and carcinogenicity testing. Current exposure guidelines are based on protection from acute injury from thermal effects and little is known about the potential for health effects from long-term exposure to RFR below the thermal hazard threshold. Epidemiology studies that have been conducted to date have demonstrated possible, but not yet causal links between cell phone RFR and some health problems in humans, however the results of these studies are complicated by confounding factors and potential biases. Additionally, exposures in the general population may not have occurred for a long enough period to account for the long latency period of some types of cancers in humans. Similar to the challenges faced in epidemiological studies, studies in laboratory animals have been complicated by limitations that researchers have faced in conducting robust studies designed to characterize the toxicity and carcinogenicity of cell phone RFR.

For years, the primary concern regarding the potential health risk of chronic exposure to cell phone RFR was brain cancer based on the proximity of wireless devices near the head during use. While the brain is an organ of concern, understanding the potential toxicity and carcinogenicity of whole-body exposure is critical. RFR is constantly emitted from wireless devices to communicate with base stations, regardless of whether the user is on a call or not. As the public has become more aware of the uncertainty regarding the potential effects of RFR on the brain, more emphasis has been placed on the use of wired or wireless headsets (like Bluetooth), which minimize RFR exposure to the head. In recent years, the density of cell towers has increased to cope with the increasing demand for capacity, resulting in installations closer to residential neighborhoods and schools. Additional RFR technologies, like SmartMeters used by power companies, transmit data in real time using RFR. These existing and emerging technologies may potentially increase the level of exposures in human populations. These and other additional sources also expose different parts of the body, not only the head.

In 2011, RFR was classified by the IARC as possibly carcinogenic to humans based on limited evidence of an association between exposure to RFR from heavy wireless phone use and glioma and vestibular schwannoma (acoustic neuroma) in human epidemiology studies and limited evidence for the carcinogenicity of RFR in experimental animals[4]. While ionizing radiation is a well-accepted human carcinogen, theoretical arguments have been raised against the possibility that nonionizing radiation could induce tumors (discussed in IARC[4]). Given the extremely large number of people who use wireless communication devices, even a very small increase in the incidence of disease resulting from exposure to the RFR generated by those devices would translate to a large number of affected individuals, which would have broad implications for public health. Due to the changing exposure patterns and use of cell phones by pregnant women and women of childbearing age, RFR exposures to the whole body, and exposures during the perinatal period (rat studies only) were selected for inclusion in these studies.

In the current studies, male and female Hsd:Sprague Dawley® SD® rats were exposed to GSM or CDMA RFR at 900 MHz in utero, during lactation, and after weaning for 9 hours and 10 minutes per day for five or seven days per week, over the course of 18 hours and 20 minutes in 10 minutes on and 10 minutes off intervals for 28 days or 2 years. Exposures were 0 (sham control), 3, 6, or 9 W/kg in the 28-day studies and 0 (sham control), 1.5, 3, or 6 W/kg in the 2-year studies for each modulation. Exposure energy levels were selected based on pilot studies of body temperature changes from these RFR power levels reported in Wyde et al.[146]. The selection of 900 MHz for the frequency for the rat studies was based on dosimetry studies by Gong et al.[147] and the video[b], day 1 a.m. at 2 hours, 37 minutes[3].

[b]https://doi.org/10.22427/NTP-VIDEO-41.

Materials and Methods

Overview

The establishment of the National Toxicology Program (NTP) research program on radio frequency radiation (RFR) has required the coordination of expertise from multiple scientific and engineering disciplines. At the initiation of the RFR research program, a collaboration was established with technical experts from the Radio-Frequency Fields Group in the Radio Frequency (RF) Technology Division, which is part of the Communications Technology Laboratory (CTL) at the National Institute of Standards and Technology (NIST, Boulder, CO). NIST evaluated the existing exposure systems and identified the types of improvements that would be required to provide a system of sufficient size and power to conduct robust toxicology and carcinogenicity studies with uniform RFR exposures in unrestrained, individually housed animals for a minimum of 6 hours a day at frequencies and modulations that reflected those in use at the time. The design of the chambers and toxicology studies required special consideration of logistical, financial, and engineering limitations.

NIST tested the feasibility of a reverberation chamber-type exposure system by conducting a series of studies on field strengths, field uniformity, and power requirements under various conditions of RFR exposure in such chambers. These studies provided critical information for the design of experimental studies with respect to the number of cages that could be placed in specific size chambers, the arrangement of cages within each chamber, and the input power requirements.

Concurrent with the collaboration with NIST, the NTP also worked with the Foundation for Research on Information Technologies in Society (IT'IS, Zurich, Switzerland), which conducted studies using computational models that simulated RFR dosimetry to provide estimates of whole-body and organ-specific internal field strengths and specific absorption rates (SARs) during exposure. Based on information and parameters obtained during the NIST feasibility studies, IT'IS built a prototype reverberation chamber as the basis for an exposure system to study health effects of long-term exposure of laboratory animals. Following completion, NIST evaluated the prototype exposure chamber to determine if it met the requirements specified by the NTP.

Institution and Role:

- National Institute of Standards and Technology (NIST) (Boulder, CO)
  - Suggested reverberation chamber exposure system
  - Conducted feasibility studies for reverberation chambers
  - Established various technical parameters for chambers
  - Evaluated the prototype chamber built by IT'IS Foundation
  - Validated the system prior to the conduct of studies at IITRI
  - Reevaluated RFR exposures prior to and after 2-year studies
- IT'IS Foundation (Zurich, Switzerland)
  - Constructed and tested prototype chamber
  - Refined technical parameters

  - Built the chambers for the NTP exposure facility
  - Installed chambers at IITRI
  - Monitored system performance throughout all phases of the studies
  - Conducted maintenance on exposure system hardware and software
- IIT Research Institute (IITRI) (Chicago, IL)
  - Tested exposure system after installation
  - Conducted maintenance of exposure system hardware
  - Conducted all toxicology and carcinogenicity studies
  - Conducted day-to-day operations

After prototype-testing by IT'IS Foundation and NIST, the IT'IS Foundation built the reverberation chambers required for the NTP RFR exposure facility. Chambers were installed at the Illinois Institute of Technology (IIT) Research Institute (IITRI, Chicago, IL). Following the installation and initial testing of the exposure system by IT'IS and IITRI, technical experts from NIST conducted an independent validation of the system. NIST confirmed that the probe readings in the system were consistent, that field uniformity was within expected specifications, and that the signal quality was acceptable. NIST performed additional evaluations prior to initiation of the 2-year studies and after completion of the studies to determine if any changes occurred in the signal quality, field uniformity, or consistency of in-chamber field measurements. All studies were conducted at IITRI with real-time monitoring of the system performance at IT'IS Foundation.

Reverberation Chamber Method of Exposure

The use of the reverberation exposure chamber as a method for exposing rats and mice to cell phone RFR was conceptualized by the NIST and further designed and tested by NIST and the IT'IS Foundation. A reverberation chamber is a resonant box where the resonances and field structure are continuously modified under the influence of metallic stirrers, introduced to change the effective geometry, such that when averaged over time, the field strength is uniform over the entire exposure volume. A reverberation chamber exposure system was chosen for the NTP for the primary benefit that controlled exposures can be achieved in unrestrained animals (rats and mice) with extended daily RFR exposure periods compared to other methods of exposure for up to 2 years.

Preliminary studies were first conducted at the NIST to test the concept of reverberation chambers. In these studies, field strengths and field uniformity were measured under various conditions of RFR exposure, including an empty chamber and a chamber loaded with water bottles (simulating animals) at different locations in the chamber. Power requirements were evaluated to achieve desired SAR levels. The effects of proximity between water bottles were also investigated to avoid electromagnetic coupling. These studies provided critical information for the design of experimental studies with respect to the number of cages that could be placed in specific size chambers, the arrangement of cages within each chamber, and the input power requirements. The results of these investigations demonstrated that while variations occurred over time and space the average RFR field was uniform over the large volume of the chamber. These studies also demonstrated that RFR field exposure occurred from all directions and all

polarizations, and that there was uniformity of SAR in reverberation chambers. Based on the information and parameters obtained during the NIST feasibility studies, a custom-built prototype reverberation chamber was constructed and tested by the IT'IS Foundation. The development of the prototype chamber involved the design of amplifiers and antennas for signal generation, the design of vertical and horizontal stirrers to improve the homogeneity of experimentally generated RF fields, the development of both hardware and software for the control and monitoring of experimentally generated RF signals and testing of chamber performance. During the design of the prototype exposure chamber, engineering studies were performed to optimize the following prior to construction:

- The uniform field volume within each chamber to minimize spatial variability in the characteristics of generated RF fields within a chamber such that all animals housed within the chamber space were exposed to comparable RF field strengths.
- The design and placement of stirrers in each chamber in order to maximize homogeneity of experimentally generated RF fields.
- The design and location of RF antennas in each chamber.
- The location of cage racks within the exposure chamber in order to provide appropriate separation of individual animal cages and cage racks from all reflective surfaces (chamber walls, chamber floor and ceiling, antennas, and stirrers) in the reverberation chamber.
- Chamber volume to provide adequate space for staff to observe animals, collect data, and perform routine animal husbandry operations, while minimizing overall chamber volume to minimize the chamber size/footprint and the RF power required to maintain target SARs.

The final reverberation chamber design for use in these studies was a fully shielded room constructed of stainless steel, equipped with a shielded room door to eliminate leakage of RFR signals, two rotating stirrers (one horizontal and one vertical), ventilation structures, and RFR excitation antennas. A detailed rationale for the selection of reverberation chambers for exposure to RFR and a full description of the exposure system are provided in Capstick et al.[148] and Gong et al.[147] and in a video[c] (day 1 a.m. at 54 minutes) on the NTP website[3].

As part of the validation of the reverberation chamber exposure system design, a team of engineers from NIST conducted an independent evaluation of chamber design and exposure system operation in order to evaluate the suitability of the reverberation chamber model for use in the program. NIST engineers evaluated the design and operation of the prototype chamber and performed an extensive series of RF measurements to support an evaluation of system performance. Further information on the exposure verification is found in the video[d] (day 1 p.m. at 0 minutes) by John Ladbury[3].

RFR Exposure Facility

The exposure facility was specifically designed to expose rats in reverberation chambers to three different power levels of modulated cell phone RFR [Global System for Mobile Communications

[c] https://doi.org/10.22427/NTP-VIDEO-41.
[d] https://doi.org/10.22427/NTP-VIDEO-42.

(GSM) or Code Division Multiple Access (CDMA)] at 900 MHz for up to 2 years to evaluate toxicity and carcinogenicity. The completed exposure facility consisted of a total of 21 RFR reverberation exposure chambers (14 designated for rats); the RFR signal generation, amplification, and monitoring systems; software for chamber operation; and hardware and software for monitoring of environmental and exposure conditions within each chamber. All system hardware and software was installed by the IT'IS Foundation.

During exposures, modulated (GSM or CDMA) RFR signals were generated by a signal generator, amplifiers amplified the signals, and the signals were delivered by antennas in the reverberation chambers. RFR field strengths were monitored in real time and were adjusted throughout the studies to achieve specific exposure levels [based on SARs quantitated in watts (W) per kg body weight]. Environmental conditions were also monitored and controlled in real time throughout the study. RFR exposures and environmental conditions were monitored and controlled by a computer in a control room at the study laboratory at IITRI; the IT'IS Foundation was also capable of remote system monitoring and control.

Facility Design and Reverberation Chambers

Each reverberation chamber was permanently programmed for a specified modulation (GSM or CDMA) of the 900 MHz RFR specified for the rat studies. Designated SARs for each chamber were selected prior to exposures. The field strength required to achieve a given target SAR (W/kg) exposure level is a function of animal body weight (kg) and were adjusted to provide consistent SARs as the animals grew. However, separate chambers were required for male and female rats because their body weights differ by almost a factor of two after a few weeks of development. To conduct robust toxicology studies with three exposure groups (low, medium, and high), six chambers were required for different levels of exposures for GSM modulation and six for CDMA modulation. Two sham exposure chambers without any cell phone RFR signal provided shared control groups for the parallel studies of the two modulations. As per these requirements, the cell phone RFR exposure facility consisted of 14 reverberation chambers for exposures in rats including:

- Three power levels for F_0 females and F_1 males exposed to GSM-modulated cell phone RFR at 900 MHz.
- Three power levels for F_1 females exposed to GSM-modulated cell phone RFR at 900 MHz.
- Three power levels for F_0 females and F_1 males exposed to CDMA-modulated cell phone RFR at 900 MHz.
- Three power levels for F_1 females exposed to CDMA-modulated cell phone RFR at 900 MHz.
- One sham control chamber for F_0 females and F_1 males with no RFR exposure.
- One sham control chamber for F_1 females with no RFR exposure.

The chamber size was designed to accommodate the RF field stirring paddles (described below), approximately 110 individually housed rats, and a minimum distance (3/4 of a wavelength) between the cages and the walls, floor, ceiling and stirrers, respectively. The interior of the chamber was suitable for cleaning using high-pressure water (after the RF antennas were protected). The internal dimensions of the chambers were 2.2 m (width) × 3.7 m (length) × 2.6 m

(height); the exterior dimensions were 2.3 m (width) × 3.8 m (length) × 2.85 m (height). A floorplan for the exposure facility and images of the interior and exterior of the chambers are presented in Figure 4 and Figure 5.

Each chamber contained two motor-controlled stirring paddles (one vertical and one horizontal) with adjustable speed control (1 to 50 rpm) and large asymmetrical reflecting surfaces. Stirring paddles were placed off center in the chamber for maximum scattering of the RFR fields to generate a statistically homogeneous field distribution when averaged over time. The horizontal stirrer was mounted on the ceiling of the chamber. The vertical stirrer was at the rear of the chamber and was protected by rack guides that prevented contact with the animal cage racks.

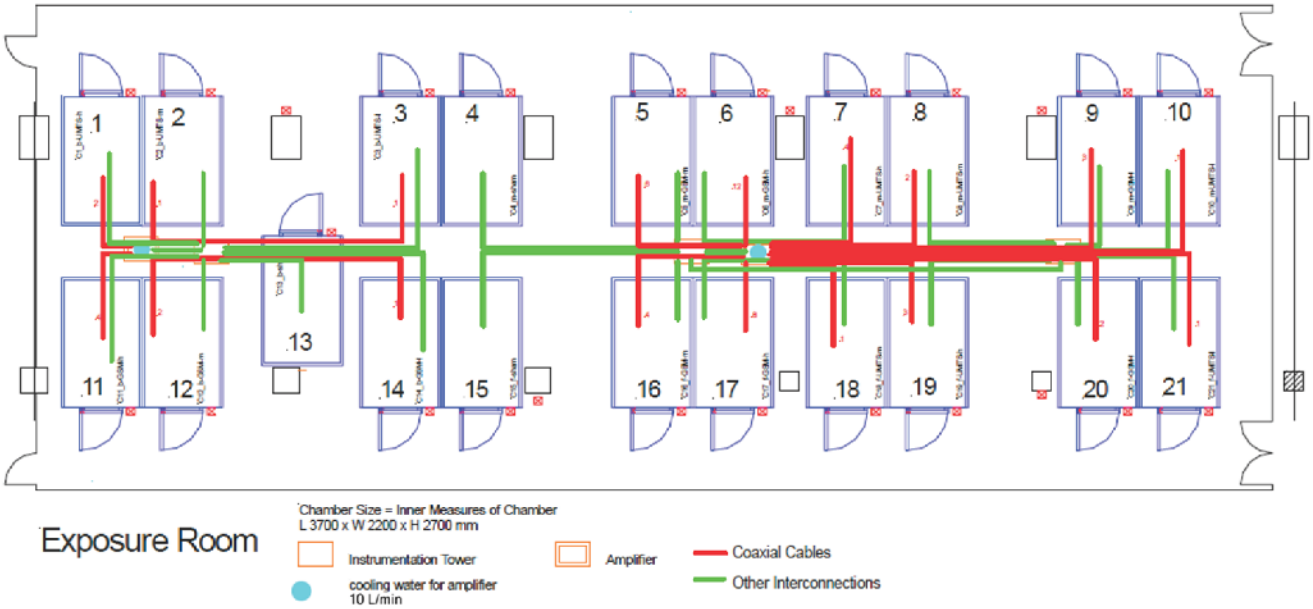

Figure 4. Exposure Facility Floor Plan for the Cell Phone RFR Studies

(Not shown are the Ethernet connections to computers in the control room.) Rat chamber designations: F_0 females and F_1 males−low GSM = 9; medium GSM = 5; high GSM = 6; low CDMA = 10; medium CDMA = 8; high CDMA = 7; sham = 4; F_1 females - low GSM = 20; medium GSM = 16; high GSM = 17; low CDMA = 21; medium CDMA = 18; high CDMA = 19; sham = 15. The seven other chambers (including six for cell phone RFR exposure and one for sham control) were designated for concurrent mouse studies.

Figure 5. Exterior View of Chambers, Empty Chamber Showing the Vertical and Horizontal Stirrers, and Chamber with Cage Racks in Place

Cage Racks and Watering System

Cages, cage racks, and watering systems for standard laboratory use contain elements that have the ability to alter the exposure of the animals or introduce potential confounding factors. Because cage racks and the drinking water delivery system were contained inside the chambers during exposure periods, it was required that these components be constructed of durable materials that had essentially no impact on the RF fields generated in the chamber. Metallic cage rack components, cage lids, feed dispensers, and cage grommets all needed to be eliminated. Hence, custom engineering was required to overcome the challenges regarding potential RFR exposure-altering aspects of the caging and cage racks used to house the animals during the studies. The safe provision of drinking water provided the largest challenge for the studies.

The absorption of RFR energy by water if supplied by nonmetallic sipper tubes and distribution systems or bottles, could lead to dose-dependent elevated water temperatures. At the same time, the potential for enhanced exposure fields by metallic sipper tubes or lixits precluded the use of water bottles or a standard automatic watering system in the reverberation chambers. The absorption of RFR energy by water could result in significant heating of the drinking water, thereby decreasing water palatability, and increasing the required RFR power to achieve the desired exposure field strength, potentially to the extent that the exposure levels could not be met. To overcome theses challenges, adaptations were made to an automatic watering system so that the delivery of drinking water to the animals would not interfere with RFR dosimetry. The water system was constructed from stainless steel ensuring no dose-dependent energy absorption in the water (avoiding exposure-dependent water temperature) and in structures around the lixits to ensure no enhanced fields that could lead to excessive SAR in the animals while drinking.

Customized, nonmetallic animal cage racks for the reverberation chambers were designed by IITRI to minimize any absorption of RFR or disruption of RF field homogeneity. Cage racks were constructed primarily of box beam fiberglass (with some angle beam fiberglass used in nonweight-bearing areas of the rack). The shelves/cage lids were constructed of a clear polycarbonate sheet with slots for increased airflow. The potential impact of the racks on RF fields was evaluated in the prototype reverberation chamber by the IT'IS Foundation. Cage racks were designed to accommodate the automatic watering system and position the perimeter of each animal cage at least one-half wavelength from any reflecting surface. The specific considerations for design and further details of the custom-designed cage racks and adapted automated watering system are provided in Capstick et al.[148] and in the video presentation[e] by Dr. Myles Capstick[3].

RFR Exposure System Control

The hardware and chambers designated for rats (using an exposure frequency of 900 MHz) were connected to a dedicated computer control system using an Ethernet protocol. The computerized control system managed and monitored the RFR exposures and environmental conditions in the chambers. A more detailed description of the computer control of RFR exposure is provided in Capstick et al.[148].

The control computer managed the exposure schedule, stirrer rotation speeds, exposure signal and level, and monitored air flow, temperature, humidity, light, and the electric and magnetic fields (E- and H-fields, respectively) in each chamber. The hardware for the exposure system

[e] https://doi.org/10.22427/NTP-VIDEO-41.

consisted of the control computer and a rack containing communications interfaces and instrumentation for signal generation, data acquisition, signal monitoring, signal amplifiers, and the chamber hardware (which included the stirrer motors and environmental and RFR sensors). The instrumentation rack contained the equipment that generated the RFR signal, acquired RFR field strengths and environmental data, and provided an interface between the components and the control computer.

RFR Signal Generation

GSM-modulated and CDMA-modulated cell phone RFR signals were generated experimentally via a SMIQ02B vector signal generator with options SMIQB11 and SMIQB20 and software options 100421–100423 (Rohde and Schwarz, Munich, Germany). Signals were amplified using 12 LSE™ amplifiers (LSE, Spanga, Sweden) in the exposure system. The outputs of each individual amplifier were set by real-time controllers on a slot-by-slot basis for GSM or CDMA modulation to control the E-field strength in each chamber. Each chamber contained at least one standard gain antenna (two half-wave dipoles) that was mounted a quarter of a wavelength in front of a reflector plate. Antennas were directed towards one of the two stirrers to maximize scattering and obtain acceptable E-field homogeneity within the chamber space. The computerized control system managed the exposure schedule, stirrer rotation speeds, and exposure signal type and level.

The RFR power introduced into a given chamber was adjusted to achieve target field strengths: to maintain constant exposure levels (W/kg) in a given chamber, the field strengths [measured in volts (V) per meter] were regularly adjusted to reflect changes in the average mass of the exposed animals. The relationship between animal mass, field strength, and SAR was determined from numerical dosimetry and programmed into the control software, hence the required exposure field strength was computed from the average animal weights entered for each exposure group. The interval at which animal weights were updated was determined on how rapidly the animals were growing, at the start of the exposure period this was once per week, and as long as up to every 4 weeks later in the studies.

Verification of RFR Exposure

Prior to initiation of the animal studies, the RF Fields Group in the Communications Technology Laboratory at the NIST performed an independent, detailed evaluation of each of the reverberation chambers (excluding the sham control chambers; Figure 4) to verify the RFR exposure fields, chamber characteristics (field uniformity), and signal quality to determine the accuracy of field values reported by the developers of the exposure system (IT'IS Foundation). This information provided in the video[f] (day 1 p.m. at 0 minutes) by John Ladbury[3]. Full reports detailing the procedures for measurements and calculations are available from the NTP. NIST performed two additional detailed evaluations: 1) in the interim period between completion of the 28-day studies and prior to initiation of the 2-year studies, and 2) following completion of the 2-year studies.

All E-field measurements were within the estimated uncertainty bounds, indicating that the chamber fields measured by the NIST agreed with the measurements provided by the IT'IS

[f] https://doi.org/10.22427/NTP-VIDEO-42.

Foundation probes. During validation, it was determined that the H-field probes at higher signal levels in the mid- and high-power GSM chambers reported higher fields than indicated by other measurements, potentially leading to a modest overestimation of chamber field strengths. In these chambers, H-field probes were replaced with E-field probes, which provided more accurate measurements of the RF fields. The magnitude of field variation throughout the volume of a fully loaded chamber was consistent with earlier values reported for the prototype chamber. However, it was determined that there may have been up to ± 2.5 dB of variation in the exposure field depending on location in the cage racks. To mitigate this positional variation, cages were routinely rotated to various locations within and between the cage racks. The quality of the modulated signals was found to be acceptable with regard to distortion and harmonic content.

Overall, NIST confirmed that the RFR reverberation chamber exposure system was operating correctly and RFR exposures were within specifications.

RFR Exposure Monitoring

During all exposure periods, experimentally generated RFR was continuously monitored by the control system via two RF sensors (E- and/or H-field probes) in each exposure chamber that measured real-time signal strengths. The use of two probes provided two independent measurements of RF field strengths and ensured that appropriate quantitation of experimentally generated RF fields continued even in the unlikely event that one probe failed. The E-field sensor measured electric field strength (V/m). The H-field sensor measured magnetic field strength [measured in amperes (A) per meter]. All chambers were instrumented either with one E-field sensor (ER3DV6) and one H-field sensor (H3DV6) [both from Schmid and Partner Engineering AG (SPEAG), Zurich, Switzerland], except for the medium- and high-power GSM chambers. These chambers were instrumented with two E-field probes because H-field probes saturated at high field strengths. This change in hardware did not result in the loss of monitoring capability. The measured E- and H-fields were communicated to the control computer in order to maintain exposure to selected levels of RFR. During daily shutdown periods when RFR exposures were not active, RF sensors monitored ambient RF fields in the exposure chambers. RF sensors were calibrated twice by the manufacturer (SPEAG); once prior to initiation of any of the animal studies and once prior to initiation of the 2-year studies. All E-field probes were calibrated in air from 100 MHz to 3.0 GHz, and had an absolute accuracy of ± 6.0% (k = 2) with a spherical isotropy of better than ± 0.4 dB. All H-field probes were calibrated in air from 200 MHz to 3.0 GHz and had an absolute accuracy of ± 6.0% (k = 2) with a spherical isotropy of better than ± 0.2 dB. Placement of probes within the chambers is discussed in the video[g] (day 1 a.m. at 1 hours, 31 minutes)[3].

Data collected by the RF sensors were transmitted to the exposure and monitoring system on a real-time basis and were recorded throughout the studies. Chamber field strengths are reported as V/m and animal exposure levels (SAR values) are reported as W/kg. The chamber field strength is the average effective E-field strength from both probes. E- and H-field strengths are related by the impedance of free space which is ~377 Ohms. Where an H-field probe was used, the value in A/m was multiplied by 377 to calculate the equivalent E-field strength in V/m; it is this effective E-field value that was used to report the chamber field strength. Field strength data reported for each day of exposure included mean ± standard deviation, minimum field strength, maximum

[g] https://doi.org/10.22427/NTP-VIDEO-41.

field strength, total number of readings in range/total number of readings for the period, and percentage of readings in range. After each exposure day, RFR exposure data were downloaded onto DVDs for long-term archival. Summaries of the 2-year RFR exposure data from the studies are presented in Appendix I. The SAR and chamber-fields in the exposure chambers were within the target ranges (defined as ± 2 dB) for >99.97% of recorded measurements over the course of the 2-year study; ≥99.25% of recorded E-field and H-field measurements were within the target ranges. All recorded broadband field measurements (<40 MHz to >6 GHz) were below the limit of detection of the probes within the sham chamber showing that there was no significant confounding exposure. In the 28-day studies, the performance of the sham control and exposure chambers was similar for SAR and field measurements as in the 2-year studies (data not shown).

As previously stated, the performance of the RFR exposure and monitoring system was independently validated by engineers from the NIST prior to the initiation of the animal studies.

Monitoring and Maintenance of Environmental Conditions

Environmental conditions including temperature, humidity, and airflow in all exposure chambers, as well as in other areas of the IITRI RFR exposure facility, were maintained by a computer-controlled environmental management system (Siemens Industries, Inc.). Monitoring instrumentation for each chamber was located in the air exhaust duct. Each chamber was fitted by the IT'IS Foundation with a sensor box that contained sensors for temperature and humidity (Type EE06; E + E Elektronik GmbH, Engerwitzdorf, Austria), oxygen level (Pewatron Type FCX-MC25; Zurich, Switzerland), air speed (model EE65A; E + E Elektronik GmbH), light (light-dependent resistor), noise (design based on WL-93 microphone; Shure Brothers, Inc., Evanston, IL), and RFR. Outputs from the sensor box were monitored using Agilent data acquisition units, with the exception of the RF sensor. The RF sensor was directly wired to a warning light as a safety precaution to indicate active RFR exposures and not intended to quantitatively measure RFR field strengths.

Exposure chambers were equipped with incandescent lights located on light bars in each corner of the chamber. All connections were RF-filtered. Chamber lighting was controlled using an adjustable daily cycle of 12 hours on, 12 hours off. In order to minimize the heat load generated by the incandescent lights, low wattage bulbs were used that maintained chamber lighting within a range that was sufficient to support normal in vivo operations, while minimally affecting chamber temperature. Further discussion of chamber lighting is found in the video[h] (day 1 a.m. at 1 hours, 27 minutes)[3].

Differences in noise levels in the exposure chambers that resulted from the heating, ventilation, and air conditioning system were equalized by the installation of sound baffles in various ducts within the system. An audible signal generated by the high intensity GSM signal was detected and equalized in all chambers by the introduction of a "pink noise" masking sound; this masking noise equalized sound levels in all chambers. As a result of the combination of these efforts, noise levels in all chambers were essentially equivalent at approximately 62 dBA and met the NC-35 noise specification. The noise criterion (NC) is a widely accepted numerical index commonly used to define the maximum allowable noise. It primarily applies to the noise produced by ventilation systems, but is applied to other noise sources, as well. Standards

[h] https://doi.org/10.22427/NTP-VIDEO-41.

organizations, such as the American National Standards Institute (ANSI), Acoustical Society of American (ASA), and International Standards Organization, provide definitions of various NCs for ambient noise in enclosed spaces. The ANSI/ASA standard (S12.2-2008) recommends NCs for various types of rooms, including private residences (NC 25-40), schools (NC 25-35), offices (NC 25-40), libraries (NC 30-35), and restaurants (NC 40-45). For further discussion of noise control in these studies see the video8 (day 1 a.m. at 2 hours, 0 minutes)[3].

Animal Source

Time-mated (F0) female Hsd:Sprague Dawley® SD® rats were obtained from Harlan Laboratories, Inc. (now Envigo, Indianapolis, IN), for use in the 28-day and 2-year studies.

Animal Welfare

Animal care and use are in accordance with the Public Health Service Policy on Humane Care and Use of Animals. All animal studies were conducted in an animal facility accredited by AAALAC International. Studies were approved by the IITRI Animal Care and Use Committee and conducted in accordance with all relevant NIH and NTP animal care and use policies and applicable federal, state, and local regulations and guidelines.

Twenty-eight-day Studies

The 28-day studies were conducted to evaluate the cumulative effects of repeated GSM- or CDMA-modulated cell phone RFR exposure and to determine the appropriate RFR power levels to be used in the 2-year studies. The exposure levels in these studies were selected based on the findings of minimal increases in body temperature observed in 5-day studies at exposures up to 12 W/kg RFR[146]. Beginning gestation day (GD) 6, separate groups of F0 female rats were housed in reverberation chambers and received whole-body exposures to GSM- or CDMA-modulated cell phone RFR at power levels of 0 (sham control), 3, 6, or 9 W/kg, for 9 hours and 10 minutes per day for 5 or 7 days per week with continuous cycling of 10 minutes on and 10 minutes off during a period of 18 hours and 20 minutes each day. The sham control animals were housed in reverberation chambers identical to those used for the exposed groups, but they were not exposed to RFR; shared groups of unexposed rats served as sham controls for both RFR modulations.

In order to evaluate potential toxicity that arises from in utero and early postnatal exposure, these developmental windows were included in the cell phone RFR studies in rats. F0 female rats were approximately 11 to 14 weeks old upon receipt. GD 1 was defined as day with evidence of mating, and F0 females were received on GD 2 and held in quarantine until GD 5. Animals were randomly assigned to GSM or CDMA exposure groups (20 F0 females/RFR power level per modulation) with a single group of 20 F0 females serving as the sham control group for both the GSM and CDMA modulations. Randomization was based on body weights that produced a similar group mean value (ToxData, version 2.1.E.11, PDS Pathology Data Systems, Inc., Basel, Switzerland).

In 10 F0 females per group, subcutaneously implanted temperature microchips and monitoring equipment (Bio Medic Data Systems, Seaford, DE) were used to monitor individual animal body temperatures. Body temperature measurements were taken prior to initial exposure (GD 6) and

on GDs 7, 11, and 16 and postnatal days (PND) 1, 4, 7, and 14 within 3.5 (GDs) or 2 (PNDs) minutes of exposure pauses at the end of the second to the last “on” cycle.

F0 females were housed individually during gestation and with their respective litters during lactation. During gestation, F0 females were weighed on GDs 6, 9, 12, 15, 18, and 21. During lactation, F0 females were weighed on PNDs 1, 4, 7, 14, 17, and 21 and individual F1 pup weights were recorded on PNDs 4, 7, 14, 17, and 21. The day of parturition was considered PND 0. From GD 20 to 25, F0 females were observed twice daily for parturition. All F0 females that did not deliver within 3 to 4 days of the anticipated delivery date were euthanized and the uteruses were examined for uterine implantations/ resorptions. On the day after parturition (PND 1), the number of live and dead F1 pups, sex ratio, whole litter weights, and litter weights/sex were recorded.

F1 litters were standardized on PND 4 to eight pups/litter, preferably with four males and four females each to equalize lactational pressure on F0 females. Litters that did not meet a minimum of eight pups were removed from the study. For continuation of exposure after weaning, three males and three females per litter from 10 F1 litters were randomly selected per exposure group. Weaning occurred on PND 21. Pups not selected and all F0 females were euthanized with 100% carbon dioxide without necropsy. Weaning marked the beginning of the 28-day prechronic phase of the study.

Groups of 10 male and 10 female F1 rats were housed in the same reverberation chambers and continued to receive whole-body exposures to GSM- or CDMA-modulated RFR at the same power levels for 9 hours and 10 minutes per day for 5 or 7 (last week of study) days per week for at least 28 days, with continuous cycling of 10 minutes on and 10 minutes off during a period of 8 hours and 20 minutes each day.

The health of the animals was monitored during the studies according to the protocols of the NTP Sentinel Animal Program (Appendix K). All test results were negative.

Animals were observed twice daily and weighed weekly. Clinical findings were recorded weekly. Subcutaneously implanted temperature microchips and monitoring equipment (Bio Medic Data Systems, Seaford, DE) were used to monitor individual animal body temperatures. Body temperature measurements were taken on day 8 after microchip implantation and on days 16, 20, and 27 within 5 minutes of exposure pauses at the end of the second to the last “on” cycle. Rats were housed individually. Feed and water were available ad libitum. To avoid interference with RFR dosimetry, feed was provided in glass (nonmetallic) jars and water was delivered in an adapted automatic watering system. Cages were changed weekly and rotated within the racks weekly; racks were changed biweekly. Further details of animal maintenance are given in Table 1. Information on feed composition and contaminants is provided in Appendix J.

Necropsies were performed on all F1 rats on PND 29 or 30. Organs weighed were the right adrenal gland, brain, heart, right kidney, liver, lung, right testis, and thymus. Tissues for microscopic examination were fixed and preserved in 10% neutral buffered formalin (except eyes, testis with epididymis, and vaginal tunics were first fixed in Davidson’s solution or modified Davidson’s solution), processed and trimmed, embedded in paraffin, sectioned to a thickness of 4 to 6 µm, and stained with hematoxylin and eosin. Complete histopathologic examinations were performed by the study laboratory pathologist on all 0 (sham control) and

9 W/kg GSM- and 9 W/kg CDMA-modulated cell phone RFR core study F1 rats. Table 1 lists the tissues and organs examined.

The laboratory reports and selected histopathology slides were reviewed by a quality assessment pathologist (QAP). Any inconsistencies in the diagnoses made by the study laboratory and QA pathologists were resolved by the NTP pathology peer review (PPR). A pathology peer review typically consists of a small group (three to eight) pathologists who examine the lesions around a multiheaded microscope. It is frequently used to review lesions in short-term studies, issues of terminology, or examine single issues that have arisen during a pathology working group (PWG – see below). Final diagnoses for reviewed lesions represent a consensus of the PPR or a consensus between the study laboratory pathologist (SP), NTP pathologist, and QAP. Details of these review procedures have been described, in part, by Maronpot and Boorman[149] and Boorman et al.[150].

A further discussion of pathology review procedures is found in the video[i] (day 2 a.m. at 1 hours, 0 minutes)[3].

Two-year Studies

Study Design

Beginning on GD 5, groups of F0 female rats were housed in reverberation chambers and received whole-body exposures to GSM- or CDMA-modulated cell phone RFR at power levels of 0 (sham control), 1.5, 3, or 6 W/kg, for 9 hours and 10 minutes per day for 7 days per week with continuous cycling of 10 minutes on and 10 minutes off during a period of 18 hours and 20 minutes each day. The sham control animals were housed in reverberation chambers identical to those used for the exposed groups but were not exposed to RFR; shared groups of unexposed rats served as sham controls for both RFR modulations.

F0 female rats were approximately 11 to 14 weeks old upon receipt. GD 1 was defined as day with evidence of mating, and F0 females were received on GD 2 and held in quarantine until GD 4. Animals were randomly assigned to GSM or CDMA exposure groups (56 F0 females/cell phone RFR power level per modulation) with a single group of 56 F0 females serving as the sham control group for both the GSM and CDMA modulations. Randomization was stratified by body weight that produced similar group mean weights (ToxData, version 3.0, PDS Pathology Data Systems, Inc., Basel, Switzerland).

F0 females were housed individually during gestation and with their respective litters during lactation. During gestation, F0 females were weighed on GDs 6, 9, 12, 15, 18, and 21. During lactation, F0 females were weighed on PNDs 1, 4, 7, 14, 17, and 21 and individual F1 pup weights were recorded on PNDs 4, 7, 14, 17, and 21. The day of parturition was considered PND 0. All time-mated females that did not deliver within 3 to 4 days of the anticipated delivery date were euthanized and the uteruses were stained for uterine implantations/ resorptions. On the day after parturition (PND 1), the number of live and dead F1 pups, sex ratio, whole litter weights, and litter weights/sex were recorded.

[i] https://doi.org/10.22427/NTP-VIDEO-43.

F1 litters were standardized on PND 4 to eight pups/litter, preferably with four males and four females each to equalize lactational pressure on F0 females. Litters that did not meet a minimum of eight pups were removed from the study. For continuation of exposure after weaning, three males and three females per litter from 35 F1 litters were randomly selected per exposure group. Weaning occurred over PND 21 and 22 and F1 rats were housed individually. Pups not selected and the F0 females were euthanized with 100% carbon dioxide without necropsy. Weaning marked the beginning of the 2-year chronic phase of the study.

Groups of 105 male and 105 female F1 rats were housed in reverberation chambers and continued to receive whole-body exposures to GSM- or CDMA-modulated cell phone RFR at the same power levels for 9 hours and 10 minutes per day for 7 days per week for 106 to 107 weeks, with continuous cycling of 10 minutes on and 10 minutes off during a period of 18 hours and 20 minutes each day. At 14 weeks, 10 rats per group were randomly selected for interim evaluation and five were designated for genetic toxicity evaluation.

The health of the animals was monitored during the studies according to the protocols of the NTP Sentinel Animal Program (Appendix K). All test results were negative.

Feed and water were available ad libitum. To avoid interference with RFR dosimetry, feed was provided in ceramic (nonmetallic) bowls and water was delivered in an adapted automatic watering system (see video[j], day 1 a.m. at 2 hours, 5 minutes)[3; 148]. Cages were changed weekly and rotated within the racks biweekly; racks were changed biweekly. Further details of animal maintenance are given in Table 1. Information on feed composition and contaminants is provided in Appendix J.

Clinical Examinations and Pathology

Animals were observed twice daily and were weighed initially, twice a week for the first 13 weeks, and at 4-week intervals from weeks 14 to 86, and then every 2 weeks from week 90 until the end of the studies. Clinical observations were recorded once during quarantine and at least every 4 weeks during the studies.

Blood was collected from 10 male and 10 female interim evaluation rats from each group at 14 weeks. Rats were anesthetized with 70% CO2/30% O2 and blood was collected from the retroorbital plexus. Blood for hematology was placed in tubes containing serum separator gel. Hematology parameters were determined on an ADVIA™ 120 automated hematology analyzer (Bayer Diagnostic Division, Tarrytown, NY), except manual hematocrit determinations were performed using a microcentrifuge. Wright Giemsa stained peripheral blood smears were prepared for the assessment of platelet, leukocyte and erythrocyte morphology and enumeration of any nucleated erythrocytes. Samples for clinical chemistry were centrifuged, the serum harvested, and parameters measured using a Synchron LX20 (Beckman Coulter, Indianapolis, IN) analyzer. The hematology and clinical chemistry parameters measured are listed in Table 1. Blood was collected from the remaining five male and five female interim evaluation rats per exposure group at 14 weeks for use in the comet and micronucleus assays; methods for these assays are presented in Appendix E.

[j]https://doi.org/10.22427/NTP-VIDEO-41.

At 14 weeks, samples were collected for sperm motility and count and vaginal cytology evaluations on 10 male and 10 female interim evaluation rats from each group. The parameters evaluated are listed in Table 1. For 16 consecutive days prior to scheduled euthanasia, the vaginal vaults of the females were moistened with saline, if necessary, and samples of vaginal fluid and cells were stained. However, due to inconsistent sample collection and slide staining, an assessment of estrous cyclicity could not be made. Male animals were evaluated for sperm count and motility. The left testis and left epididymis were isolated and weighed. The tail of the epididymis (cauda epididymis) was then removed from the epididymal body (corpus epididymis) and weighed. Modified Tyrode's buffer was applied to slides and a small incision was made at the distal border of the cauda epididymis. The sperm effluxing from the incision were dispersed in the buffer on the slides, and the numbers of motile and nonmotile spermatozoa were counted for five fields per slide by two observers. Following completion of sperm motility estimates, each left cauda epididymis was placed in buffered saline solution. Caudae were finely minced, and the tissue was incubated in the saline solution and then heat fixed at 65°C. Sperm density was then determined microscopically with the aid of a hemacytometer. To quantify spermatogenesis, the testicular spermatid head count was determined by removing the tunica albuginea and homogenizing the left testis in phosphate-buffered saline containing 10% dimethyl sulfoxide. Homogenization-resistant spermatid nuclei were counted with a hemacytometer.

A complete necropsy was conducted on every animal at study termination. For the 14-week interim evaluation rats, the cerebellum, frontal cortex, hippocampus, and liver were collected from five male and five female rats per exposure group for use in the comet assay; methods for this assay are presented in Appendix E. Microscopic examinations were performed on 10 male and 10 female interim evaluation rats in each group at 14 weeks and all core study rats, including those found dead or euthanized moribund. At the interim evaluation, the brain, right and left epididymides, heart, right and left kidneys, liver, lung, right and left ovaries, right and left testes, and thymus were weighed. At necropsy, all organs and tissues were examined for grossly visible lesions, and all major tissues were fixed and preserved in 10% neutral buffered formalin (except eyes, testes, vaginal tunics, and epididymides were first fixed in Davidson's solution or a modified Davidson's solution), processed and trimmed, embedded in paraffin, sectioned to a thickness of 4 to 6 µm, and stained with hematoxylin and eosin for microscopic examination. For all paired organs (e.g., adrenal gland, kidney, ovary), samples from each organ were examined. Tissues examined microscopically are listed in Table 1.

Microscopic evaluations were completed by the study laboratory pathologist, and the pathology data were entered into the Toxicology Data Management System Enterprise. The report, slides, paraffin blocks, residual wet tissues, and pathology data were sent to the NTP Archives for inventory and NTP PPR. All data and materials are available for review upon request from the NTP Archives.

NTP Pathology Review Process

Typically, the initial reading of the slides and the first steps of the pathology review are done by an open, or non-blinded, evaluation by the pathologists involved. This is standard practice for the NTP, as well as the toxicologic pathology industry as a whole, and is in accordance with the recommendations of the Society of Toxicologic Pathologists[151-156]. If issues arise where subtle lesions need to be identified or graded by a blinded evaluation, the pathologist will perform this.

The primary goals of the NTP pathology review are to reach consensus agreement on the diagnosis of all potentially treatment-related findings, confirm the diagnoses of all neoplasms, confirm that consistent and acceptable nomenclature is being used, and confirm the diagnosis of any unusual lesions. There are several elements in this process:

Pathology Data Review (PDR) is a complete review of the pathology data generated by the study laboratory to identify potential target organs and discrepant data and to harmonize terminology. The review involves a multi-disciplinary meeting by the NTP staff and pathology support-contract pathologists to determine the organs and lesions to be reviewed by the quality assessment pathologist (QAP), including all neoplasms.

Audit of Pathology Specimens (APS) is a review of the physical data and residual wet tissues (typically from 10% of the animals) to ensure all gross lesions were evaluated microscopically; of the slides and blocks (typically from 10% of the animals) to ensure correct labeling and quality of sections; and of the submitted reports to ensure accuracy. Also evaluated is whether or not the study laboratory adhered to NTP pathology specifications.

Quality Assessment is a review of the slides of target organs and lesions identified in the PDR by a pathologist from one of the NTP's pathology support contract laboratories not involved with the initial pathology evaluation of the study. All differences in diagnoses between the study pathologist (SP) and QAP are identified in the Differences Report prepared by the QAP. The NTP pathologist attempts to resolve the discrepant diagnoses between the SP and QAP; those that are not resolved are reviewed by the pathology working group (PWG).

Pathology Working Group is a review of selected slides by a panel of pathologists in order to confirm the diagnoses of all treatment-related neoplastic and non-neoplastic lesions and unusual lesions, resolve discrepancies between the SP, QAP, and NTP pathologist, harmonize nomenclature, propose further characterization of the lesions, and address possible mechanisms. The QAP, with oversight from the NTP pathologist, selects slides for the PWG and conducts the PWG. Typically, experts in a particular organ of interest are invited to participate.

Pathology Peer Review is a peer review meeting that convenes to resolve minor issues or issues limited in scope (such as review of short-term studies with limited findings), or review findings of post-PWG actions. Reports are prepared for all these activities. Once the PWG and/or PPR is complete, all written documentation of data changes is reviewed for accuracy and the study data are updated. The pathology data and all written documentation of data changes are then submitted to an outside independent auditor to ensure the accuracy of the updated data. Once all issues identified by the independent auditor have been addressed, the final pathology data tables are generated. For subsequent analyses of the pathology data, the decision of whether to evaluate the diagnosed lesions for each tissue type separately or combined was generally based on the guidelines of Brix et al.[157].

Pathology Review of Cell Phone RFR Studies in Rats

The pathology data presented in this report on cell phone RFR were subjected to a rigorous review process. All elements of the NTP review process were performed, but the sequence of events was altered. Identification of increased incidences of lesions in the brain and heart of male rats in the original study laboratory report warranted a more immediate review than would occur in the standard NTP review process. Malignant glioma of the brain and schwannomas have been

observed in human studies, so the observation of an apparent increase in these same lesions in the rat study prompted the need for an expedited review given the magnitude of human exposure to cell phone RFR and therefore the need to communicate this information to our regulatory partners and the public as soon as possible. Data for the brain and heart were reported to the U.S. Food and Drug Administration and Federal Communications Commission and published in a partial report[158]. For this expedited review, an APS (APS 1) was performed on the hearts and brains. This entailed reviewing the residual wet tissues to ensure all gross lesions were trimmed and processed to slide, reviewing the slides and blocks to ensure quality, and reviewing the data tables. For the expedited review, a QA review was done on all lesions in the central and peripheral nervous systems, all proliferative lesions from the heart, and all schwannomas in other organs. The QAPs, with oversight from the NTP pathologist, selected lesions for PWG review. These lesions were reviewed in several PWGs. Four separate PWGs were held for the brains and hearts. The first PWG (PWG 1), held on January 29, 2016, evaluated the proliferative glial lesions and some reactive glial lesions in the brain, some additional non-neoplastic brain lesions, and schwannomas and Schwann cell hyperplasias in the heart. The second PWG (PWG 2) evaluated schwannomas in the head and neck region and nonglial proliferative lesions in the brain (e.g., granular cell tumors and meningiomas). Due to the volume of slides to be reviewed, PWG 2 was conducted over four sessions (February 11, February 12, March 23, and April 11, 2016). Due to the need for definitive criteria for glial cell and Schwann cell hyperplasia, two additional PWGs composed of experts in neuropathology and cardiovascular pathology, respectively, from around the country were convened. The first (PWG 3) reviewed glial lesions in the brain and was held on February 25, 2016. The second (PWG 4), held on March 3, 2016, reviewed cardiac lesions and schwannomas in organs other than the heart and head and neck region. Subsequent to the release of NTP's Report of Partial Findings[158], the remaining tissues were reviewed. After the Report of Partial Findings, the remaining tissues, and the nonproliferative lesions in the heart, were reviewed (including PWGs 5 and 6). These tissues were subjected to the standard NTP pathology review process as described earlier.

Table 1. Experimental Design and Materials and Methods in the Whole-body Exposure Studies of GSM- and CDMA-modulated Cell Phone RFR

Twenty-eight-day Studies	Two-year Studies
Study Laboratory	
IIT Research Institute (Chicago, IL)	Same as 28-day studies
Strain and Species	
Sprague Dawley (Hsd:Sprague Dawley® SD®) rats	Same as 28-day studies
Animal Source	
Harlan Laboratories, Inc. (Indianapolis, IN)	Same as 28-day studies
Time Held Before Studies	
4 days (F0 females)	3 days (F0 females)
Average Age When Studies Began	
13 to 16 weeks (F0 females)	12 to 15 weeks (F0 females)
Date of First Exposure	
November 1, 2010 (F0 females)	August 8, 2012 (F0 females)

Twenty-eight-day Studies	Two-year Studies
December 9, 2010 (F1 rats)	September 16, 2012 (F1 rats)
Duration of Exposure	
9 hours and 10 minutes per day, over an 18-hour and 20-minute period as exposures cycled between modulations every 10 minutes, 7 days per week for perinatal phase and last week of prechronic phase, and 5 days per week otherwise	9 hours and 10 minutes per day, over an 18-hour and 20-minute period as exposures cycled between modulations every 10 minutes, 7 days per week for 14 weeks (interim evaluation) or 106 (males) or 107 (females) weeks (2-year studies)
Date of Last Exposure	
December 9, 2010 (F0 females) January 6-7, 2011 (F1 groups)	September 16, 2012 (F0 females) December 18 and 20 (males) or 17 and 20 (females), 2012 (interim evaluation) September 15-22 (males) or 22-30 (females), 2014 (chronic study)
Necropsy Dates	
January 6-7, 2011 (F1 groups)	December 18 and 20 (males) or 17 and 20 (females), 2012 (interim evaluation) September 15-22 (males) or 22-30 (females), 2014 (chronic study)
Age at Necropsy	
7 to 8 weeks	Interim: 17 weeks Study termination: 108 to 109 weeks (males) or 109 to 110 weeks (females)
Size of Study Groups	
F0 females: 20 per exposure group F1 rats: 10 males and 10 females	F0 females: 56 per exposure group F1 core study: 90 males and 90 females F1 interim evaluation: 10 males and 10 females F1 genetic toxicity: five males and five females
Method of Distribution	
Animals were distributed randomly into groups of approximately equal initial mean body weights.	Same as 28-day studies
Animals per Cage	
1 except during lactation when pups were housed with nursing dams	Same as 28-day studies
Method of Animal Identification	
F0 females: Tail marking with permanent pen F1 rats: Tail tattoo	Same as 28-day studies
Diet	
Irradiated NIH-07 rodent wafer diet (perinatal phase) or irradiated NTP-2000 rodent wafer diet (prechronic phase) (Zeigler Brothers, Inc., Gardners, PA), available ad libitum, glass jars changed weekly	Same as 28-day studies, except ceramic bowls

Twenty-eight-day Studies	Two-year Studies
Water	
Tap water (Chicago municipal supply) via an adapted automatic watering system (SE Lab Group, Cincinnati, OH), available ad libitum	Same as 28-day studies
Cages	
Solid polycarbonate (Allentown Caging, Allentown, NJ), changed and rotated weekly, except rotated every 2 weeks during parturition	Same as 28-day studies
Bedding	
Certified, irradiated hardwood bedding (P.J. Murphy Forest Products Corp., Montville, NJ), changed weekly	Same as 28-day studies
Racks	
Custom-designed fiberglass cage racks (Ultra, Inc., Milwaukee, WI), changed every 2 weeks	Same as 28-day studies
Reverberation Chambers	
Fully-shielded, stainless steel room equipped with a stainless steel door to eliminate leakage of RFR signals, RFR excitation antennas, and two rotating stirrers; chambers were cleaned at least once weekly.	Same as 28-day studies
Reverberation Chamber Environment	
Temperature: 72° ± 3° F Relative humidity: 50% ± 15% Room incandescent light: 12 hours/day Chamber air changes: at least 10/hour	Same as 28-day studies
Exposure Concentrations	
Time-averaged whole-body SARs of 0 (sham control), 3, 6, and 9 W/kg GSM- or CDMA-modulated cell phone RFR	Time-averaged whole-body SARs of 0 (sham control), 1.5, 3, and 6 W/kg GSM- or CDMA-modulated cell phone RFR
Type and Frequency of Observation	
F0 females: Observed twice daily. Body temperature was measured on GD 6 and within 3.5 minutes of exposure pauses at the end of the second to last “on” cycle on GDs 7, 11, and 16. Body temperature during lactation was measured within 2 minutes of exposure pauses at the end of the second to last “on” cycle on PNDs 1, 4, 7, and 14. Animals were weighed on GDs 6, 9, 12, 15, 18, and 21, and PNDs 1, 4, 7, 14, and 21. Clinical findings were recorded weekly. F1 rats: Observed twice daily. Body temperature was measured on day 8 and within 5 minutes of exposure pauses at the end of the second to last “on” cycle on study days 16, 20, and 27. Animals were weighed during the perinatal phase on PND 1 (litter weights by sex), 4, 7, 14, and 21 and weekly during the prechronic phase. Clinical findings were recorded weekly.	F0 females: Observed twice daily; animals were weighed on GDs 6, 9, 12, 15, 18, and 21, and on PNDs 1, 4, 7, 14, and 21. Clinical findings were recorded on GD 6 through PND 21. F1 rats: Observed twice daily; during perinatal phase, number, sex, and viability status were determined on PND 1. Animals were weighed on PNDs 1 (litter weights by sex), 4, 7, 14, 17, and 21. During the chronic phase, animals were weighed on day 1, twice a week through week 13, at 4-week intervals during weeks 14 to 86, and then every 2 weeks from week 90 until the end of the studies. Clinical findings were recorded at 4-week intervals.

Twenty-eight-day Studies	Two-year Studies
Method of Euthanasia	
Carbon dioxide asphyxiation	Same as 28-day studies
Necropsy	
Necropsies were performed on all rats. Organs weighed were the right adrenal gland, brain, heart, right kidney, liver, lung, right testis, and thymus.	Necropsies were performed on all rats. Organs weighed in 10 rats per exposure group at 14 weeks were the brain, heart, kidney (left and right), liver, lung, ovary (left and right), testis (left and right) with epididymis (left and right), and thymus
Clinical Pathology	
None	Blood was collected from the retroorbital sinus of 10 rats per group at 14 weeks for hematology and clinical chemistry. *Hematology*: hematocrit (auto and manual); hemoglobin concentration; erythrocyte, reticulocyte, nucleated erythrocyte, and platelet counts; mean cell volume; mean cell hemoglobin; mean cell hemoglobin concentration; and leukocyte count and differentials. *Clinical chemistry*: urea nitrogen, creatinine, glucose, total protein, albumin, cholesterol, triglycerides, alanine aminotransferase, alkaline phosphatase, creatine kinase, sorbitol dehydrogenase, and bile acid.
Histopathology	
Complete histopathology was performed on all 0 (sham control) and 9 W/kg groups. In addition to gross lesions and tissue masses, the following tissues were examined: adrenal gland, aorta, bone with marrow, brain, clitoral gland, epididymis, esophagus, eyes, Harderian gland, heart, large intestine (cecum, colon, rectum), small intestine (duodenum, jejunum, ileum), kidney, liver, lung, lymph nodes (mandibular and mesenteric), mammary gland, nose, ovary, pancreas, parathyroid gland, pituitary gland, preputial gland, prostate gland, salivary gland, seminal vesicle, skin, spleen, stomach (forestomach and glandular), testis, thymus, thyroid gland, trachea, urinary bladder, and uterus.	Complete histopathology was performed on 10 F1 rats from each exposure group at 14 weeks, on all rats that died early, and on all rats surviving to the end of the studies. In addition to gross lesions and tissue masses, the following tissues were examined: adrenal gland, aorta, bone with marrow, brain, clitoral gland, esophagus, eyes, Harderian gland, heart, large intestine (cecum, colon, rectum), small intestine (duodenum, jejunum, ileum), kidney, liver, lung with bronchi, lymph nodes (mandibular and mesenteric), mammary gland, muscle, nerve (sciatic, trigeminal, and peripheral), nose, ovary, pancreas, parathyroid gland, pituitary gland, preputial gland, prostate gland, salivary gland, seminal vesicle, skin, spinal cord, spleen, stomach (forestomach and glandular), testis with epididymis, thymus, thyroid gland, trachea, urinary bladder, and uterus.
Sperm Motility and Count and Vaginal Cytology	
None	Spermatid and sperm samples were collected from 10 male rats in each group at 14 weeks. The following parameters were evaluated: spermatid heads per testis and per gram testis, sperm motility, and sperm per cauda epididymis and per gram cauda epididymis. The left cauda, left epididymis, and left testis were weighed. Vaginal samples were collected from 10 females in each group for 16 days prior to the 14-week interim evaluation.

Statistical Methods

P values less than 0.05 were considered statistically significant. Statistical significance is one component of the "weight of evidence" of carcinogenic activity described on page 16 of this Technical Report.

Survival Analyses

The probability of survival was estimated by the product-limit procedure of Kaplan and Meier[159] and is presented in the form of graphs. Animals found dead of other than natural causes or missing were censored; animals dying from natural causes were not censored. Statistical analyses for possible dose-related effects on survival used Cox's[160] method for testing two groups for equality and Tarone's[161] life table test to identify dose-related trends. All reported P values for the survival analyses are two sided.

Calculation of Incidence

The incidences of neoplasms or non-neoplastic lesions are presented in Table A-1, Table A-9, Table B-1, Table B-7, Table C-1, Table C-7, Table D-1, and Table D-8 as the numbers of animals bearing such lesions at a specific anatomic site and the numbers of animals with that site examined microscopically. For calculation of statistical significance, the incidences of most neoplasms (Table A-2, Table B-2, Table C-2, and Table D-2) and all non-neoplastic lesions are given as the numbers of animals affected at each site examined microscopically. However, when macroscopic examination was required to detect neoplasms in certain tissues (e.g., mesentery, pleura, peripheral nerve, skeletal muscle, tongue, tooth, and Zymbal's gland) before microscopic evaluation, the denominators consist of the number of animals that had a gross abnormality. When neoplasms had multiple potential sites of occurrence (e.g., leukemia or lymphoma), the denominators consist of the number of animals on which a necropsy was performed. Table A-2, Table B-2, Table C-2, and Table D-2 also give the survival-adjusted neoplasm rate for each group and each site-specific neoplasm. This survival-adjusted rate (based on the Poly-3 method described below) accounts for differential mortality by assigning a reduced risk of neoplasm, proportional to the third power of the fraction of time on study that it survived, only to site-specific, lesion-free animals that do not reach terminal euthanasia.

Analysis of Neoplasm and Non-neoplastic Lesion Incidences

Statistical analyses of neoplasm and non-neoplastic lesion incidences took into account two features of the data. Some animals did not survive the entire 2 years of the study, so survival differences between groups had to be taken into account. Also, up to three animals per sex were randomly selected from each litter to participate in the study. The statistical analysis of lesion incidences used the Poly-3 test to account for survival differences, with a Rao-Scott adjustment for litter effects, as described below.

The Poly-k test[162-164] was used to assess neoplasm and non-neoplastic lesion prevalence. This test is a survival-adjusted quantal-response procedure that modifies the Cochran-Armitage linear trend test to take survival differences into account. More specifically, this method modifies the denominator in the quantal estimate of lesion incidence to approximate more closely the total number of animal years at risk. For analysis of a given site, each animal is assigned a risk weight. This value is one if the animal had a lesion at that site or if it survived until terminal

euthanasia; if the animal died prior to terminal euthanasia and did not have a lesion at that site, its risk weight is the fraction of the entire study time that it survived, raised to the kth power.

This method yields a lesion prevalence rate that depends only upon the choice of a shape parameter for a Weibull hazard function describing cumulative lesion incidence over time[162]. Unless otherwise specified, a value of k = 3 was used in the analysis of site-specific lesions. This value was recommended by Bailer and Portier[162] following an evaluation of neoplasm onset time distributions for a variety of site-specific neoplasms in control F344 rats and B6C3F1 mice[165]. Bailer and Portier[162] showed that the Poly-3 test gave valid results if the true value of k was anywhere in the range from 1 to 5. A further advantage of the Poly-3 method is that it does not require lesion lethality assumptions. Variation introduced by the use of risk weights, which reflect differential mortality, was accommodated by adjusting the variance of the Poly-3 statistic as recommended by Bieler and Williams[166]. Poly-3 tests used the continuity correction described by Nam[167].

Because up to three pups per sex per litter were in the core study and in the 28-day study, the Poly-3 test was modified to accommodate litter effects using the Rao-Scott approach[168]. Litter effects arise when littermates are more similar to each other than they are to animals from other litters. If intra-litter correlations are present but ignored in the statistical analysis, the variance of the data will be underestimated, leading to P values that are too small. The Rao-Scott approach accounts for litter effects by estimating the ratio of the variance in the presence of litter effects to the variance in the absence of litter effects. This ratio is then used to adjust the sample size downward to yield the estimated variance in the presence of litter effects. The Rao-Scott approach was implemented in the Poly-3 test as recommended by Fung et al.[169], formula $\bar{T}$RS2.

Tests of significance included pairwise comparisons of each dosed group with controls and a test for an overall dose-related trend. Continuity-corrected Rao-Scott-adjusted Poly-3 tests were used in the analysis of lesion incidence and reported P values are one sided. The significance of lower incidences or decreasing trends in lesions is represented as 1–P with the letter N added (e.g., P = 0.99 is presented as P = 0.01N). For neoplasms and non-neoplastic lesions observed without litter structure (e.g., at the interim evaluation), Poly-3 tests that included the continuity correction, but without adjustment for potential litter effects, was used for trend and pairwise comparisons to the control group.

To evaluate litter incidences, the proportions of litters affected by each lesion type were tested among groups. Cochran-Armitage trend tests and Fisher exact tests[170] were used to test for trends and pairwise differences from the control group, respectively.

The statistical analysis of brain gliomas and heart schwannomas reported in the NTP's Report of Partial Findings[158] differed from those presented here. In the previously reported analyses, only gliomas of the brain and schwannomas of the heart were analyzed. Because these are rare tumors and did not occur in more than one animal per litter and because effective statistical methods for litter effect adjustments had not been programmed at that point, Poly-3 and Poly-6 tests were used without adjustment for potential litter effects. The rarity of the tumors and the fact that no litter had more than one animal with the tumors indicated that the adjustment for litter effects would be negligible.

Analysis of Continuous Variables

Two approaches were employed to assess the significance of pairwise comparisons between dosed and control groups in the analysis of continuous variables. In the 28-day and 2-year studies, pup organ and body weight data, and body temperatures, which historically have approximately normal distributions, were analyzed with mixed effects linear models, for trend and pairwise tests with a Dunnett[171]-Hsu[172] adjustment, where litters were the random effect. Body temperatures for dams in all studies were analyzed using the parametric multiple comparison procedures of Dunnett[171] and Williams[173; 174]. At the 14-week interim evaluations in the 2-year studies, hematology, clinical chemistry, spermatid, and epididymal spermatozoal data, which have typically skewed distributions, were analyzed using the non-parametric multiple comparison methods of Shirley[175] (as modified by Williams[176]) and Dunn[177]. Litter sizes, pup survival, implantations, number of resorptions, and proportions of male pups per litter for all studies were also analyzed using these nonparametric methods. For all quantitative endpoints unaffected by litter structure, Jonckheere's test[178] was used to assess the significance of the dose-related trends and to determine at the 0.01 level of significance, whether a trend-sensitive test (Williams' or Shirley's test) was more appropriate for pairwise comparisons than a test that does not assume a monotonic dose-related trend (Dunnett's or Dunn's test). Prior to statistical analysis, extreme values identified by the outlier test of Dixon and Massey[179], for small samples ($n \leq 20$), and Tukey's outer fences method[180], for large samples ($n > 20$), were examined by NTP personnel, and implausible values were eliminated from the analysis.

Post-weaning body weights were measured on three pups per sex per litter in the 2-year study and up to three pups per sex per litter in the 28-day study (with a total of 10 animals per dose group). More than three pups per sex per litter were possible in pre-weaning body weight measurements. The analyses of pup body weights and body weights adjusted for litter size (described below) of these animals took litter effects into account by use of mixed effects regression, where litters were the random effects. Dam body weights, dam body weights adjusted for litter size during gestation, as well as dam body weights during lactation were analyzed with the parametric multiple comparison procedures of Dunnett[171] and Williams[173; 174], depending on whether Jonckheere's test indicated the use of a trend-sensitive test.

P values for these analyses are two sided.

Analysis of Gestational and Fertility Indices

Significances of trends in gestational and fertility indices across dose groups was tested using Cochran-Armitage trend tests. Pairwise comparisons of each dosed group with the control group were conducted using the Fisher exact test. P values for these analyses are two sided.

Body Weight Adjustments

Adjusted dam body weights and adjusted pup body weights were calculated to account for litter size. Dam weights measured during gestation were adjusted for litter size using gestational-day-specific analyses of variance on dam weight as a function of litter size and dose. Dam body weights were adjusted to the overall mean PND 1 total litter size of all groups under analysis, combined, and the residuals from the analyses of covariance were added back in to retain the original variances. Pre-weaning pup body weights were adjusted for PND 1 live litter size using the same analysis of covariance approach, with the additional random effect of litter added to the

models to account for litter effects. Although the same sham control group was used to analyze GSM and CDMA exposed groups, adjusted body weights for the sham control group differ between GSM and CDMA because the overall mean PND 1 live litter size differs between the GSM and CDMA analyses. Post-weaning pup body weights were adjusted using a random effect of litter to account for litter effects without accounting for overall mean PND 1 litter size.

Historical Control Data

The historical control Hsd:Sprague Dawley® SD® rat data used in the current studies are limited to data obtained from three recent finalized studies and differ from the historical control data provided in NTP's Report of Partial Findings[158]. When the NTP's Report of Partial Findings was released very limited data were available describing the prevalence of glial and Schwann cell lesions in Harlan Sprague Dawley rats in NTP studies. Consequently, an effort was made to review control groups from as many comparable studies as possible regardless of whether they had been subjected to peer review.

In NTP's Report of Partial Findings, control groups of male Harlan Sprague Dawley rats from the cell phone RFR studies and nine (for brain) and 12 (for heart) other recently completed NTP studies were tabulated to increase the sample size of rats from which control rates of malignant gliomas of the brain or schwannoma of the heart could be determined. For evaluation of the heart lesions, the 12 studies included black cohosh, resveratrol, sodium tungstate dehydrate, tris(chloroisopropyl) phosphate, indole-3-carbinol, perfluorooctanoic acid, dietary zinc, p-chloro-α,α,α-trifluorotoluene, dibutyl phthalate, 2-hydroxy-4-methoxy-benzophenone, and diethylhexyl phthalate (2 studies). Three fewer studies were available for evaluation of brain lesions than for the evaluation of heart lesions because of a recent change by the NTP to increase the standard number of examined sections from three to seven. Because the sectioning in these three studies differed, the studies with three brain sections (indole-3-carbinol, perfluorooctanoic acid, and dietary zinc) were excluded from evaluation of brain lesions. For studies in which the in-life portion was completed, but the final pathology data were not yet available, special reviews of the control rat brains for malignant gliomas and hearts for schwannomas were performed.

Quality Assurance Methods

The 28-day and 2-year studies were conducted in compliance with Food and Drug Administration Good Laboratory Practice Regulations[181]. In addition, the 28-day and 2-year study reports were audited retrospectively by an outside independent QA contractor against study records submitted to the NTP Archives. Separate audits covered completeness and accuracy of the pathology data, pathology specimens, final pathology tables, and a draft of this NTP Technical Report. Audit procedures and findings are presented in the reports and are on file at NIEHS. The audit findings were reviewed and assessed by NTP staff, and all comments were resolved or otherwise addressed during the preparation of this Technical Report.

Genetic Toxicology

The genetic toxicity of GSM- and CDMA-modulated cell phone RFR was assessed by measuring the frequency of micronucleated erythrocytes in peripheral blood and DNA damage in five different tissues of male and female rats following 14 weeks of exposure. Micronuclei (literally "small nuclei" or Howell-Jolly bodies) are biomarkers of induced structural or numerical chromosomal alterations and are formed when acentric fragments or whole chromosomes fail to

incorporate into either of two daughter nuclei during cell division[182; 183]. The alkaline (pH > 13) comet assay[184] (also known as the single cell gel electrophoresis assay) detects DNA damage in any of a variety of eukaryotic cell types[185-188]; cell division is not required. The type of DNA damage detected includes nicks, adducts, strand breaks, and abasic sites that are converted to DNA strand breaks after treatment of cells in an alkaline (pH > 13) solution. Transient DNA strand breaks generated by the process of DNA excision repair may also be detected. DNA damage caused by crosslinking agents has been detected as a reduction of DNA migration[189; 190]. The fate of the DNA damage detected by the comet assay is varied; most of the damage is rapidly repaired resulting in no sustained impact on the tissue but some may result in cell death or may be incorrectly processed by repair proteins and result in a fixed mutation or chromosomal alteration. The detailed protocols for these studies and the results are given in Appendix E.

The genetic toxicity studies have grown out of an earlier effort by the NTP to develop a comprehensive database permitting a critical anticipation of a test article's carcinogenicity in experimental animals based on the results from a number of in vitro and in vivo short-term tests measuring functionally distinct genotoxicity endpoints. The short-term tests were originally developed to clarify proposed mechanisms of chemical-induced DNA damage based on the relationship between electrophilicity and mutagenicity[191] and the somatic mutation theory of cancer[192; 193]. However, it should be noted that not all cancers arise through genotoxic mechanisms, and in these studies, the test article is not a chemical. Many studies have established the genotoxicity of some forms of radiation including, for example, ultraviolet radiation and X-ray radiation, which are both forms of ionizing radiation. Because exposure to RFR requires specialized and highly technical exposure protocols, only in vivo biomarkers associated with genotoxicity could be investigated.

Clearly positive results in long-term peripheral blood micronucleus tests have high predictivity for rodent carcinogenicity; a weak response in one sex only or negative results in both sexes in this assay do not correlate well with either negative or positive results in rodent carcinogenicity studies[194]. The relationship between comet assay results and rodent carcinogenicity was investigated previously and a close association was observed[195]; however, this assay is best employed as a hazard identification assay. Because of the theoretical and observed associations between induced genetic damage and adverse effects in somatic and germ cells, the determination of in vivo genetic effects is important to the overall understanding of the risks associated with exposure to a particular test article.

Further discussion of the genetic toxicology assays used in these studies can be found in the video[k] (day 2 a.m. at 2 hours, 48 minutes)[3].

[k] https://doi.org/10.22427/NTP-VIDEO-43.

Results

Data Availability

The National Toxicology Program (NTP) evaluated all study data. Data relevant for evaluating toxicological findings are presented here. All study data are available in the NTP Chemical Effects in Biological Systems (CEBS) database: https://doi.org/10.22427/NTP-DATA-TR-595.

GSM

Twenty-eight-day Study

Perinatal Study

No exposure-related effects were observed on survival or littering rates (littering/pregnant ratio) (Table 2). Gestation body weights were unaffected by exposure to GSM-modulated cell phone RFR by pairwise comparison (Table 3). A significant negative trend was observed in gestation day (GD) 21 body weights that was likely due to reduced body weight gain in late gestation in the 9 W/kg group (Table 3). There was an overall (GD 6 to GD 21) lower body weight gain of 9% in the 9 W/kg group compared to that of the sham controls.

Table 2. Summary of Disposition during Perinatal Exposure and F_1 Allocation in the 28-day Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Time-mated Females	20	20	20	20
Pregnant Females	20	19	18	20
Nonpregnant Females	0	1	2	0
Pregnant Dams Not Delivering	0	0	0	0
Died	0	0	0	0
Littered	20	19	18	20
Pregnant/Mated Percentage[a]	100.0%	95.0%	90.0%	100.0%
Littered/Pregnant Percentage[a]	100.0%	100.0%	100.0%	100.0%
Litters Removed (Insufficient Size) (PND 4)	0	0	0	0
Litters Post Standardization (PND 4)	20	19	18	20
Weaned/Sex (PND 21)[b]	30	30	30	30

[a]Number in the numerator as proportion of the number in the denominator was tested using the Fisher exact test for pairwise comparisons against the sham control group.
[b]Total number of weaned animals per sex from 10 litters.

Table 3. Mean Body Weights and Mean Body Weight Gains of F_0 Female Rats during Gestation in the 28-day Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Gestation Day				
6	238.3 ± 2.2 (20)[b]	236.7 ± 2.4 (19)	238.8 ± 2.6 (18)	238.3 ± 2.3 (20)
9	250.7 ± 2.4 (20)	249.8 ± 2.2 (19)	251.4 ± 2.5 (18)	249.8 ± 2.5 (20)
12	266.2 ± 2.5 (20)	263.0 ± 2.3 (19)	265.9 ± 2.6 (18)	264.2 ± 2.6 (20)
15	282.5 ± 2.9 (20)	280.7 ± 2.8 (19)	283.0 ± 2.9 (18)	281.3 ± 2.5 (20)
18	319.3 ± 3.0 (20)	316.9 ± 3.2 (19)	319.4 ± 3.6 (18)	313.6 ± 2.6 (20)
21	366.4 ± 4.3 (20) ▲▲	360.2 ± 4.8 (19)	363.6 ± 4.5 (18)	354.8 ± 3.3 (20)
Gestation Day Interval				
6–9	12.5 ± 1.2 (20)	13.1 ± 0.9 (19)	12.6 ± 1.1 (18)	11.5 ± 0.5 (20)
9–12	15.5 ± 1.1 (20)	13.3 ± 0.8 (19)	14.5 ± 0.6 (18)	14.4 ± 0.7 (20)
12–15	16.3 ± 1.0 (20)	17.7 ± 1.0 (19)	17.1 ± 0.5 (18)	17.1 ± 0.6 (20)
15–18	36.7 ± 0.9 (20) ▲▲	36.2 ± 1.3 (19)	36.4 ± 1.1 (18)	32.3 ± 0.8 (20)**
18–21	47.1 ± 1.8 (20) ▲▲	43.3 ± 2.0 (19)	44.2 ± 1.3 (18)	41.2 ± 1.5 (20)*
6–21	128.1 ± 3.3 (20) ▲▲	123.5 ± 4.3 (19)	124.8 ± 3.0 (18)	116.5 ± 2.6 (20)*

▲▲Significant trend ($P \leq 0.01$).
*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.
[b]Number of dams.

Total and live litter size on postnatal day (PND) 1 was unaffected by exposure and there was no statistically significant effect on live litter size throughout lactation (Table 4). However, there were higher numbers of dead pups in the exposed groups from PND 1 to 4 and single incidences in the 6 and 9 W/kg groups from PND 5 to 21 (Table 5). The number of dead pups per litter was significantly increased from PND 1 to 4 in addition to a decreased survival ratio in the 9 W/kg group.

Table 4. Mean Number of Surviving F_1 Male and Female Rats during Lactation in the 28-day Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Total Pups per Litter				
PND 1	11.95 ± 0.38 (20)[b]	11.74 ± 0.55 (19)	12.78 ± 0.37 (18)	12.40 ± 0.48 (20)
Live Pups per Litter				
PND 1	11.90 ± 0.39 (20)	11.63 ± 0.54 (19)	12.78 ± 0.37 (18)	12.20 ± 0.51 (20)
PND 4 (preculling)	11.85 ± 0.39 (20)	11.53 ± 0.54 (19)	12.44 ± 0.37 (18)	11.45 ± 0.51 (20)
PND 4 (postculling)	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.94 ± 0.06 (18)	7.90 ± 0.07 (20)
PND 7	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.89 ± 0.08 (18)	7.90 ± 0.07 (20)
PND 10	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.89 ± 0.08 (18)	7.90 ± 0.07 (20)
PND 14	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.89 ± 0.08 (18)	7.85 ± 0.08 (20)

	Sham Control	3 W/kg	6 W/kg	9 W/kg
PND 17	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.89 ± 0.08 (18)	7.85 ± 0.08 (20)
PND 21	7.95 ± 0.05 (20)	7.84 ± 0.28 (19)	7.89 ± 0.08 (18)	7.85 ± 0.08 (20)
Live Males per Litter				
PND 1	6.10 ± 0.42 (20)	5.95 ± 0.39 (19)	6.72 ± 0.52 (18)	5.80 ± 0.55 (20)
PND 4 (preculling)	6.05 ± 0.41 (20)	5.74 ± 0.38 (19)	6.50 ± 0.52 (18)	5.45 ± 0.54 (20)
PND 4 (postculling)	4.00 ± 0.15 (20)	3.95 ± 0.18 (19)	3.94 ± 0.13 (18)	3.75 ± 0.24 (20)
Live Females per Litter				
PND 1	5.80 ± 0.42 (20)	5.68 ± 0.36 (19)	6.06 ± 0.45 (18)	6.40 ± 0.60 (20)
PND 4 (preculling)	5.80 ± 0.43 (20)	5.79 ± 0.37 (19)	5.94 ± 0.40 (18)	6.00 ± 0.50 (20)
PND 4 (postculling)	3.95 ± 0.14 (20)	3.89 ± 0.11 (19)	4.00 ± 0.14 (18)	4.15 ± 0.24 (20)

[a]All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.
[b]Number of dams.

Table 5. Offspring Mortality and Survival Ratio of Rats during Lactation in the 28-day Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR[a]

Pup Survival per Litter	Sham Control	3 W/kg	6 W/kg	9 W/kg
Total Dead PND 1–4[b]	2 (238/20)	4 (221/19)	6 (230/18)	19 (244/20)
Total Dead PND 5–21	0 (159/20)	0 (149/19)	1 (143/18)	1 (158/20)
Dead/Litter PND 1–4	0.100 ± 0.069 (20)▲▲	0.211 ± 0.123 (19)	0.333 ± 0.229 (18)	0.950 ± 0.223 (20)**
Dead/Litter PND 4–21	0.000 ± 0.000 (20)	0.000 ± 0.000 (19)	0.056 ± 0.056 (18)	0.050 ± 0.050 (20)
Survival Ratio PND 1–4[c]	0.996 ± 0.004 (20)▲▲	0.991 ± 0.006 (19)	0.976 ± 0.016 (18)	0.940 ± 0.014 (20)**
Survival Ratio PND 4–21[d]	1.000 ± 0.000 (20)	1.000 ± 0.000 (19)	0.993 ± 0.007 (18)	0.994 ± 0.006 (20)

▲▲Significant trend ($P \leq 0.01$).
**Significantly different ($P \leq 0.01$) from the sham control group.
[a]All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.
[b]Number of pups/number of dams.
[c]Number of pups preculling on PND 4/total number of viable pups on PND 1 (does not include pups dead on PND 1).
[d]Number of pups alive on PND 21/number of pups at postculling on PND 4.

Exposed dams had decreased weight gain during lactation (PND 1 through 21), and maternal body weights of the 9 W/kg group were up to 11% lower than those of the sham controls (Table 6). Combined F_1 body weights were 8% lower starting on PND 1 in the 9 W/kg group when adjusted for litter size (Table 7). As lactation progressed, the adjusted pup weights (combined) were up to 17% lower in the 9 W/kg group and up to 8% lower in the 6 W/kg group compared to sham controls. The magnitude of the effect was consistent between males and females.

Table 6. Mean Body Weights and Mean Body Weight Gains of F_0 Female Rats during Lactation in the 28-day Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Postnatal Day				
1	272.7 ± 2.7 (20)b	267.8 ± 2.3 (19)	271.6 ± 2.7 (18)	263.5 ± 2.5 (20)*
4	263.8 ± 3.6 (20)	265.1 ± 2.9 (19)	269.9 ± 4.2 (18)	264.1 ± 2.2 (20)
7	284.1 ± 2.7 (20)▲▲	280.3 ± 1.9 (19)	281.8 ± 2.7 (18)	270.0 ± 2.3 (20)**
14	292.4 ± 2.6 (20)▲▲	289.9 ± 2.9 (19)	286.4 ± 2.8 (18)	266.1 ± 2.6 (20)**
21	279.7 ± 3.5 (20)▲▲	267.3 ± 3.7 (19)**	265.8 ± 3.6 (11)**	248.5 ± 2.3 (20)**
Postnatal Day Interval				
1–4	−8.9 ± 3.2 (20)▲	−2.7 ± 2.8 (19)	−1.7 ± 2.8 (18)	0.6 ± 1.5 (20)*
4–7	20.2 ± 2.3 (20)▲▲	15.2 ± 2.0 (19)	−12.0 ± 2.6 (18)**	5.9 ± 1.1 (20)**
7–14	8.3 ± 1.4 (20)▲▲	9.6 ± 2.1 (19)	4.6 ± 2.0 (18)	−4.0 ± 1.5 (20)**
4–21	−12.7 ± 3.5 (20)	−22.7 ± 3.2 (19)*	−16.9 ± 3.1 (11)	−17.6 ± 1.7 (20)
1–21	7.0 ± 3.8 (20)▲▲	−0.5 ± 2.6 (19)	−1.9 ± 2.5 (11)	−15.0 ± 1.8 (20)**

▲Significant trend ($P \leq 0.05$).
▲▲$P \leq 0.01$.
*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's test (trend) and Williams' or Dunnett's (pairwise) tests.
[b]Number of dams.

Table 7. Adjusted Mean Body Weights of F_1 Male and Female Rats during Lactation in the 28-day Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Adjusted Male Live Pup Weight				
PND 1b	6.70 ± 0.10 (20)▲▲[c]	6.71 ± 0.09 (19)	6.46 ± 0.10 (18)	5.99 ± 0.09 (20)▲▲
PND 4 (preculling)	9.68 ± 0.14 (121/20)**[d]	9.59 ± 0.13 (109/19)	9.08 ± 0.13 (117/18)*	8.09 ± 0.19 (108/20)**
PND 4 (postculling)	9.75 ± 0.13 (81/20)**	9.67 ± 0.14 (74/19)	9.18 ± 0.13 (71/18)*	8.20 ± 0.16 (75/20)**
PND 7	16.22 ± 0.19 (81/20)**	15.56 ± 0.21 (74/19)	14.84 ± 0.28 (71/18)**	13.06 ± 0.31 (75/20)**
PND 14	31.52 ± 0.42 (81/20)**	31.29 ± 0.31 (74/19)	30.05 ± 0.42 (71/18)*	26.63 ± 0.47 (75/20)**
PND 21	53.19 ± 0.72 (81/20)**	52.86 ± 0.61 (74/19)	51.20 ± 0.71 (71/18)	45.52 ± 0.94 (75/20)**
Adjusted Female Live Pup Weight				
PND 1	6.28 ± 0.10 (20)▲▲	6.43 ± 0.07 (19)	6.17 ± 0.11 (18)	5.89 ± 0.09 (20)▲▲
PND 4 (preculling)	9.09 ± 0.15 (116/20)**	9.16 ± 0.14 (110/19)	8.76 ± 0.16 (107/18)	7.95 ± 0.18 (121/20)**
PND 4 (postculling)	9.13 ± 0.16 (79/20)**	9.23 ± 0.14 (73/19)	8.74 ± 0.16 (73/18)	8.11 ± 0.17 (83/20)**
PND 7	15.17 ± 0.24 (79/20)**	14.91 ± 0.23 (73/19)	13.96 ± 0.30 (72/18)**	12.95 ± 0.31 (83/20)**
PND 14	29.76 ± 0.40 (79/20)**	30.03 ± 0.36 (73/19)	28.67 ± 0.48 (72/18)	26.41 ± 0.42 (82/20)**
PND 21	49.78 ± 0.73 (79/20)**	49.45 ± 0.58 (73/19)	48.47 ± 0.76 (72/18)	44.25 ± 0.87 (82/20)**

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Adjusted Combined Live Pup Weight				
PND 1	6.48 ± 0.10 (20)▲▲	6.57 ± 0.08 (19)	6.33 ± 0.10 (18)	5.95 ± 0.08 (20)▲▲
PND 4 (preculling)	9.38 ± 0.13 (237/20)**	9.38 ± 0.14 (219/19)	8.94 ± 0.13 (224/18)	8.05 ± 0.17 (229/20)**
PND 4 (postculling)	9.44 ± 0.13 (160/20)**	9.45 ± 0.14 (147/19)	8.96 ± 0.14 (144/18)	8.16 ± 0.16 (158/20)**
PND 7	15.69 ± 0.19 (160/20)**	15.24 ± 0.21 (147/19)	14.40 ± 0.27 (143/18)**	13.00 ± 0.30 (158/20)**
PND 14	30.62 ± 0.39 (160/20)**	30.66 ± 0.32 (147/19)	29.35 ± 0.43 (143/18)	26.51 ± 0.43 (157/20)**
PND 21	51.46 ± 0.67 (160/20)**	51.17 ± 0.55 (147/19)	49.84 ± 0.70 (143/18)	44.87 ± 0.88 (157/20)**

▲▲Significantly different ($P \leq 0.01$) for PND 1 endpoint (statistical significance in the sham control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant pairwise comparison against the sham control group).
*Significantly different ($P \leq 0.05$) for PNDs after PND 1 endpoints (statistical significance in the sham control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant pairwise comparison against the sham control group).
**$P \leq 0.01$.
[a]Body weights in grams. Data are displayed as mean ± standard error. PND = postnatal day. Values listed as PND 1 refer to the total pup weight divided by the number of pups in litter at PND 1, and the statistical analysis performed is by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests. Values listed for all other days refer to individual pups, and the statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as random effect with Dunnett-Hsu adjustment method. Individual pup body weights first adjusted for live PND 1 litter size via the analysis of covariance.
[b]Values listed as PND 1 refer to the total pup weight divided by the number of pups in litter at PND 1.
[c]Number of dams.
[d]Number of pups/number of dams.

Postnatal Study

All rats survived to the end of the study (Table 8). There were lower mean body weights in the male 6 (6% to 9%) and 9 (16% to 19%) W/kg groups compared to sham controls at all time points including terminal sacrifice (Table 8 and Figure 6). Mean body weight gains were also lower in these groups (10% to 16%) (Data not presented). In 3 W/kg males, mean body weights were lower on day 22 (5%) and at terminal sacrifice (7%), but body weight gains were comparable to that of the sham controls. In females, mean body weights were lower on days 1, 8, 15, and 22 in the 9 W/kg group (8% to 11%) and on days 8 and 15 in the 6 W/kg group (5%). However, mean body weights at terminal sacrifice and mean body weight gains in all exposed female groups were similar to those of the sham controls. There were no notable clinical observations in either sex during the study.

Table 8. Mean Body Weights and Survival of Rats Exposed to GSM-modulated Cell Phone RFR for 28 Days

Day	Sham Control		3 W/kg			6 W/kg			9 W/kg		
	Av. Wt. (g)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt.(g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
Male											
1	60.8	10	60.3	99.1	10	57.4	94.3	11	50.8	83.5	10
8	94.9	10	91.3	96.3	10	87.1	91.9	11	77.3	81.5	10
15	144.5	10	137.2	94.9	10	132.4	91.6	11	117.7	81.4	10
22	195.3	10	184.7	94.5	10	178.0	91.1	11	158.6	81.2	10
29	248.7	10	231.3	93.0	10	227.0	91.3	10	204.6	82.3	10
Female											
1	55.9	10	54.4	97.3	10	53.7	96.1	10	49.6	88.8	10
8	83.1	10	80.0	96.2	10	78.9	94.9	10	73.6	88.5	10
15	119.8	10	114.8	95.8	10	113.9	95.1	10	107.5	89.7	10
22	146.5	10	142.9	97.6	10	143.1	97.7	10	134.6	91.9	10
30	166.5	10	163.1	98.0	10	168.0	100.9	10	155.7	93.5	10

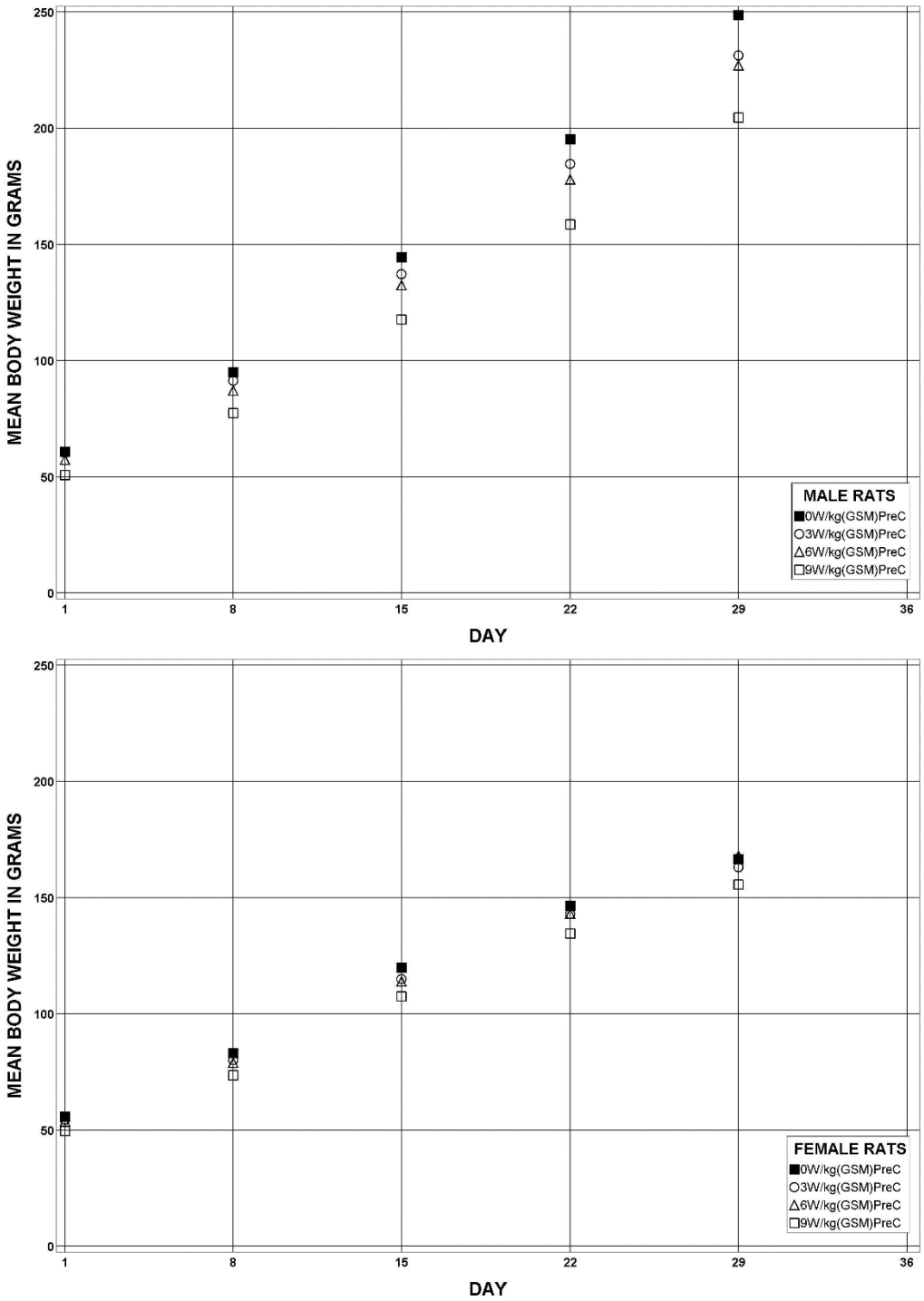

Figure 6. Growth Curves for Rats Exposed to GSM-modulated Cell Phone RFR for 28 Days

The average temperature over gestation (GD 7 through 16) was increased compared to sham controls in the 6 and 9 W/kg groups by 0.4 and 0.5 degrees, respectively (Table 9). During lactation, the average (LD 1 through 14) temperature was also increased in the 6 and 9 W/kg groups by 0.7 and 1.0 degrees, respectively. In the F1 offspring, average (study day 16 through

27; ~PND 37 through 48) body temperatures were decreased by 0.5 degrees in the 3 W/kg male group and by 0.9 degrees in the 6 W/kg female group (Table 9 and Table G-1).

The relative brain and testis weights in exposed male groups were increased compared to the sham control group, but this was considered to be due to the lower necropsy body weights (8%, 10%, and 20% lower than sham controls in the 3, 6, and 9 W/kg groups, respectively) and not an exposure-related effect (Table G-2). Significant decreases in absolute heart weights were observed in 6 and 9 W/kg males (14% and 22%, respectively), but the relative heart weights were not affected. Similarly, absolute right kidney and liver weights were decreased in 9 W/kg males (24% and 19%, respectively), but without corresponding effects in the relative weights. These decreases in organ weights could be related to the lower body weights in males. No effects in organ weights were observed in female rats.

In females, there were increased incidences of chronic progressive nephropathy in the kidney of 3 and 9 W/kg groups (sham controls, 0/10; 3 W/kg, 4/10; 6 W/kg, 3/10; 9 W/kg, 4/10) compared to the sham controls. The severity of these lesions was minimal (1.0). Chronic progressive nephropathy was characterized by scattered tubular segments with basophilic epithelial cells with crowded nuclei, slightly thickened basement membranes, and occasional mononuclear inflammatory cells. There were no exposure-related renal lesions in male rats.

Exposure Level Selection Rationale: Based on pup mortality, reduced maternal and pup body weights, increased F0 dam body temperature measurements at 9 W/kg in the 28-day studies, and increased body temperature in adult rats at ≥8 W/kg in the thermal pilot studies[146], the highest exposure level selected for the 2-year studies was 6 W/kg. In the thermal pilot studies and in the 28-day study, exposure to 6 W/kg resulted in some increases in core body temperature, but these increases were less than 1°C. Therefore, 6 W/kg would provide an exposure adequate to challenge the animals without causing excessive heating or disruption of the thermoregulatory process. The lowest exposure level selected for the 2-year studies was 1.5 W/kg, which is close to the 1.6 W/kg maximum output limit for cell phone devices in the United States.

Table 9. Mean Body Temperatures of Rats Exposed to GSM-modulated Cell Phone RFR for 28 Days[a]

	Sham Control		3 W/kg		6 W/kg		9 W/kg	
Day	Temperature (°C)	No. Measured	Temperature (°C)	No. Measured	Temperature (°C)	No. Measured	Temperature (°C)	No. Measured
F0 Female[b]								
GD 6	36.7 ± 0.1	10[c]	37.4 ± 0.2**	9	36.5 ± 0.1	9	36.8 ± 0.2	10
GD 7	36.6 ± 0.1▲▲	10	36.7 ± 0.1	9	37.1 ± 0.1*	9	37.2 ± 0.1**	10
GD 11	36.7 ± 0.2▲▲	10	36.5 ± 0.1	9	37.1 ± 0.1	9	37.2 ± 0.1*	10
GD 16	36.5 ± 0.1▲▲	10	36.5 ± 0.1	9	36.8 ± 0.1	9	37.0 ± 0.1**	10
GD 7–16[d]	36.6 ± 0.1▲▲	10	36.6 ± 0.1	9	37.0 ± 0.1**	9	37.1 ± 0.0**	10
LD 1	37.7 ± 0.1▲▲	10	37.8 ± 0.1	9	38.1 ± 0.2	9	38.4 ± 0.2**	10
LD 4	36.7 ± 0.1▲▲	10	37.1 ± 0.2	9	37.5 ± 0.2**	9	37.9 ± 0.1**	10
LD 7	36.8 ± 0.2	10	37.1 ± 0.2	9	37.2 ± 0.2	9	37.2 ± 0.2	10

	Sham Control		3 W/kg		6 W/kg		9 W/kg	
Day	Temperature (°C)	No. Measured	Temperature (°C)	No. Measured	Temperature (°C)	No. Measured	Temperature (°C)	No. Measured
LD 14	36.9 ± 0.2▲▲	10	37.1 ± 0.1	9	37.8 ± 0.2**	9	38.3 ± 0.2**	8
LD 1–14[d]	37.0 ± 0.1▲▲	10	37.3 ± 0.1	9	37.7 ± 0.1**	9	38.0 ± 0.1**	10
F1 Male[e]								
16	37.4 ± 0.2	4	37.0 ± 0.1	4	37.2 ± 0.2	4	37.2 ± 0.1	4
20	37.6 ± 0.1	4	37.0 ± 0.1**	4	37.2 ± 0.2	4	37.4 ± 0.1	4
27	37.3 ± 0.2	4	37.0 ± 0.1	4	37.2 ± 0.0	3	37.4 ± 0.1	4
16–27[d]	37.5 ± 0.1	4	37.0 ± 0.0*	4	37.2 ± 0.1	4[f]	37.4 ± 0.1	4
F1 Female[e]								
16	38.0 ± 0.3	4	37.0 ± 0.2**	4	37.0 ± 0.1*	4	37.4 ± 0.1	4
20	38.1 ± 0.2	4	37.6 ± 0.1	4	37.0 ± 0.1**	4	37.6 ± 0.1	4
27	37.9 ± 0.2	4	37.8 ± 0.3	4	37.3 ± 0.3	4	37.6 ± 0.0	4
16–27[d]	38.0 ± 0.2▲	4	37.4 ± 0.1	4	37.1 ± 0.1**	4	37.5 ± 0.0	4

▲Significant trend ($P \leq 0.05$).
▲▲$P \leq 0.01$.
*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Temperatures are given as mean ± standard error. GD = gestation day; LD = lactation day.
[b]Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.
[c]For F_0 females, number measured refers to individual animals; for F_1 pups, number measured refers to litters.
[d]Average of days.
[e]Statistical analysis for linear trends was performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method.
[f]There were three litters on day 27.

Two-year Study

Perinatal Exposure

No exposure-related effects were observed on pregnancy status, maternal survival, or the percent of pregnant animals that littered (Table 10). Maternal body weights during gestation were similar to those of the sham control group (Table 11). Body weight gains were generally unaffected across time intervals except in the 6 W/kg group at the GD 5 through 18 interval where body weight gain was 10% lower than that of the sham control group and the GD 6 through 21 interval where body weight gain was 7% lower than that of the sham control group.

Table 10. Summary of Disposition during Perinatal Exposure and F_1 Allocation in the Two-year Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Time-mated Females	56	56	56	56
Pregnant Females	52	50	50	52
Nonpregnant Females	4	6	6	4
Pregnant Dams Not Delivering	2	3	3	4

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Died[a]	1	0	0	0
Littered	50	47	47	48
Pregnant/Mated Percentage[b]	92.9%	89.3%	89.3%	92.9%
Littered/Pregnant Percentage[b]	96.2%	94.0%	94.0%	92.3%
Litters Removed (Insufficient Size)	2	4	5	2
Litters Post Standardization	48	43	42	46
Weaned/Sex[c]	105	105	105	105

[a]One pregnant female died on GD 25 with pups in uterus.
[b]Number in the numerator as proportion of the number in the denominator was tested using the Fisher exact test for pairwise comparison against sham control group.
[c]Total number of weaned animals per sex from 35 litters.

Table 11. Mean Body Weights and Mean Body Weight Gains of F_0 Female Rats during Gestation in the Two-year Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Gestation Day				
6	238.4 ± 1.4 (51)b	239.9 ± 1.4 (47)	239.0 ± 1.4 (47)	238.9 ± 1.3 (48)
9	256.2 ± 1.6 (51)	256.3 ± 1.6 (47)	254.9 ± 1.6 (47)	254.7 ± 1.3 (48)
12	270.5 ± 1.6 (51)	270.4 ± 1.7 (47)	269.2 ± 1.6 (47)	268.1 ± 1.4 (48)
15	290.0 ± 1.9 (51)	289.7 ± 2.0 (47)	288.8 ± 1.8 (47)	287.6 ± 1.6 (48)
18	332.7 ± 2.3 (51)▲	329.8 ± 2.6 (47)	328.9 ± 2.2 (47)	326.1 ± 2.1 (48)
21	380.2 ± 2.8 (51)▲	376.9 ± 3.6 (47)	374.6 ± 3.5 (47)	371.2 ± 3.0 (48)
Gestation Day Interval				
6–9	17.7 ± 0.8 (51)	16.4 ± 0.6 (47)	15.9 ± 0.6 (47)	15.8 ± 0.5 (48)
9–12	14.3 ± 0.6 (51)	14.1 ± 0.5 (47)	14.2 ± 0.5 (47)	13.4 ± 0.5 (48)
12–15	19.6 ± 0.6 (51)	19.3 ± 0.8 (47)	19.7 ± 0.6 (47)	19.5 ± 0.6 (48)
15–18	42.7 ± 1.0 (51)▲▲	40.2 ± 1.1 (47)	40.1 ± 0.9 (47)	38.5 ± 0.9 (48)**
18–21	47.5 ± 1.0 (51)	47.1 ± 1.5 (47)	45.6 ± 1.5 (47)	45.1 ± 1.3 (48)
6–21	141.7 ± 2.2 (51)▲▲	137.0 ± 3.3 (47)	135.5 ± 2.9 (47)	132.3 ± 2.6 (48)*

▲Significant trend ($P \leq 0.05$).
▲▲$P \leq 0.01$.
*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.
[b]Number of dams.

Total litter size on PND 1 and live litter size at all time points were unaffected by exposure with no effects observed in pup mortality or survival ratio in early postnatal development (PND 1 through 4) or thereafter (PND 4 through 21) (Table 12 and Table 13).

Table 12. Mean Number of Surviving F_1 Male and Female Rats during Lactation in the Two-year Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Total Pups per Litter				
PND 1	12.76 ± 0.32 (50)[b]	12.06 ± 0.43 (47)	12.26 ± 0.51 (47)	12.31 ± 0.39 (48)
Live Pups per Litter				
PND 1	12.56 ± 0.40 (50)	12.04 ± 0.43 (47)	12.23 ± 0.51 (47)	12.29 ± 0.39 (48)
PND 4 (preculling)	12.73 ± 0.30 (48)	12.65 ± 0.26 (43)	12.98 ± 0.27 (42)	12.41 ± 0.32 (46)
PND 4 (postculling)	8.00 ± 0.00 (48)	8.00 ± 0.00 (43)	8.00 ± 0.00 (42)	8.00 ± 0.00 (46)
PND 7	8.00 ± 0.00 (48)	7.98 ± 0.02 (43)	7.95 ± 0.03 (42)	7.98 ± 0.02 (46)
PND 10	8.00 ± 0.00 (48)	7.98 ± 0.02 (43)	7.93 ± 0.04 (42)	7.98 ± 0.02 (46)
PND 14	8.00 ± 0.00 (48)	7.98 ± 0.02 (43)	7.93 ± 0.04 (42)	7.98 ± 0.02 (46)
PND 17	8.00 ± 0.00 (48)	7.98 ± 0.02 (43)	7.93 ± 0.04 (42)	7.98 ± 0.02 (46)
PND 21	8.00 ± 0.00 (48)	7.98 ± 0.02 (43)	7.93 ± 0.04 (42)	7.98 ± 0.02 (46)
Live Males per Litter				
PND 1	6.20 ± 0.30 (50)	6.00 ± 0.34 (47)	6.11 ± 0.38 (47)	6.02 ± 0.32 (48)
PND 4 (preculling)	6.33 ± 0.28 (48)	6.35 ± 0.30 (43)	6.62 ± 0.32 (42)	6.15 ± 0.30 (46)
PND 4 (postculling)	3.96 ± 0.05 (48)	3.98 ± 0.07 (43)	4.00 ± 0.07 (42)	4.02 ± 0.10 (46)
Live Females per Litter				
PND 1	6.36 ± 0.28 (50)	6.04 ± 0.32 (47)	6.13 ± 0.33 (47)	6.27 ± 0.33 (48)
PND 4 (preculling)	6.40 ± 0.25 (48)	6.30 ± 0.26 (43)	6.36 ± 0.30 (42)	6.26 ± 0.34 (46)
PND 4 (postculling)	4.04 ± 0.05 (48)	4.02 ± 0.07 (43)	4.00 ± 0.07 (42)	3.98 ± 0.10 (46)

[a]All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.
[b]Number of dams.

Table 13. Offspring Mortality and Survival Ratio of Rats during Lactation in the Two-year Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR[a]

Pup Survival per Litter	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Total Dead PND 1–4[b]	19 (628/49)[c]	4 (566/43)	12 (575/42)	10 (590/46)
Total Dead PND 5–21	0 (348/48)	1 (344/43)	3 (336/42)	1 (368/46)
Dead/Litter PND 1–4	0.388 ± 0.193 (49)	0.093 ± 0.045 (43)	0.286 ± 0.104 (42)	0.217 ± 0.076 (46)
Dead/Litter PND 4–21	0.000 ± 0.000 (48)	0.023 ± 0.023 (43)	0.071 ± 0.040 (42)	0.022 ± 0.022 (46)
Survival Ratio PND 1–4[d]	0.986 ± 0.005 (48)	0.994 ± 0.003 (43)	0.981 ± 0.007 (42)	0.985 ± 0.005 (46)
Survival Ratio PND 4–21[e]	1.000 ± 0.000 (48)	0.997 ± 0.003 (43)	0.991 ± 0.005 (42)	0.997 ± 0.003 (46)

[a]All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.
[b]Includes dead on PND 1. Survival information on PND 4 was not available for some non-acceptable litters, so these were excluded from the analysis.
[c]Number of pups/number of dams.
[d]Number of pups preculling on PND 4/total number of viable pups on PND 1 (does not include pups dead on PND 1).
[e]Number of pups alive on PND 21/number of pups at postculling on PND 4.

During the lactation period, maternal body weights of the 3 and 6 W/kg groups were significantly decreased (up to 5% and 9%, respectively) compared to those of sham controls from PND 4 through 21 (Table 14). At PND 1, male and female pup weights in the 6 W/kg groups were 4% to 5% less than those of the sham controls (Table 15). Male and female pup weights were also significantly decreased compared to the sham controls with a 4% to 8% decrease across most time points in the 3 W/kg groups and a 6% to 8% decrease across all time points in the 6 W/kg groups.

Table 14. Mean Body Weights and Mean Body Weight Gains of F_0 Female Rats during Lactation in the Two-year Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Postnatal Day				
1	280.9 ± 2.0 (50)▲▲[b]	280.6 ± 2.0 (47)	277.0 ± 1.7 (47)	275.9 ± 1.7 (48)
4	289.7 ± 2.1 (48)▲▲	288.7 ± 2.0 (43)	284.3 ± 1.9 (42)	280.6 ± 1.8 (46)**
7	297.1 ± 2.2 (48)▲▲	293.7 ± 2.1 (43)	290.4 ± 1.9 (42)*	286.4 ± 1.9 (46)**
14	314.4 ± 2.0 (48)▲▲	310.1 ± 2.3 (43)	302.3 ± 1.8 (42)**	290.7 ± 2.2 (46)**
17	313.9 ± 2.2 (48)▲▲	309.2 ± 2.4 (43)	299.2 ± 1.8 (42)**	285.3 ± 2.5 (46)**
21	299.7 ± 2.2 (48)▲▲	295.9 ± 2.4 (43)	287.6 ± 1.8 (42)**	278.1 ± 2.4 (45)**
Postnatal Day Interval				
1–4	9.2 ± 0.9 (48)▲▲	8.1 ± 1.0 (43)	7.3 ± 1.1 (42)	4.8 ± 1.1 (46)**
4–7	7.4 ± 1.5 (48)	5.0 ± 1.2 (43)	6.1 ± 1.3 (42)	5.8 ± 0.9 (46)
7–14	17.4 ± 1.4 (48)▲▲	16.4 ± 1.4 (43)	11.9 ± 1.1 (42)**	4.3 ± 1.2 (46)**
14–17	–0.6 ± 1.0 (48)▲▲	–0.9 ± 1.4 (43)	–3.1 ± 1.0 (42)	–5.5 ± 1.1 (46)**
17–21	–14.1 ± 1.3 (48)▲▲	–13.3 ± 1.7 (43)	–11.6 ± 1.2 (42)	–7.1 ± 1.8 (45)**
1–21	19.3 ± 1.5 (48)▲▲	15.3 ± 1.3 (43)	10.6 ± 1.8 (42)**	2.4 ± 2.0 (45)**

▲▲Significant trend ($P \leq 0.01$).
*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.
[b]Number of dams.

Table 15. Adjusted Mean Body Weights of F_1 Male and Female Rats during Lactation in the Two-year Perinatal and Postnatal Study of GSM-modulated Cell Phone RFR[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adjusted Male Live Pup Weight				
PND 1[b]	7.22 ± 0.07 (49)▲▲[c]	7.18 ± 0.06 (46)	7.06 ± 0.07 (46)	6.84 ± 0.08 (48)▲▲
PND 4	10.90 ± 0.12 (190/48)**[d]	10.70 ± 0.12 (171/43)	10.24 ± 0.14 (168/42)**	9.90 ± 0.12 (185/46)**
PND 7	17.22 ± 0.19 (189/48)**	17.21 ± 0.19 (170/43)	15.97 ± 0.26 (166/42)**	15.64 ± 0.21 (185/46)**
PND 14	35.22 ± 0.43 (181/46)**	34.56 ± 0.35 (166/42)	33.04 ± 0.53 (162/41)**	32.37 ± 0.37 (185/46)**

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
PND 17	42.46 ± 0.48 (190/48)**	41.97 ± 0.43 (165/42)	40.75 ± 0.59 (162/41)*	39.79 ± 0.45 (185/46)**
PND 21	58.50 ± 0.62 (190/48)**	58.40 ± 0.61 (170/43)	56.36 ± 0.78 (166/42)	54.99 ± 0.67 (185/46)**
Adjusted Female Live Pup Weight				
PND 1[b]	6.83 ± 0.06 (49)▲▲	6.84 ± 0.08 (47)	6.68 ± 0.09 (46)	6.54 ± 0.07 (48)▲▲
PND 4	10.46 ± 0.11 (194/48)**	10.23 ± 0.12 (172/43)	9.78 ± 0.14 (168/42)**	9.57 ± 0.15 (183/46)**
PND 7	16.49 ± 0.18 (194/48)**	16.53 ± 0.18 (172/43)	15.35 ± 0.22 (168/42)**	15.05 ± 0.23 (182/46)**
PND 14	33.89 ± 0.38 (192/48)**	33.47 ± 0.31 (165/41)	32.42 ± 0.37 (164/41)*	31.34 ± 0.38 (181/46)**
PND 17	40.82 ± 0.44 (194/48)**	40.42 ± 0.36 (168/42)	39.65 ± 0.42 (167/42)	38.30 ± 0.45 (182/46)**
PND 21	55.42 ± 0.53 (194/48)**	55.28 ± 0.45 (169/42)	54.17 ± 0.53 (167/42)	52.24 ± 0.64 (182/46)**
Adjusted Combined Live Pup Weight				
PND 1[b]	7.03 ± 0.06 (49)▲▲	7.00 ± 0.08 (47)	6.92 ± 0.08 (47)	6.71 ± 0.07 (48)▲▲
PND 4	10.68 ± 0.11 (384/48)**	10.47 ± 0.11 (343/43)	10.01 ± 0.13 (336/42)**	9.74 ± 0.13 (368/46)**
PND 7	16.84 ± 0.18 (383/48)**	16.87 ± 0.18 (342/43)	15.66 ± 0.22 (334/42)**	15.35 ± 0.21 (367/46)**
PND 14	34.48 ± 0.40 (373/48)**	34.01 ± 0.31 (331/42)	32.70 ± 0.41 (326/42)**	31.86 ± 0.36 (366/46)**
PND 17	41.62 ± 0.46 (384/48)**	41.19 ± 0.38 (333/42)	40.20 ± 0.46 (329/42)	39.06 ± 0.43 (367/46)**
PND 21	56.93 ± 0.56 (384/48)**	56.88 ± 0.50 (339/43)	55.23 ± 0.60 (333/42)	53.65 ± 0.63 (367/46)**

▲▲Significantly different ($P \leq 0.01$) for PND 1 endpoint (statistical significance in the sham control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant pairwise comparison against the sham control group).
*Significantly different ($P \leq 0.05$) for PNDs after PND 1 endpoints (statistical significance in the sham control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant pairwise comparison against the sham control group).
**$P \leq 0.01$
[a]Body weights in grams. Data are displayed as mean ± standard error. PND = postnatal day. Values listed as PND 1 refer to the total pup weight divided by the number of pups in litter at PND 1, and the statistical analysis performed is by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests. Values listed for all other days refer to individual pups, and the statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. Individual pup body weights first adjusted for live PND 1 litter size via the analysis of covariance.
[b]Values listed as PND 1 refer to the total pup weight divided by the number of pups in litter at PND 1.
[c]Number of dams.
[d]Number of pups/number of dams.

Postnatal Exposure

Survival

Estimates of 2-year survival probabilities for male and female rats are shown in Table 16 and in the Kaplan-Meier survival curves (Figure 7). Survival of all male exposed groups was significantly greater than that of the sham controls. Decreased survival in the sham control group was largely attributed to the higher severity of chronic progressive nephropathy in the kidney. Survival of exposed females was similar to that of the sham controls.

Table 16. Survival of Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Animals initially in study	105	105	105	105
Fourteen-week interim evaluation[a]	15	15	15	15
Accidental deathsb	1	0	0	1
Moribund	44	24	19	13
Natural deaths	20	21	21	16
Animals surviving to study termination	25	45	50	60
Percent probability of survival at end of study[c]	28	50	56	68
Mean survival (days)[d]	642	675	690	684
Survival analysis[e]	P < 0.001N	P = 0.002N	P ≤ 0.001N	P ≤ 0.001N
Female				
Animals initially in study	105	105	105	105
Fourteen-week interim evaluation	15	15	15	15
Accidental death	1	0	0	0
Moribund	30	26	31	22
Natural deaths	11	11	11	11
Animals surviving to study termination	48[f]	53[f]	48[f]	57
Percent probability of survival at end of study	54	59	53	63
Mean survival (days)	659	682	662	676
Survival analysis	P = 0.300N	P = 0.412N	P = 1.000	P = 0.226N

[a]Excluded from survival analysis.
[b]Censored in the survival analysis.
[c]Kaplan-Meier determinations.
[d]Mean of all deaths (uncensored, censored, and terminal euthanasia).
[e]The result of the life table trend test[161] is in the sham control column, and the results of the life table pairwise comparisons[160] with the sham controls are in the exposed group columns. A negative trend or lower mortality in an exposure group is indicated by N.
[f]Includes one animal that died during the last week of the study.

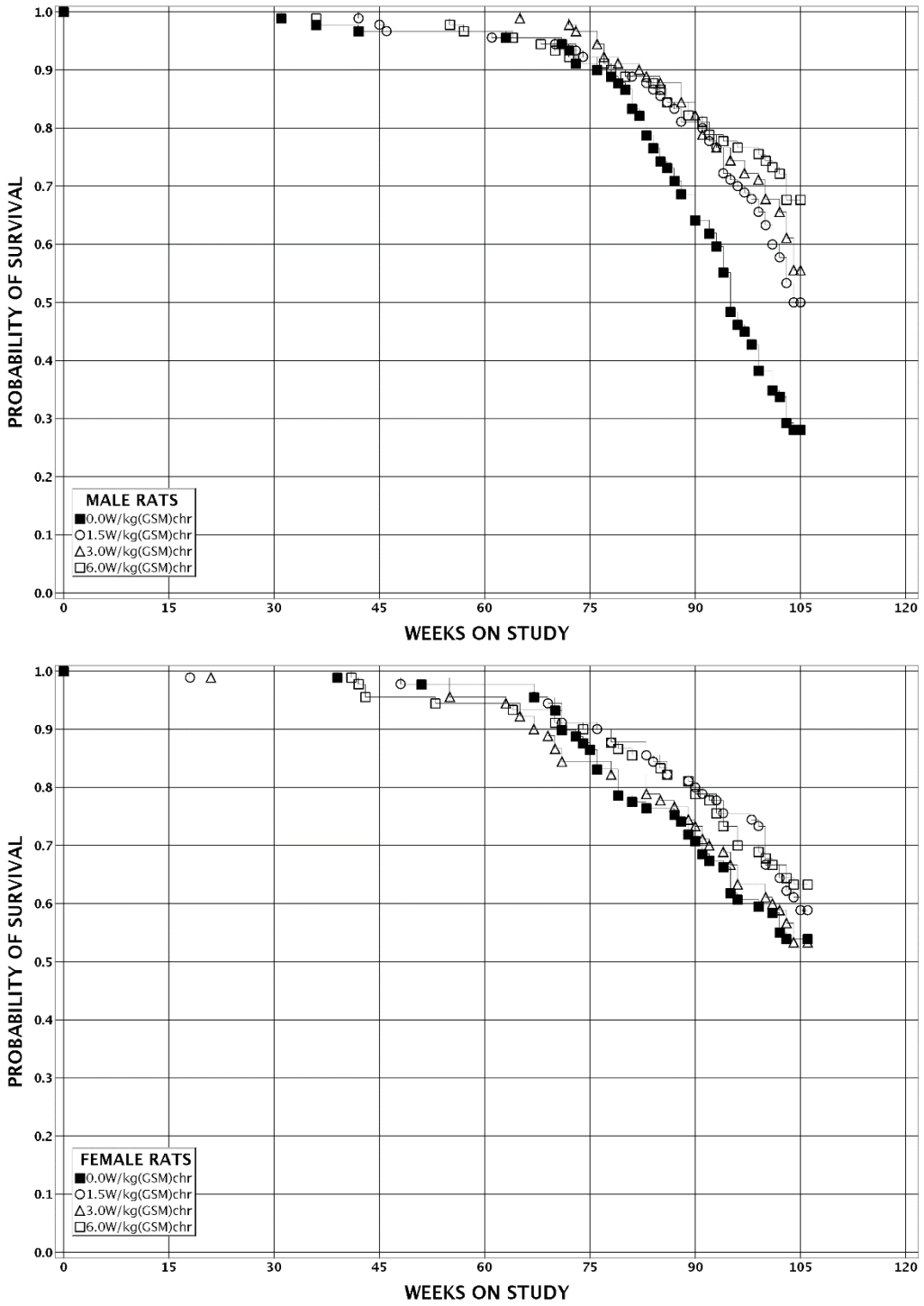

Figure 7. Kaplan-Meier Survival Curves for Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

Body Weights and Clinical Observations

In 6 W/kg males, mean body weights were lower (3% to 6%) at all time points through day 401 (Table 17 and Figure 8); however, from day 541 to terminal sacrifice the mean body weights were greater than those of the sham controls (up to 7.2% greater on day 681). In the 1.5 and 3 W/kg male groups, mean body weights were 5% to 7% greater than the sham controls at some time points, but the increases were sporadic. However, at the end of the study, the mean body weights of these male groups were similar to those of the sham controls. In exposed female groups, the mean body weights and mean body weight gains were similar to those of the sham controls throughout the study (Table 18 and Figure 8). There were no exposure-related clinical observations in males or females.

Table 17. Mean Body Weights and Survival of Male Rat Litters Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
Day	Av. Wt. (g)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters
1	64.3	35	63.9	99.4	35	62.0	96.5	35	60.9	94.8	35
5	78.7	35	77.6	98.7	35	75.1	95.4	35	74.3	94.4	35
9	99.0	35	98.1	99.1	35	94.9	95.8	35	93.9	94.9	35
12	117.6	35	115.7	98.4	35	112.9	96.0	35	111.6	94.9	35
16	145.5	35	143.1	98.4	35	139.5	95.9	35	137.9	94.8	35
19	167.0	35	165.4	99.1	35	161.1	96.5	35	159.2	95.3	35
23	197.8	35	194.9	98.5	35	189.9	96.0	35	187.1	94.6	35
26	217.1	35	213.7	98.4	35	209.6	96.5	35	205.0	94.4	35
30	246.5	35	241.3	97.9	35	236.6	96.0	35	231.7	94.0	35
33	264.3	35	260.4	98.5	35	254.7	96.4	35	249.2	94.3	35
37	290.4	35	285.9	98.4	35	280.2	96.5	35	274.1	94.4	35
40	304.5	35	298.7	98.1	35	294.9	96.8	35	287.3	94.4	35
44	322.9	35	318.0	98.5	35	313.7	97.1	35	306.4	94.9	35
47	333.8	35	329.8	98.8	35	324.3	97.1	35	313.9	94.0	35
51	349.0	35	343.7	98.5	35	338.9	97.1	35	331.4	95.0	35
54	357.6	35	353.5	98.8	35	348.5	97.5	35	341.0	95.3	35
58	370.2	35	366.4	99.0	35	361.3	97.6	35	350.7	94.7	35
61	379.1	35	370.9	97.8	35	370.2	97.6	35	357.2	94.2	35
65	389.6	35	383.0	98.3	35	380.3	97.6	35	370.2	95.0	35
68	395.2	35	389.9	98.6	35	387.0	97.9	35	377.4	95.5	35
72	404.0	35	395.9	98.0	35	395.2	97.8	35	384.1	95.1	35
75	409.0	35	402.5	98.4	35	400.0	97.8	35	389.2	95.2	35
79	417.9	35	408.3	97.7	35	408.0	97.6	35	396.5	94.9	35

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
Day	Av. Wt. (g)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters
82	422.7	35	412.2	97.5	35	413.1	97.7	35	401.4	95.0	35
86	427.7	35	420.4	98.3	35	418.5	97.8	35	409.1	95.7	35
89	432.4	35	422.6	97.7	35	422.7	97.8	35	410.2	94.9	35
93	439.3	35	432.3	98.4	35	429.8	97.8	35	419.3	95.4	35
121[a]	471.3	35	470.7	99.9	35	465.0	98.7	35	458.3	97.3	35
149	501.7	35	496.8	99.0	35	494.4	98.5	35	481.9	96.1	35
177	522.0	35	518.6	99.3	35	514.1	98.5	35	503.8	96.5	35
205	540.4	35	539.0	99.7	35	532.9	98.6	35	523.0	96.8	35
233	560.8	35	558.1	99.5	35	552.1	98.4	35	539.5	96.2	35
261	575.2	35	573.9	99.8	35	568.3	98.8	35	555.4	96.5	35
289	590.2	35	589.6	99.9	35	583.4	98.8	35	570.3	96.6	35
317	608.0	35	606.3	99.7	35	597.4	98.3	35	585.0	96.2	35
345	618.4	35	617.4	99.8	35	608.4	98.4	35	596.3	96.4	35
373	628.4	35	632.3	100.6	35	619.4	98.6	35	606.0	96.4	35
401	636.3	35	641.2	100.8	35	630.1	99.0	35	615.0	96.6	35
429	641.4	35	646.9	100.9	35	637.2	99.4	35	624.5	97.4	35
457	639.9	35	652.0	101.9	35	641.3	100.2	35	630.3	98.5	35
485	645.0	35	659.8	102.3	35	652.3	101.1	35	640.1	99.2	35
513	654.1	35	675.1	103.2	35	667.1	102.0	35	650.2	99.4	35
541	649.5	35	687.0	105.8	35	676.3	104.1	35	657.5	101.2	35
569	651.2	33	686.5	105.4	35	672.1	103.2	35	659.9	101.3	35
597	659.2	32	687.6	104.3	35	679.4	103.1	34	664.7	100.8	35
625	652.8	32	685.7	105.0	35	672.9	103.1	34	667.1	102.2	35
639	645.9	32	682.5	105.7	35	670.2	103.8	33	665.3	103.0	33

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
Day	Av. Wt. (g)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters
653	630.8	31	675.3	107.1	35	663.5	105.2	33	664.8	105.4	33
667	632.9	28	673.2	106.4	34	659.0	104.1	33	668.1	105.6	33
681	621.0	26	657.9	105.9	33	652.9	105.1	33	668.1	107.6	33
695	625.8	21	656.3	104.9	32	638.8	102.1	33	663.3	106.0	32
709	624.0	20	650.5	104.2	30	638.1	102.3	31	650.9	104.3	32
723	624.9	19	637.1	101.9	29	633.1	101.3	28	641.4	102.6	31
Mean for Weeks											
1–14	297.9	–	292.9	98.3	–	289.7	97.2	–	282.6	94.9	–
15–52	554.2	–	552.3	99.7	–	546.2	98.6	–	534.8	96.5	–
53–104	639.0	–	663.9	103.9	–	653.2	102.2	–	649.2	101.6	–

[a]Interim evaluation occurred during week 14.

Table 18. Mean Body Weights and Survival of Female Rat Litters Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
Day	Av. Wt. (g)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters
1	60.4	35	59.9	99.3	35	58.7	97.2	35	58.0	96.1	35
5	72.1	35	71.0	98.5	35	70.4	97.7	35	69.6	96.5	35
9	88.7	35	87.2	98.3	35	85.8	96.7	35	85.0	95.8	35
12	103.3	35	100.7	97.5	35	99.6	96.5	35	98.6	95.5	35
16	123.2	35	120.1	97.5	35	118.8	96.4	35	117.8	95.6	35
19	136.3	35	134.4	98.6	35	133.0	97.6	35	131.2	96.3	35
23	151.5	35	150.4	99.3	35	148.6	98.0	35	147.7	97.5	35
26	158.5	35	160.3	101.1	35	156.78	98.9	35	155.8	98.3	35
30	169.1	35	170.2	100.7	35	166.4	98.4	35	167.2	98.9	35

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
Day	Av. Wt. (g)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters
33	176.4	35	178.3	101.1	35	175.0	99.2	35	174.6	99.0	35
37	188.7	35	190.8	101.1	35	185.1	98.1	35	186.4	98.8	35
40	195.8	35	196.8	100.5	35	193.2	98.7	35	192.3	98.2	35
44	203.7	35	205.4	100.9	35	201.8	99.1	35	202.2	99.3	35
47	209.9	35	210.9	100.5	35	208.5	99.3	35	207.1	98.7	35
51	218.1	35	220.7	101.2	35	214.5	98.3	35	216.4	99.2	35
54	220.1	35	225.2	102.3	35	220.6	100.2	35	220.7	100.3	35
58	228.6	35	230.6	100.9	35	225.8	98.8	35	226.7	99.2	35
61	233.9	35	235.2	100.6	35	230.2	98.4	35	229.9	98.3	35
65	239.0	35	238.7	99.9	35	236.2	98.8	35	235.2	98.4	35
68	241.7	35	242.5	100.3	35	240.5	99.5	35	240.6	99.5	35
72	244.9	35	243.9	99.6	35	242.0	98.8	35	240.7	98.3	35
75	247.6	35	246.6	99.6	35	246.7	99.7	35	246.3	99.5	35
79	251.6	35	251.4	99.9	35	249.7	99.3	35	249.2	99.1	35
82	253.5	35	252.9	99.8	35	252.2	99.5	35	250.9	99.0	35
86	254.4	35	255.8	100.6	35	253.8	99.8	35	253.5	99.6	35
89	256.2	35	255.1	99.5	35	255.5	99.7	35	254.1	99.2	35
93	257.4	35	261.3	101.4	35	257.2	99.8	35	258.8	100.5	35
121[a]	275.6	35	278.4	101.0	35	274.3	99.5	35	274.9	99.7	35
149	284.8	35	287.3	100.9	35	283.0	99.4	35	285.1	100.1	35
177	295.0	35	296.7	100.6	35	294.0	99.7	35	295.7	100.2	35
205	302.8	35	307.5	101.5	35	302.4	99.9	35	304.4	100.5	35
233	311.4	35	315.6	101.4	35	311.3	100.0	35	314.1	100.9	35
261	317.9	35	323.3	101.7	35	317.3	99.8	35	321.8	101.2	35
289	330.8	35	330.0	99.7	35	329.4	99.6	35	325.5	98.4	35

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
Day	**Av. Wt. (g)**	**No. of Litters**	**Av. Wt. (g)**	**Wt. (% of Controls)**	**No. of Litters**	**Av. Wt. (g)**	**Wt. (% of Controls)**	**No. of Litters**	**Av. Wt. (g)**	**Wt. (% of Controls)**	**No. of Litters**
317	340.8	35	340.9	100.0	35	336.3	98.7	35	337.1	98.9	35
345	348.2	35	347.5	99.8	35	345.0	99.1	35	343.7	98.7	35
373	355.2	35	355.1	100.0	35	355.0	99.9	35	348.8	98.2	35
401	366.7	35	366.0	99.8	35	365.3	99.6	34	359.4	98.0	35
429	375.9	35	375.6	99.9	35	376.5	100.2	34	367.4	97.7	35
457	386.6	35	384.0	99.3	35	387.9	100.3	34	375.3	97.1	35
485	400.1	35	395.1	98.7	33	396.1	99.0	33	389.5	97.4	35
513	410.3	35	411.7	100.3	33	403.2	98.3	33	401.8	97.9	35
541	421.4	35	423.8	100.6	33	413.3	98.1	33	411.4	97.6	35
570	426.0	35	432.9	101.6	33	423.3	99.4	33	425.9	100.0	34
598	433.7	35	439.3	101.3	33	433.6	100.0	33	437.4	100.9	34
626	447.7	34	446.6	99.8	33	436.4	97.5	32	451.3	100.8	34
640	454.9	33	453.2	99.6	33	442.2	97.2	32	455.2	100.1	34
654	459.5	33	454.9	99.0	33	445.8	97.0	32	456.3	99.3	34
668	448.5	31	463.2	103.3	33	450.5	100.4	32	458.8	102.3	34
682	450.7	31	471.6	104.6	33	443.1	98.3	31	460.8	102.2	34
696	450.0	31	488.4	108.5	33	449.9	100.0	31	461.5	102.5	34
710	447.9	30	464.7	103.8	30	455.4	101.7	30	471.8	105.3	34
724	448.8	30	464.8	103.6	29	463.7	103.3	30	468.1	104.3	34
Mean for Weeks											
1–14	192.0	–	192.5	100.3	–	189.9	98.9	–	189.5	98.7	–
15–52	311.9	–	314.1	100.7	–	310.3	99.5	–	311.4	99.8	–
53–104	422.6	–	428.9	101.5	–	420.1	99.4	–	423.6	100.2	–

[a]Interim evaluation occurred during week 14.

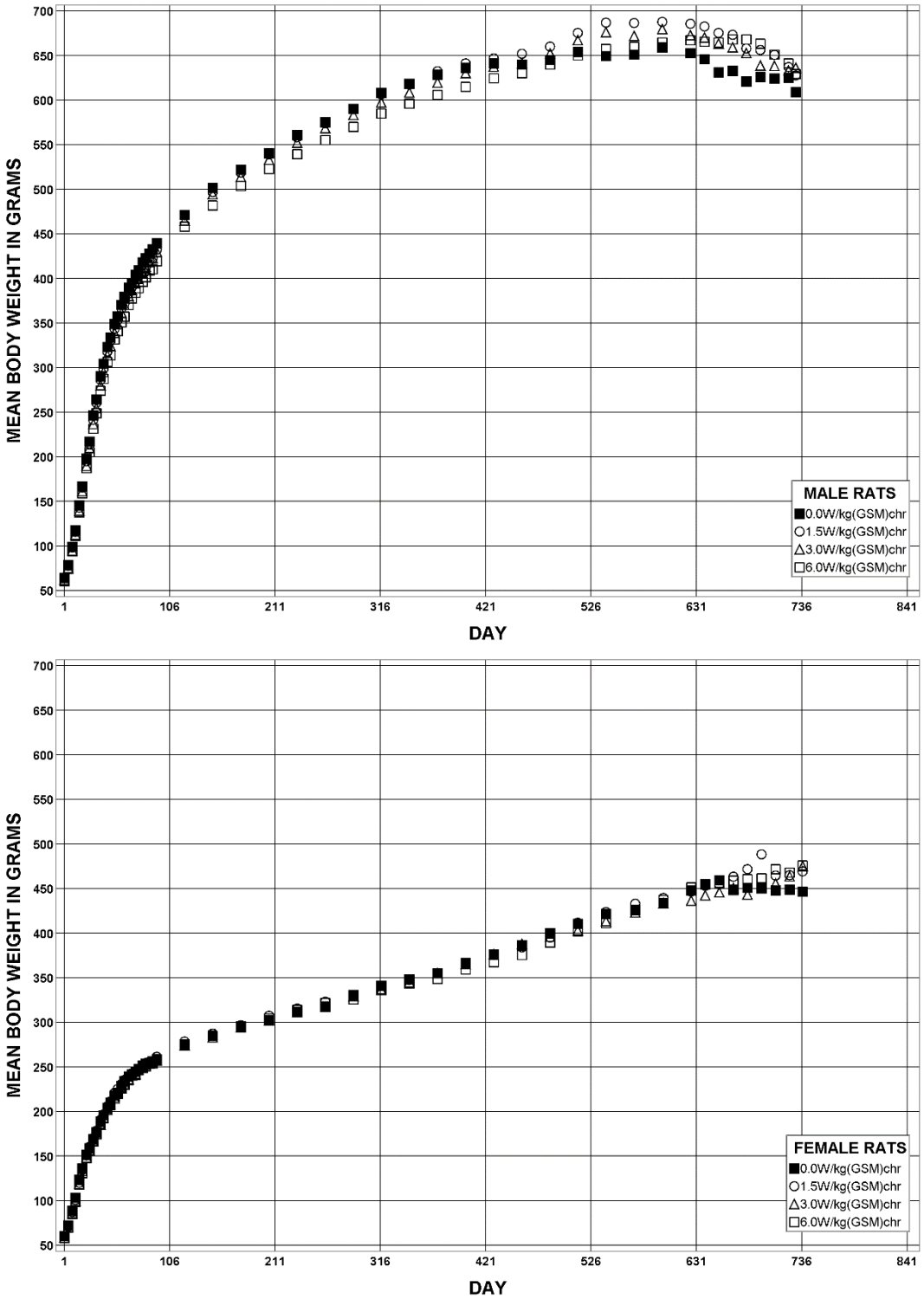

Figure 8. Growth Curves for Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

Fourteen-week Interim Evaluation

There were significantly decreased leukocyte and lymphocyte counts (22%) in 3 W/kg females (Table F-1). While these decreases may have been stress related, they only occurred in the

3 W/kg group and only in females, thus the biological relevance is uncertain. In 6 W/kg females, there were significant decreases in cholesterol (19%) and triglyceride (38%) concentrations. These decreases may have been due to changes in lipid metabolism.

In males, the absolute left and right kidney weights were significantly decreased in the 1.5 (9% and 10%, respectively) and 6 W/kg (15% and 11%, respectively) groups (Table G-4). The absolute liver weight of 6 W/kg males was also significantly decreased (10%). The relative weights of these organs were not significantly decreased, which was likely due to the lower terminal mean body weights of the 1.5 (4%) and 6 W/kg (8%) groups. In females, the relative brain weights of the 1.5 and 6 W/kg groups were significantly increased, which was likely due to lower mean body weights in these two groups (Table G-4). The absolute left kidney weights were significantly decreased in all exposed female groups (9% to 14%). Similarly, the absolute right kidney weights were significantly decreased in the 3 and 6 W/kg females (8% and 12%, respectively). There were also significant decreases in the absolute lung weight in 6 W/kg females (16%) and the absolute thymus weights in 1.5 (18%) and 6 W/kg (22%) females. The absolute liver weights were significantly decreased in 1.5 and 6 W/kg females (17% and 20%, respectively), with corresponding decreased relative weights. None of these changes were associated with histopathologic findings.

There were no GSM exposure-related effects on reproductive organ weights, testis spermatid concentrations, caudal epididymal sperm concentrations, or sperm motility in males (Table H-1). Due to the poor diagnostic quality of the cytology slides, the estrous cycle in females was not evaluated.

In the heart, the incidences of cardiomyopathy in the right ventricle only in males was greater in the 3 and 6 W/kg groups compared to the sham controls, although the increases were not statistically significant (Table 19). In the entire heart (i.e., all sites combined) in males, the incidences of cardiomyopathy were increased in the 3 and 6 W/kg groups compared to the sham controls, but only the increase in the 3 W/kg group was statistically significant. The average severity was comparable to that of the sham control group. Cardiomyopathy was initially diagnosed separately in the right ventricle, but was also seen in the left ventricular free wall and interventricular septum. The incidences of cardiomyopathy in the left ventricle and interventricular septum (i.e., excluding the right ventricle) were similar in all exposed groups compared to the sham controls. In females, the incidence of cardiomyopathy in the exposed groups was not considered to be related to exposure to GSM-modulated cell phone RFR.

Cardiomyopathy was characterized by degeneration and necrosis of myofibers with a mild inflammatory response of macrophages and lymphocytes with occasional neutrophils. In the right ventricle, the cardiomyopathy was most prominent in the subepicardial region in the lower half (toward the apex) of the heart. In more severe cases, the lesions in the right ventricle extended deeper into the myocardium. In some areas, there appeared to be areas of cardiomyocyte loss, characterized by linear, clear areas containing a few scattered cell nuclei and variable amounts of linear, eosinophilic material (likely collagen bundles).

In the mandibular lymph node of males, there was an increased incidence of lymphocyte hyperplasia in the 6 W/kg group compared to the sham control group (Table 19). In all cases, the severity of the lesions was minimal. There were no similar findings in other lymph nodes or in females in either modulation.

Table 19. Incidences of Selected Non-neoplastic Lesions at the 14-week Interim Evaluation in Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 w/kg
Male				
Heart[a]	10	10	10	10
Cardiomyopathy (Excluding Right Ventricle)[b]	2 (1.5)[c]	4 (1.0)	3 (1.0)	4 (1.0)
Ventricle Right, Cardiomyopathy	1 (1.0)	1 (2.0)	5 (1.2)	5 (1.0)
Cardiomyopathy, All Sites	3 (1.3)	5 (1.2)	8* (1.1)	7 (1.0)
Lymph Node, Mandibular	10	10	10	10
Hyperplasia, Lymphocyte	0	0	0	4* (1.0)
Proliferation, Plasma Cell	0	0	0	2 (1.0)
Female				
Heart	10	10	10	10
Cardiomyopathy (Excluding Right Ventricle)	0	2 (1.0)	1 (1.0)	2 (1.0)
Ventricle Right, Cardiomyopathy	0	0	1 (1.0)	0
Cardiomyopathy, All Sites	0	2 (1.0)	2 (1.0)	2 (1.0)
Lymph Node, Mandibular	10	10	10	10
Hyperplasia, Lymphocyte	0	0	0	2 (1.0)
Proliferation, Plasma Cell	0	0	0	3 (1.0)

*Significantly different ($P \leq 0.05$) from the sham control group by the Fisher exact test.
[a]Number of animals with tissue examined microscopically.
[b]Number of animals with lesion.
[c]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.

Pathology and Statistical Analyses

This section describes the statistically significant or biologically noteworthy changes in the incidences of neoplasms and non-neoplastic lesions of the heart, brain, adrenal medulla, prostate gland, pituitary gland (pars distalis), pancreatic islets, thyroid gland, adrenal cortex, kidney and other organs, mammary gland, pancreas, and seminal vesicle. Summaries of the incidences of neoplasms and non-neoplastic lesions, statistical analyses of primary neoplasms that occurred with an incidence of at least 5% in at least one animal group, and historical incidences for the neoplasms mentioned in this section are presented in Appendix A for male rats and Appendix B for female rats.

Heart: Malignant schwannomas were observed in all exposed male groups. No schwannomas were observed in the sham controls. The incidences in the exposed groups occurred with a significant positive trend (Table 20, Table A-1, and Table A-2). In females, schwannoma occurred in two 3 W/kg animals (Table 20 and Table B-1). Endocardial Schwann cell hyperplasia, a putative preneoplastic Schwann cell lesion, was diagnosed in one 1.5 W/kg male and two 6 W/kg males (Table 20 and Table A-9); there were none in females. Schwannomas were seen in other organs, including the pituitary gland, tri-geminal nerve, salivary glands, Harderian gland, eye, thymus gland, uterus, ovary, and vagina. When the incidences of

schwannoma in all organs (including the heart) were combined, they were generally higher in exposed males, but not significantly different from the sham controls.

In the heart, some schwannomas were endocardial (Plates 1, 2, and 3) and others were myocardial (Plates 4 and 5). They were not recorded separately in the final data because their biological behavior and morphology are similar. The endocardial schwannomas typically arose in the subendocardial region of the left ventricle, though some were found in the right ventricle. They were composed of two morphologically distinct cell populations, both of which have indistinct cell boundaries. One population, immediately adjacent to the endocardium, was elongated and had ovoid nuclei, prominent nucleoli, and scant, pale cytoplasm. The other population, which was far more abundant, was more spindloid, with fusiform, hyperchromatic nuclei oriented in parallel.

The schwannomas were invasive, dissecting between cariomyocytes. In a few larger neoplasms, the nuclei of the spindle-shaped cells lined up adjacent to each other (early palisading of nuclei) or exhibited a wavy pattern. The myocardial schwannomas had a similar appearance, but they were less cellular than endocardial schwannomas. They were composed of spindle-shaped cells in a loose arrangement. Endocardial Schwann cell hyperplasia (Plate 6) was similar in appearance to endocardial schwannomas, but was less extensive and did not invade into the adjacent myocardium.

Cardiomyopathy of the right ventricular free wall was seen in all male and female groups (including sham controls) (Table 20, Table A-9, and Table B-7). In males and females, the incidences were higher in all exposed groups compared to sham controls; the increases were statistically significant in the 3 and 6 W/kg groups. There was also a slight elevation in the severity in 3 and 6 W/kg males, but there was no such elevation in females. Cardiomyopathy was diagnosed separately in the right ventricle because the study pathologist observed an unusual predilection for, and a slightly different morphology in the right ventricle as compared to the left ventricular free wall and the interventricular septum. A higher incidence was found in the right ventricle in the 6 W/kg groups compared to the sham controls. Cardiomyopathy is a very common spontaneous disease in rats and was also seen in other sites in the heart (most notably the left ventricular free wall, but also the interventricular septum). In the current study, it was more prominent in the apex and lower half (toward the apex) of the heart. When cardiomyopathy was evaluated in the entire heart (including the right ventricle), there was no significant difference in the incidences or average severities in exposed male groups when compared to sham controls. In females, there were significantly decreased incidences of cardiomyopathy of the whole heart in the 1.5 and 6 W/kg groups. Cardiomyopathy (Plate 7) was characterized by degeneration and necrosis of myofibers with a mild inflammatory response of macrophages and lymphocytes with occasional neutrophils. In later stages of the disease, fibrosis may be prominent. In the right ventricle, the cardiomyopathy was most prominent in the subepicardial region in the lower half (toward the apex) of the heart. In more severe cases, the lesions extended deeper into the right ventricular wall. The lesions were similar to those described above. In some areas, there appeared to be areas of cardiomyocyte loss, characterized by linear, clear areas containing a few scattered cell nuclei and variable amounts of linear, eosinophilic material (likely collagen bundles). Fibrosis was sometimes fairly prominent. The effect of GSM-modulated cell phone RFR on the incidence of cardiomyopathy appears to be specific to the right ventricular free wall.

Table 20. Incidences of Malignant Schwannoma and Neoplasms and Non-neoplastic Lesions of the Heart in Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Heart[a]	90	90	90	90
Cardiomyopathy[b]	79 (1.9)[c]	82 (1.8)	78 (2.1)	79 (1.6)
Ventricle Right, Cardiomyopathy	54 (1.1)	62 (1.5)	72* (1.9)	74** (1.8)
Endocardium, Hyperplasia, Schwann Cell	0	1 (1.0)	0	2 (2.0)
Endocardium, Schwannoma Malignant	0	1	1	2
Myocardium, Schwannoma Malignant	0	1	0	3
Malignant Schwannoma[d]				
Overall rate[e]	0/90 (0%)	2/90 (2%)	1/90 (1%)	5/90 (6%)
Litters rate[f]	0/35 (0%)	2/35 (6%)	1/35 (3%)	5/35 (14%)
Adjusted rate[g]	0.0%	2.7%	1.3%	6.4%
Terminal rate[h]	0/25 (0%)	2/45 (4%)	1/50 (2%)	3/60 (5%)
First incidence (days)	–[j]	730 (T)	730 (T)	582
Rao-Scott adjusted Poly-3 test[i]	P = 0.041	P = 0.297	P = 0.540	P = 0.080
All Organs: Malignant Schwannoma[k]				
Overall rate	3/90 (3%)	3/90 (3%)	5/90 (6%)	7/90 (8%)
Litters rate	3/35 (9%)	3/35 (9%)	5/35 (14%)	7/35 (20%)
Adjusted rate	4.5%	4.0%	6.4%	8.9%
Terminal rate	1/25 (4%)	2/45 (4%)	3/50 (6%)	4/60 (7%)
First incidence (days)	555	720	661	582
Rao-Scott adjusted Poly-3 test	P = 0.133	P = 0.577N	P = 0.435	P = 0.238
Female				
Heart	90	90	90	90
Cardiomyopathy	40 (1.1)	30* (1.2)	39 (1.1)	27* (1.1)
Ventricle Right, Cardiomyopathy	4 (1.0)	9 (1.1)	14* (1.1)	15* (1.2)
Endocardium, Schwannoma Malignant	0	0	1	0
Myocardium, Schwannoma Malignant	0	0	1	0
Malignant Schwannoma[l]				
Overall rate	0/90 (0%)	0/90 (0%)	2/90 (2%)	0/90 (0%)
Litters rate	0/35 (0%)	0/35 (0%)	2/35 (6%)	0/35 (0%)
Adjusted rate	0.0%	0.0%	2.8%	0.0%
Terminal rate	0/48 (0%)	0/53 (0%)	1/48 (2%)	0/57 (0%)
First incidence (days)	–	–	578	–
Rao-Scott adjusted Poly-3 test	P = 0.640	–[m]	P = 0.365	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
All Organs: Malignant Schwannoma[n]				
Overall rate	4/90 (4%)	1/90 (1%)	5/90 (6%)	2/90 (2%)
Litters rate	3/35 (9%)	1/35 (3%)	5/35 (14%)	2/35 (6%)
Adjusted rate	5.7%	1.3%	7.0%	2.7%
Terminal rate	2/48 (4%)	0/53 (0%)	2/48 (4%)	1/57 (2%)
First incidence (days)	489	480	578	622
Rao-Scott adjusted Poly-3 test	P = 0.428N	P = 0.212N	P = 0.519	P = 0.354N

*Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted Poly-3 test.
**$P \leq 0.01$.
(T) = terminal euthanasia.
[a]Number of animals with tissue examined microscopically.
[b]Number of animals with lesion.
[c]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.
[d]Historical control incidence for other 2-year studies (all routes): 1/50, 1/50, 0/50.
[e]Number of animals with neoplasm per number of animals necropsied.
[f]Number of litters with animals with neoplasm per number of litters necropsied.
[g]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[h]Observed incidence at terminal euthanasia.
[i]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
[j]Not applicable; no neoplasms in animal group.
[k]Historical control incidence for other 2-year studies (all routes): 1/50, 2/50, 0/50.
[l]Historical control incidence for other 2-year studies (all routes): 0/49, 0/50, 0/50.
[m]Value of statistic cannot be computed.
[n]Historical control incidence for other 2-year studies (all routes): 0/50, 2/50, 2/50.

Brain: In males, malignant glioma and glial cell hyperplasia occurred in all exposed groups (Table 21, Table A-1, and Table A-9) and neither lesion occurred in the sham control group; however, the incidences were not significant compared to those in the sham controls. In females, malignant glioma occurred in one 6 W/kg animal and glial cell hyperplasia occurred one 3 W/kg animal (Table 21, Table B-1, and Table B-7).

The malignant gliomas (Plates 8, 9, and 10) were generally large neoplasms with indistinct borders. The cells were typically densely packed and were clustered around blood vessels (perivascular cuffing) or aggregated around neurons (satellitosis). The cells invaded the meninges in some cases. The cells had small to moderate amounts of eosinophilic cytoplasm and indistinct cell margins. They had small, round to elongated, hyperchromatic nuclei. A few mitotic figures were present. Glial cell hyperplasia (Plate 11) had a similar appearance, but was smaller with less densely packed cells (all cells were separated by neuropil). Satellitosis and perivascular cuffing were minimal and there was no meningeal invasion and no mitotic figures. In the glial cell hyperplasia, there were no reactive, degenerative, or necrotic elements within the associated parenchyma or other evidence of damage (e.g., hemorrhage, edema). The hyperplastic cells were sometimes hypertrophied, but there were no gitter cells or gemistocytes.

In males, there were three benign granular cell tumors of the meninges in each exposed group and one in the sham control group (Table 21 and Table A-1). There was also a single malignant granular cell tumor in the 3 W/kg group. Granular cell hyperplasia, which is thought to be on a

continuum with benign and malignant granular cell tumors, occurred in one sham control male and one 3 W/kg male (Table 21 and Table A-9). In females, the incidences of granular cell tumors were 1%, 1%, 2%, 0% in the sham control, 1.5, 3, and 6 W/kg groups, respectively (Table 21 and Table B-1). There was also a single malignant granular cell tumor in a 6 W/kg female. Granular cell hyperplasia occurred in one female each from the sham control and 3 W/kg groups.

Granular cell tumors were observed in the meninges or choroid plexus and were composed of sheets of large, densely packed polygonal cells. The cells had abundant, eosinophilic, granular cytoplasm with indistinct cell borders and small, uniform, round to oval nuclei. A few smaller cells with dark basophilic nuclei and sparser, less granular cytoplasm were also present. No mitotic figures were observed. Benign granular cell tumors were discrete, non-invasive masses that caused variable compression of the adjacent brain parenchyma. Granular cell hyperplasias were similar in appearance, but were smaller, non-invasive, and non-compressive. Malignant granular cell tumors were invasive, extending into the underlying neuropil, and had some nuclear pleomorphism.

Table 21. Incidences of Neoplasms and Non-neoplastic Lesions of the Brain in Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	90	90	90	90
Glial Cell, Hyperplasia[a]	0	2 (2.0)[b]	3 (3.0)	1 (4.0)
Meninges, Hyperplasia, Granular Cell	1 (1.0)	0	1 (1.0)	0
Glioma Malignant[c]				
Overall rate[d]	0/90 (0%)	3/90 (3%)	3/90 (3%)	2/90 (2%)
Litters rate[e]	0/35 (0%)	3/35 (9%)	3/35 (9%)	2/35 (6%)
Adjusted rate[f]	0.0%	4.0%	3.8%	2.6%
Terminal rate[g]	0/25 (0%)	2/45 (4%)	1/50 (2%)	1/60 (2%)
First incidence (days)	–[i]	715	649	716
Rao-Scott adjusted Poly-3 test[h]	P = 0.382	P = 0.155	P = 0.166	P = 0.277
Meninges, Granular Cell Tumor Benign[j]				
Overall rate	1/90 (1%)	3/90 (3%)	3/90 (3%)	3/90 (3%)
Litters rate	1/35 (3%)	3/35 (9%)	3/35 (9%)	3/35 (9%)
Adjusted rate	1.5%	4.0%	3.9%	3.9%
Terminal rate	1/25 (4%)	1/45 (2%)	3/50 (6%)	3/60 (5%)
First incidence (days)	730 (T)	655	730 (T)	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.350	P = 0.328	P = 0.342	P = 0.341
Meninges, Granular Cell Tumor Malignant[k]	0	0	1	0
Meninges, Granular Cell Tumor Benign or Malignant[j]				
Overall rate	1/90 (1%)	3/90 (3%)	4/90 (4%)	3/90 (3%)
Litters rate	1/35 (3%)	3/35 (9%)	4/35 (11%)	3/35 (9%)
Adjusted rate	1.5%	4.0%	5.1%	3.9%
Terminal rate	1/25 (4%)	1/45 (2%)	3/50 (6%)	3/60 (5%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
First incidence (days)	730 (T)	655	699	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.343	P = 0.327	P = 0.220	P = 0.340
Female				
Number Examined Microscopically	90	90	90	90
Glial Cell, Hyperplasia	0	0	1 (4.0)	0
Meninges, Hyperplasia, Granular Cell	1 (3.0)	0	1 (3.0)	0
Glioma Malignant[l]				
Overall rate	0/90 (0%)	0/90 (0%)	0/90 (0%)	1/90 (1%)
Litters rate	0/35 (0%)	0/35 (0%)	0/35 (0%)	1/35 (3%)
Adjusted rate	0.0%	0.0%	0.0%	1.3%
Terminal rate	0/48 (0%)	0/53 (0%)	0/48 (0%)	0/57 (0%)
First incidence (days)	–	–	–	669
Rao-Scott adjusted Poly-3 test	–[m]	–	–	–
Meninges, Granular Cell Tumor Benign[n]				
Overall rate	1/90 (1%)	1/90 (1%)	2/90 (2%)	0/90 (0%)
Litters rate	1/35 (3%)	1/35 (3%)	2/35 (6%)	0/35 (0%)
Adjusted rate	1.4%	1.3%	2.8%	0.0%
Terminal rate	1/48 (2%)	1/53 (2%)	1/48 (2%)	0/57 (0%)
First incidence (days)	730 (T)	737 (T)	669	–
Rao-Scott adjusted Poly-3 test	P = 0.375N	P = 0.722N	P = 0.503	P = 0.489N
Meninges, Granular Cell Tumor Malignant[k]	0	0	0	1
Meninges, Granular Cell Tumor Benign or Malignant[n]				
Overall rate	1/90 (1%)	1/90 (1%)	2/90 (2%)	1/90 (1%)
Litters rate	1/35 (3%)	1/35 (3%)	2/35 (6%)	1/35 (3%)
Adjusted rate	1.4%	1.3%	2.8%	1.3%
Terminal rate	1/48 (2%)	1/53 (2%)	1/48 (2%)	1/57 (2%)
First incidence (days)	737 (T)	737 (T)	669	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.594	P = 0.712N	P = 0.485	P = 0.713N

(T) = terminal euthanasia.
[a]Number of animals with lesion.
[b]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.
[c]Historical control incidence for other 2-year studies (all routes): 0/50, 2/50.
[d]Number of animals with neoplasm per number of animals with brain examined microscopically.
[e]Number of litters with animals with neoplasm per number of litters with brain examined microscopically.
[f]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[g]Observed incidence at terminal euthanasia.
[h]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
[i]Not applicable; no neoplasms in animal group.
[j]Historical control incidence for other 2-year studies (all routes): 2/50, 0/50.
[k]Historical control incidence for other 2-year studies (all routes): 0/50, 0/50.
[l]Historical control incidence for other 2-year studies (all routes): 0/50, 1/50.
[m]Value of statistic cannot be computed.
[n]Historical control incidence for other 2-year studies (all routes): 1/50, 0/50.

Adrenal Medulla: In males, there were significantly increased incidences of benign pheochromocytoma and benign, malignant, or complex pheochromocytoma (combined) in the 1.5 and 3 W/kg groups (Table 22, Table A-1, and Table A-2). The incidence of malignant pheochromocytoma was slightly increased in 3 W/kg males, but none were recorded in 6 W/kg males. The upper range of benign pheochromocytoma in the available historical control data for male Hsd:Sprague Dawley® SD® rats is 24% (mean historical incidence is 16%) (Table A-5). There were decreased incidences of hyperplasia in exposed male groups, and the severity was similar to that in the sham controls (Table 22 and Table A-9). In females, there were slightly increased incidences of benign pheochromocytoma in exposed groups, (Table 22 and Table B-1). There was a significant positive trend in the incidences of hyperplasia and the incidence in 6 W/kg females was significant; however, as in males, the average severities in the exposed groups were similar to that in the sham controls (Table 22 and Table B-7). Benign pheochromocytomas were characterized by a well-delineated mass that compressed the adjacent tissue and was composed of cells arranged in large solid clusters or thick trabeculae. Cells exhibited mild to marked alteration in size, shape, and/or staining qualities. Cellular atypia and pleomorphism were common. A diagnosis of malignant pheochromocytoma was made when there was evidence of invasion of the capsule or metastasis.

Table 22. Incidences of Neoplasms and Non-neoplastic Lesions of the Adrenal Medulla in Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	88	90	89	87
Hyperplasia[a]	42 (2.0)[b]	24** (2.1)	26** (1.9)	35 (2.0)
Benign Pheochromocytom[a], Bilateral	1	2	4	0
Benign Pheochromocytom[a], Multiple	1	0	2	0
Benign Pheochromocytoma (includes bilateral and multiple)[c]				
Overall rate[d]	10/88 (11%)	23/90 (26%)	25/89 (28%)	14/87 (16%)
Litters rate[e]	8/35 (23%)	19/35 (54%)	21/35 (60%)	12/35 (34%)
Adjusted rate[f]	15.2%	29.9%	31.7%	18.3%
Terminal rate[g]	3/23 (13%)	13/45 (29%)	14/49 (29%)	11/59 (19%)
First incidence (days)	510	599	526	631
Rao-Scott adjusted Poly-3 test[h]	P = 0.472N	P = 0.030	P = 0.017	P = 0.384
Malignant Pheochromocytoma, Bilateral[i]	0	1	0	0
Malignant Pheochromocytoma (includes bilateral)[i]	1	1	4	0
Complex Pheochromocytoma[j]	1	0	0	0
Benign, Malignant, or Complex Pheochromocytoma[k]				
Overall rate	11/88 (13%)	24/90 (27%)	28/89 (31%)	14/87 (16%)
Litters rate	9/35 (26%)	19/35 (54%)	23/35 (66%)	12/35 (34%)
Adjusted rate	16.7%	31.1%	35.3%	18.3%
Terminal rate	3/23 (13%)	13/45 (29%)	15/49 (31%)	11/59 (19%)
First incidence (days)	510	599	526	631
Rao-Scott adjusted Poly-3 test	P = 0.409N	P = 0.035	P = 0.010	P = 0.472

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Female				
Number Examined Microscopically	86	90	90	86
Hyperplasia	13 (1.5)	19 (1.2)	14 (1.4)	25* (1.8)
Benign Pheochromocytoma, Bilateral	0	0	0	1
Benign Pheochromocytoma (includes bilateral)[l]				
Overall rate	1/86 (1%)	3/90 (3%)	3/90 (3%)	2/86 (2%)
Litters rate	1/35 (3%)	3/35 (9%)	3/35 (9%)	2/35 (6%)
Adjusted rate	1.5%	4.0%	4.2%	2.8%
Terminal rate	1/45 (2%)	3/53 (6%)	3/48 (6%)	2/53 (4%)
First incidence (days)	737 (T)	737 (T)	737 (T)	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.486	P = 0.347	P = 0.319	P = 0.500

*Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted Poly-3 test.
**$P \leq 0.01$.
(T) = terminal euthanasia.
[a]Number of animals with lesion.
[b]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.
[c]Historical control incidence for other 2-year studies (all routes): 9/50, 5/50, 12/50.
[d]Number of animals with neoplasm per number of animals with adrenal medulla examined microscopically.
[e]Number of litters with animals with neoplasm per number of litters with adrenal medulla examined microscopically.
[f]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[g]Observed incidence at terminal euthanasia.
[h]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend is indicated by N.
[i]Historical control incidence for other 2-year studies (all routes): 3/50, 2/50, 2/50.
[j]Historical control incidence for other 2-year studies (all routes): 0/50, 1/50, 0/50.
[k]Historical control incidence for other 2-year studies (all routes): 12/50, 8/50, 14/50.
[l]Historical control incidence for other 2-year studies (all routes): 3/50, 0/50, 0/49.

Prostate Gland: There were increased incidences of adenoma and adenoma or carcinoma (combined) in the 3 W/kg groups compared to sham controls, but the increase was not statistically significant (Table 23, Table A-1, and Table A-2). A single carcinoma occurred in the 3 W/kg group. Prostate gland carcinomas are rare neoplasms in rats, with a mean historical control incidence of 0/240 in Hsd:Sprague Dawley® SD® rats (Table A-3), 0.57% in Wistar Han rats (range 0%–2%), and 0.43% in F344/N rats (range 0%–4%); the combined incidence in the 3 W/kg group exceeded the historical control ranges for all rat strains that have been used by the National Toxicology Program[196]. The incidences and severities of epithelial hyperplasia were slightly increased in all exposed groups (Table 23 and Table A-9). Prostate gland adenomas were expansile lesions that filled the lumen of at least one acinus and compressed the adjacent tissue. The cuboidal to columnar cells formed papillary or cribriform patterns, and there was little cellular pleomorphism.

Pituitary Gland (Pars Distalis): There were increased incidences of adenoma in all exposed male groups compared to the sham controls, but none were statistically significant (Table 24, Table A-1, and Table A-2). In females, the incidences of adenoma in the 1.5 and 6 W/kg groups were significantly decreased (Table 24, Table B-1, and Table B-2). The incidences and severities of hyperplasia in exposed groups of males and females were similar to those of the sham controls (Table 24, Table A-9, and Table B-7).

Table 23. Incidences of Neoplasms and Non-neoplastic Lesions of the Prostate Gland in Male Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Number Examined Microscopically	90	90	90	90
Epithelium, Hyperplasia[a]	5 (1.2)[b]	13 (1.6)	11 (1.9)	11 (2.4)
Adenoma[c]				
Overall rate[d]	2/90 (2%)	2/90 (2%)	6/90 (7%)	3/90 (3%)
Litters rate[e]	2/35 (6%)	2/35 (6%)	5/35 (14%)	3/35 (9%)
Adjusted rate[f]	3.0%	2.7%	7.7%	3.9%
Terminal rate[g]	1/25 (4%)	0/45 (0%)	6/50 (12%)	3/60 (5%)
First incidence (days)	642	591	730 (T)	730 (T)
Rao-Scott adjusted Poly-3 test[h]	P = 0.419	P = 0.625N	P = 0.224	P = 0.566
Carcinoma[c]	0	0	1	0
Adenoma or Carcinoma[c]				
Overall rate	2/90 (2%)	2/90 (2%)	7/90 (8%)	3/90 (3%)
Litters rate	2/35 (6%)	2/35 (6%)	6/35 (17%)	3/35 (9%)
Adjusted rate	3.0%	2.7%	9.0%	3.9%
Terminal rate	1/25 (4%)	0/45 (0%)	6/50 (12%)	3/60 (5%)
First incidence (days)	642	591	717	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.412	P = 0.626N	P = 0.161	P = 0.566

(T) = terminal euthanasia.
[a]Number of animals with lesion.
[b]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.
[c]Historical control incidence for other 2-year studies (all routes): 0/50, 0/50, 0/50.
[d]Number of animals with neoplasm per number of animals with prostate gland examined microscopically.
[e]Number of litters with animals with neoplasm per number of litters with prostate gland examined microscopically.
[f]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[g]Observed incidence at terminal euthanasia.
[h]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A lower incidence in an exposure group is indicated by N.

Table 24. Incidences of Neoplasms and Non-neoplastic Lesions of the Pituitary Gland (Pars Distalis) in Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	89	90	90	90
Hyperplasia[a]	32 (2.4)[b]	34 (2.4)	35 (2.4)	32 (2.2)
Cyst	5	9	15*	16*
Adenoma, Multiple	0	0	1	0
Adenoma (includes multiple)[c]				
Overall rate[d]	17/89 (19%)	28/90 (31%)	26/90 (29%)	26/90 (29%)
Litters rate[e]	13/35 (37%)	23/35 (66%)	19/35 (54%)	22/35 (63%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adjusted rate[f]	24.9%	35.2%	32.2%	32.4%
Terminal rate[g]	5/25 (20%)	15/45 (33%)	17/50 (34%)	19/60 (32%)
First incidence (days)	527	309	537	384
Rao-Scott adjusted Poly-3 test[h]	P = 0.301	P = 0.126	P = 0.216	P = 0.210
Female				
Number Examined Microscopically	90	90	90	90
Hyperplasia	20 (2.5)	26 (2.0)	22 (1.9)	22 (2.0)
Adenoma, Multiple	1	3	3	0
Adenoma (includes multiple)[i]				
Overall rate	43/90 (48%)	33/90 (37%)	38/90 (42%)	32/90 (36%)
Litters rate	28/35 (80%)	24/35 (69%)	26/35 (74%)	23/35 (66%)
Adjusted rate	57.1%	42.5%	51.2%	41.6%
Terminal rate	28/48 (58%)	23/53 (43%)	24/48 (50%)	24/57 (42%)
First incidence (days)	464	578	545	565
Rao-Scott adjusted Poly-3 test	P = 0.077N	P = 0.049N	P = 0.283N	P = 0.038N

*Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted Poly-3 test.
[a]Number of animals with lesion.
[b]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.
[c]Historical control incidence for other 2-year studies (all routes): 14/50, 5/50, 11/50.
[d]Number of animals with neoplasm per number of animals with pituitary gland examined microscopically.
[e]Number of litters with animals with neoplasm per number of litters with pituitary gland examined microscopically.
[f]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[g]Observed incidence at terminal euthanasia.
[h]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
[i]Historical control incidence for other 2-year studies (all routes): 19/50, 18/50, 18/50.

Adenomas were characterized by a well-delineated mass composed of solid sheets of cells that compressed the adjacent tissue. Cells were often hypertrophied and cellular atypia and pleomorphism were not uncommon. Vascular patterns were often altered. The incidences of cysts in males also increased with increasing exposure SAR; the incidences were statistically significant by the Rao-Scott adjusted Poly-3 test in the 3 and 6 W/kg exposure groups (Table 24 and Table B-7). Cysts are generally considered developmental abnormalities and the toxicologic significance of these is uncertain.

Pancreatic Islets: In males, there were increased incidences of adenoma or carcinoma (combined) in all exposed groups, but only the incidence in the 1.5 W/kg group was significant (Table 25, Table A-1, and Table A-2). There were also increased incidences of adenoma in all exposed groups and of carcinoma in the 1.5 and 3 W/kg groups, but they were not statistically significant. In females, the incidences of adenoma and carcinoma were similar to those in the sham controls (Table B-1 and Table B-2). There were decreased incidences of hyperplasia in all exposed male and female groups, and the decreases were statistically significant in 1.5 and 3 W/kg males and 1.5 W/kg females (Table 25, Table A-9, and Table B-7).

Table 25. Incidences of Neoplasms and Non-neoplastic Lesions of the Pancreatic Islets in Male Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Number Examined Microscopically	90	89	86	85
Hyperplasia[a]	12 (1.5)[b]	5* (2.8)	5* (2.0)	7 (1.7)
Adenoma, Multiple	0	2	1	1
Adenoma (includes multiple)[c]				
Overall rate[d]	5/90 (6%)	14/89 (16%)	10/86 (12%)	11/85 (13%)
Litters rate[e]	5/35 (14%)	12/35 (34%)	9/35 (26%)	11/35 (31%)
Adjusted rate[f]	7.6%	18.5%	13.2%	14.8%
Terminal rate[g]	2/25 (8%)	10/45 (22%)	9/50 (18%)	11/60 (18%)
First incidence (days)	624	531	677	730 (T)
Rao-Scott adjusted Poly-3 test[h]	P = 0.282	P = 0.051	P = 0.204	P = 0.140
Carcinoma, Multiple	0	2	0	0
Carcinoma (includes multiple)[i]				
Overall rate	8/90 (9%)	15/89 (17%)	10/86 (12%)	5/85 (6%)
Litters rate	8/35 (23%)	12/35 (34%)	10/35 (29%)	4/35 (11%)
Adjusted rate	12.0%	19.7%	13.1%	6.7%
Terminal rate	3/25 (12%)	7/45 (16%)	8/50 (16%)	4/60 (7%)
First incidence (days)	663	531	537	544
Rao-Scott adjusted Poly-3 test	P = 0.088N	P = 0.173	P = 0.517	P = 0.220N
Adenoma or Carcinoma[c]				
Overall rate	13/90 (14%)	27/89 (30%)	19/86 (22%)	16/85 (19%)
Litters rate	12/35 (34%)	19/35 (54%)	17/35 (50%)	14/35 (40%)
Adjusted rate	19.4%	35.2%	24.8%	21.3%
Terminal rate	5/25 (20%)	16/45 (36%)	16/50 (32%)	15/60 (25%)
First incidence (days)	624	531	537	544
Rao-Scott adjusted Poly-3 test	P = 0.344N	P = 0.032	P = 0.282	P = 0.462

*Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted Poly-3 test.
(T) = terminal euthanasia.
[a]Number of animals with lesion.
[b]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.
[c]Historical control incidence for other 2-year studies (all routes): 3/50, 2/50, 8/50.
[d]Number of animals with neoplasm per number of animals with pancreatic islets examined microscopically
[e]Number of litters with animals with neoplasm per number of litters with pancreatic islets examined microscopically.
[f]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[g]Observed incidence at terminal euthanasia.
[h]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
[i]Historical control incidence for other 2-year studies (all routes): 0/50, 0/50, 0/50The adenomas were well circumscribed, occasionally encapsulated nodules that typically compressed the surrounding pancreatic tissue. The polygonal cells were arranged in cords or small nests and there was minimal to mild cellular pleomorphism in some of the larger masses.

Thyroid Gland: In females, the incidences of C-cell hyperplasia were significantly increased in all exposed groups compared to the sham controls (Table 26 and Table B-7). In males, there was a slightly increased incidence of C-cell hyperplasia in the 1.5 W/kg group, but the increase was not significant (Table 26 and Table A-9).

Adrenal Cortex: The incidence of hypertrophy was significantly increased in 6.0 W/kg males compared to the sham controls (Table 27 and Table A-9). However, the average severity of the lesion did not increase with increasing exposure concentration.

Table 26. Incidences of C-Cell Hyperplasia of the Thyroid Gland in Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	89	89	89	87
C-Cell, Hyperplasia[a]	16 (1.8)[b]	24 (1.9)	18 (1.9)	14 (1.6)
Female				
Number Examined Microscopically	90	88	90	88
C-Cell, Hyperplasia	28 (2.3)	49** (1.6)	45** (1.8)	43* (1.7)

*Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted Poly-3 test.
**$P \leq 0.01$.
[a]Number of animals with lesion.
[b]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.

Table 27. Incidences of Non-neoplastic Lesions of the Adrenal Cortex in Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	90	90	90	88
Hyperplasia[a]	47 (1.7)[b]	46 (1.8)	46 (1.8)	45 (1.9)
Hypertrophy	35 (1.5)	43 (1.6)	50 (1.4)	54* (1.3)
Vacuolation, Cytoplasmic	20 (1.5)	32 (1.4)	25 (1.6)	22 (1.3)
Female				
Number Examined Microscopically	90	90	89	90
Hyperplasia	14 (1.9)	26 (1.8)	40** (1.9)	26* (1.6)
Hypertrophy	52 (1.5)	54 (1.8)	51 (1.8)	56 (1.5)
Vacuolation, Cytoplasmic	18 (1.5)	21 (1.4)	11 (1.6)	8* (1.1)

*Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted Poly-3 test.
**$P \leq 0.01$.
[a]Number of animals with lesion.
[b]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.

There was a similar finding in the CDMA-exposed males, but there was no such effect in females exposed to either modulation. In females, there were significantly increased incidences of adrenal cortical hyperplasia in the 3 and 6 W/kg groups, but the average severity was similar

between exposed and sham control groups (Table 27 and Table B-7). There was a similar response in the CDMA-exposed females, but not in the males exposed to either modulation. In 6 W/kg females, there was a significantly decreased incidence of cytoplasmic vacuolation and the average severity was also slightly decreased for this lesion. The incidences of this lesion in exposed males were similar to those in the sham controls.

Kidney and Other Organs: The severity of chronic progressive nephropathy was lower in all exposed male groups compared to the sham controls (Table 28). There were decreased incidences in a number of lesions in other organs in exposed male groups, some statistically significant, that were thought to be secondary to the chronic progressive nephropathy, either directly or indirectly (Table 28 and Table A-9). These lesions included hyperplasia of the parathyroid gland; mineral in the blood vessels in the cecum, colon, liver, mesentery, pancreas, salivary glands, brain, heart, kidney, skeletal muscle, glandular stomach, and aorta; fibrous osteodystrophy of bone; polyarteritis nodosa (chronic active inflammation of the blood vessels) of the epididymis, testis, cecum, liver, pancreas, salivary glands, and thymus; germ cell degeneration of the testis; edema, erosion, epithelial regeneration, acute inflammation, chronic active inflammation, and ulcer of the cecum; epithelial regeneration of the colon; red pulp atrophy and white pulp atrophy of the spleen; and exfoliated germ cell and hypospermia of the epididymis.

Chronic progressive nephropathy (Plate 12) in the 2-year study was characterized by dilated tubules with flattened epithelium, primarily in the cortex, but extending into the medulla. Many tubules were atrophied, and some had hyperplastic epithelium. The basement membranes around the renal tubules in the cortex were variably thickened, some markedly so. There was some fibrosis scattered throughout the cortical interstitium, with scattered mononuclear inflammatory cells. In more severe cases, there was fibrosis of the Bowman's capsule around the glomeruli and varying degrees of glomerular sclerosis. Severity was based on the percentage of the kidney that was affected.

Other Lesions: In females, there was a significant negative trend (P = 0.038) in the incidences of mammary gland adenoma, and the incidences in the 3 and 6 W/kg groups were decreased but not significantly (Table B-2). The incidence in the sham controls (9%) was at the high end of the historical control range for this neoplasm in Hsd:Sprague Dawley® SD® rats, and the incidences in the 3 and 6 W/kg groups (2%) were within the historical control range (15/240 [5.7% ± 4.0%], range 0%-9%). The biological significance of these findings is unclear.

In males, the incidences of acinar hyperplasia of the pancreas were significantly decreased in the 3 and 6 W/kg groups compared to sham controls (Table A-9). The incidences of decreased secretory fluid in the seminal vesicle were significantly decreased in all exposed groups compared to sham controls (Table A-9). The incidences of both lesions decreased in an exposure-related fashion; however, the biological significance of these decreases is unclear.

Table 28. Incidences of Non-neoplastic Lesions Associated with the Decreased Severity of Chronic Progressive Nephropathy of the Kidney in Male Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Kidney[a]	90	90	90	90
Nephropathy, Chronic Progressive[b]	88 (3.7)[c]	89 (3.2)	90 (2.9)	89 (2.6)
Aorta	90	90	90	90
Mineral	30 (2.1)	7** (2.3)	12** (1.6)	6** (1.5)
Bone	90	90	90	90
Fibrous Osteodystrophy	46 (1.4)	18** (1.1)	14** (1.0)	6** (1.5)
Brain	90	90	90	90
Mineral	5 (1.0)	4 (1.0)	6 (1.2)	2 (1.0)
Epididymis	90	90	90	90
Artery, Inflammation, Chronic Active	2 (2.5)	2 (2.0)	1 (4.0)	2 (2.5)
Exfoliated Germ Cell	51 (1.9)	26** (1.5)	29** (1.4)	15** (1.5)
Hypospermia	28 (3.4)	20 (3.2)	23 (3.0)	8** (3.3)
Heart	90	90	90	90
Artery, Mineral	20 (2.5)	7** (1.9)	3** (2.0)	2** (1.5)
Intestine Large, Cecum	75	75	79	80
Artery, Inflammation, Chronic Active	20 (2.1)	9* (2.0)	5** (1.8)	6** (1.8)
Artery, Mineral	1 (2.0)	0	0	0
Edema	11 (2.0)	1** (2.0)	0**	4 (1.8)
Epithelium, Regeneration	14 (2.4)	0**	0**	2** (2.5)
Erosion	10 (2.5)	0**	0**	3 (2.0)
Inflammation, Acute	10 (2.8)	1* (2.0)	0**	2* (1.5)
Inflammation, Chronic Active	1 (3.0)	0	0	0
Ulcer	6 (2.3)	0	0*	0*
Intestine Large, Colon	81	83	81	82
Artery, Mineral	2 (2.0)	0	0	0
Epithelium, Regeneration	5 (2.6)	0	0	2 (1.0)
Intestine Large, Rectum	83	81	85	87
Epithelium, Regeneration	3 (2.3)	0	0	0
Kidney	90	90	90	90
Artery, Mineral	2 (2.0)	1 (1.0)	0	0
Liver	90	90	90	90

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Artery, Inflammation, Chronic Active	2 (3.5)	5 (2.4)	1 (1.0)	0
Artery, Mineral	1 (1.0)	0	0	0
Mesentery	39	19	17	7
Artery, Mineral	21 (2.1)	4* (2.3)	5 (2.6)	2 (1.5)
Pancreas	90	89	88	86
Artery, Inflammation, Chronic Active	48 (2.3)	28**(2.1)	26** (2.4)	14** (2.0)
Artery, Mineral	11 (1.8)	3* (1.7)	3* (2.0)	1** (3.0)
Parathyroid Gland	83	87	87	81
Hyperplasia	51 (2.5)	35** (2.0)	46 (2.0)	28** (1.6)
Salivary Glands	90	90	90	90
Artery, Inflammation, Chronic Active	11 (2.5)	7 (2.6)	3* (2.7)	1** (3.0)
Artery, Mineral	2 (2.5)	0	0	0
Skeletal Muscle	90	90	90	90
Mineral	2 (1.0)	0	0	0
Spleen	90	90	89	90
Red Pulp, Atrophy	26 (2.2)	10** (2.0)	10** (2.4)	3** (2.3)
White Pulp, Atrophy	30 (2.1)	16** (1.8)	13** (1.8)	11** (2.1)
Stomach, Glandular	86	88	87	86
Mineral	31 (2.5)	7** (2.9)	8** (2.4)	4** (2.5)
Testis	90	90	90	90
Artery, Inflammation, Chronic Active	52 (2.9)	40* (2.9)	37** (2.9)	20** (2.7)
Germ Cell, Degeneration	51 (2.3)	35* (2.2)	42 (2.1)	20** (2.0)
Thymus	88	86	88	86
Artery, Inflammation, Chronic Active	6 (2.7)	3 (3.0)	2 (2.5)	1 (2.0)

*Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted Poly-3 test.
*$P \leq 0.01$.
[a]Number of animals with tissue examined microscopically.
[b]Number of animals with lesion.
[c]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.

CDMA

Twenty-eight-day Study

Perinatal Study

No exposure-related effects were observed on survival or littering rates (littering/pregnant ratio) (Table 29). A single incidence of whole litter resorption was observed in the 9 W/kg group, and it was unclear if this was related to exposure due to the low incidence. Gestation body weights were unaffected by exposure to CDMA (Table 30). An overall (GD 1 through 16) lower body weight gain of 11% compared to sham controls was observed.

Table 29. Summary of Disposition during Perinatal Exposure and F_1 Allocation in the 28-day Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Time-mated Females	20	20	20	20
Pregnant Females	20	17	17	16
Nonpregnant Females	0	3	3	3
Pregnant Dams Not Delivering	0	0	0	1
Died	0	0	0	0
Littered	20	19	18	20
Pregnant/Mated Percentage[a]	100.0%	85.0%	85.0%	80.0%
Littered/Pregnant Percentage[a]	100.0%	100.0%	100.0%	94.1%
Litters Removed (Insufficient Size) (PND 4)	0	0	0	0
Litters Post Standardization (PND 4)	20	17	17	16
Weaned/Sex (PND 21)[b]	30	30	30	30

[a]Number in the numerator as proportion of the number in the denominator was tested using the Fisher exact test for pairwise comparisons against the sham control group.
[b]Total number of weaned animals per sex from 10 litters.

Table 30. Mean Body Weights and Mean Body Weight Gains of F_0 Female Rats during Gestation in the 28-day Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Gestation Day				
6	238.3 ± 2.2 (20)b	237.2 ± 1.8 (17)	236.9 ± 2.6 (17)	237.8 ± 2.2 (16)
9	250.7 ± 2.4 (20)	250.4 ± 2.4 (17)	252.3 ± 2.8 (17)	250.4 ± 2.1 (16)
12	266.2 ± 2.5 (20)	265.1 ± 2.3 (17)	266.6 ± 3.4 (17)	263.8 ± 2.5 (16)
15	282.5 ± 2.9 (20)	281.9 ± 2.7 (17)	283.5 ± 3.6 (17)	279.8 ± 2.7 (16)
18	319.3 ± 3.0 (20)	316.9 ± 2.3 (17)	318.0 ± 4.8 (17)	312.2 ± 3.1 (16)
21	366.4 ± 4.3 (20)▲	359.7 ± 3.5 (17)	360.0 ± 6.9 (17)	352.0 ± 3.6 (16)

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Gestation Day Interval				
6–9	12.5 ± 1.2 (20)	13.2 ± 0.9 (17)	15.4 ± 0.9 (17)	12.6 ± 0.8 (16)
9–12	15.5 ± 1.1 (20)	14.7 ± 0.9 (17)	14.3 ± 1.3 (17)	13.4 ± 0.8 (16)
2–15	16.3 ± 1.0 (20)	16.8 ± 0.9 (17)	16.9 ± 1.3 (17)	16.0 ± 0.7 (16)
15–18	36.7 ± 0.9 (20)▲	35.0 ± 1.1 (17)	34.5 ± 1.9 (17)	32.4 ± 0.9 (16)*
18–21	47.1 ± 1.8 (20)▲▲	42.8 ± 1.6 (17)	42.0 ± 3.0 (17)	39.7 ± 1.4 (16)*
6–21	128.1 ± 3.3 (20)▲	122.5 ± 2.7 (17)	123.1 ± 6.2 (17)	114.2 ± 2.3 (16)*

▲Significant trend ($P \leq 0.05$).
▲▲$P \leq 0.01$.
*Significantly different ($P \leq 0.05$) from the sham control group.
[a]Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.
[b]Number of dams.

Total and live litter size on PND 1 was unaffected by exposure and there was no statistically significant effect on live litter size throughout lactation (Table 31). How-ever, there were higher numbers of dead pups in the exposed groups from PND 1 to 4 and in the 6 and 9 W/kg groups from PND 5 to 21 (Table 32).

Table 31. Mean Number of Surviving F_1 Male and Female Rats during Lactation in the 28-day Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Total Pups per Litter				
PND 1	11.95 ± 0.38 (20)b	11.76 ± 0.30 (17)	11.18 ± 1.03 (17)	11.88 ± 0.48 (16)
Live Pups per Litter				
PND 1	11.90 ± 0.39 (20)	11.65 ± 0.32 (17)	10.82 ± 1.04 (17)	11.63 ± 0.50 (16)
PND 4 (Preculling)	11.85 ± 0.39 (20)	11.47 ± 0.33 (17)	10.47 ± 1.09 (17)	11.38 ± 0.56 (16)
PND 4 (Postculling)	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	7.06 ± 0.57 (17)	8.13 ± 0.09 (16)
PND 7	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	6.94 ± 0.57 (17)	7.75 ± 0.39 (16)
PND 10	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	6.88 ± 0.61 (17)	7.63 ± 0.52 (16)
PND 14	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	6.82 ± 0.64 (17)	7.63 ± 0.52 (16)
PND 17	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	6.82 ± 0.64 (17)	7.63 ± 0.52 (16)
PND 21	7.95 ± 0.05 (20)	8.00 ± 0.00 (17)	6.82 ± 0.64 (17)	7.63 ± 0.52 (16)
Live Males per Litter				
PND 1	6.10 ± 0.42 (20)	5.71 ± 0.43 (17)	5.00 ± 0.67 (17)	6.44 ± 0.47 (16)
PND 4 (Preculling)	6.05 ± 0.41 (20)	5.82 ± 0.50 (17)	5.12 ± 0.63 (17)	6.25 ± 0.52 (16)
PND 4 (Postculling)	4.00 ± 0.15 (20)	4.18 ± 0.23 (17)	3.53 ± 0.27 (17)	4.50 ± 0.37 (16)
Live Females per Litter				
PND 1	5.80 ± 0.42 (20)	5.94 ± 0.52 (17)	5.82 ± 0.69 (17)	5.19 ± 0.56 (16)
PND 4 (Preculling)	5.80 ± 0.43 (20)	5.65 ± 0.58 (17)	5.35 ± 0.65 (17)	5.13 ± 0.59 (16)
PND 4 (Postculling)	3.95 ± 0.14 (20)	3.82 ± 0.23 (17)	3.53 ± 0.33 (17)	3.63 ± 0.36 (16)

[a]All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.
[b]Number of dams.

Table 32. Offspring Mortality and Survival Ratio of Rats during Lactation in the 28-day Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR[a]

Pup Survival per Litter	Sham Control	3 W/kg	6 W/kg	9 W/kg
Total Dead PND 1–4	2 (238/20)[b]	5 (198/17)	12 (184/17)	8 (186/16)
Total Dead PND 5–21	0 (159/20)	0 (136/17)	4 (120/17)	8 (130/16)
Dead/Litter PND 1–4	0.100 ± 0.069 (20)	0.294 ± 0.143 (17)	0.706 ± 0.318 (17)	0.500 ± 0.204 (16)
Dead/Litter PND 4–21	0.000 ± 0.000 (20)	0.000 ± 0.000 (17)	0.235 ± 0.136 (17)	0.500 ± 0.500 (16)
Survival Ratio PND 1–4[c]	0.996 ± 0.004 (20)	0.985 ± 0.008 (17)	0.942 ± 0.031 (17)	0.975 ± 0.011 (16)
Survival Ratio PND 4–21[d]	1.000 ± 0.000 (20)	1.000 ± 0.000 (17)	0.868 ± 0.081 (17)	0.938 ± 0.063 (16)

[a]All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.
[b]Number of pups/number of dams.
[c]Number of pups preculling on PND 4/total number of viable pups on PND 1 (does not include pups dead on PND 1).
[d]Number of pups alive on PND 21/number of pups at postculling on PND 4.

During the lactation period, 9 W/kg dams had decreased weight gains, and their body weights were 5% to 12% lower than those in the sham controls from PND 7 through 21 (Table 33). F_1 body weights were 8% lower starting on PND 1 in the 9 W/kg group whether male, female, or combined when not adjusted or adjusted for litter size (Table 34). As lactation progressed, the adjusted pup weights (combined) were up to 23% lower in the 9 W/kg group and up to 16% lower in the 6 W/kg group compared to sham controls. The magnitude of the effect was consistent between males and females with a recovery in the 6 W/kg group on PND 14 and 21.

Table 33. Mean Body Weights and Mean Body Weight Gains of F_0 Female Rats during Lactation in the 28-day Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Postnatal Day				
1	272.7 ± 2.7 (20)[b]	268.9 ± 2.4 (17)	271.7 ± 4.1 (17)	263.8 ± 2.6 (16)
4	263.8 ± 3.6 (20)	264.1 ± 2.9 (16)	269.3 ± 4.1 (17)	264.6 ± 2.1 (16)
7	284.1 ± 2.7 (20)▲▲	282.5 ± 2.7 (17)	282.1 ± 4.0 (17)	269.6 ± 1.9 (16)**
14	292.4 ± 2.6 (20)▲▲	293.3 ± 2.7 (17)	284.9 ± 4.1 (17)	264.1 ± 3.1 (16)**
21	279.7 ± 3.5 (20)▲▲	282.6 ± 3.0 (17)	272.9 ± 3.7 (17)	245.9 ± 3.1 (16)**
Postnatal Day Interval				
1–4	–8.9 ± 3.2 (20)▲	–3.8 ± 3.3 (16)	–2.4 ± 3.1 (17)	0.8 ± 1.4 (16)
4–7	20.2 ± 2.3 (20)▲▲	18.1 ± 3.2 (16)	12.8 ± 2.2 (17)*	5.1 ± 1.2 (16)**
7–14	8.3 ± 1.4 (20)▲▲	10.8 ± 1.8 (17)	2.9 ± 2.3 (17)*	–5.5 ± 2.0 (16)**
14–21	–12.7 ± 3.5 (20)	–10.7 ± 2.8 (17)	–12.1 ± 2.7 (17)	–18.2 ± 2.4 (16)
1–21	7.0 ± 3.8 (20)▲▲	13.7 ± 2.7 (17)	1.1 ± 2.8 (17)	–17.9 ± 3.3 (16)**

▲Significant trend ($P \leq 0.05$).
▲▲$P \leq 0.01$.
*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.
[b]Number of dams.

Table 34. Adjusted Mean Body Weights of F_1 Male and Female Rats during Lactation in the 28-day Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Adjusted Male Live Pup Weight				
PND 1[b]	6.73 ± 0.10 (20)▲▲[c]	6.76 ± 0.11 (17)	6.52 ± 0.10 (17)	6.16 ± 0.11 (16)▲▲
PND 4 (Preculling)	9.78 ± 0.14 (121/20)**[d]	9.43 ± 0.16 (99/17)	8.78 ± 0.30 (87/17)**	8.14 ± 0.28 (100/16)**
PND 4 (Postculling)	9.84 ± 0.14 (81/20)**	9.57 ± 0.15 (69/17)	8.82 ± 0.30 (61/17)**	8.19 ± 0.27 (71/16)**
PND 7	16.33 ± 0.19 (81/20)**	15.30 ± 0.27 (69/17)	13.64 ± 0.67 (61/17)**	12.52 ± 0.61 (64/16)**
PND 14	31.61 ± 0.42 (81/20)**	30.91 ± 0.41 (69/17)	29.73 ± 0.47 (59/15)**	26.75 ± 0.47 (63/15)**
PND 21	53.33 ± 0.72 (81/20)**	52.57 ± 0.71 (69/17)	51.42 ± 0.75 (59/15)	45.11 ± 0.84 (63/15)**
Adjusted Female Live Pup Weight				
PND 1[b]	6.30 ± 0.10 (20)▲▲	6.43 ± 0.11 (17)	6.14 ± 0.12 (16)	5.77 ± 0.13 (14)▲▲
PND 4 (Preculling)	9.11 ± 0.16 (116/20)**	8.85 ± 0.15 (96/17)	8.75 ± 0.17 (91/15)	7.86 ± 0.18 (82/14)**
PND 4 (Postculling)	9.19 ± 0.16 (79/20)**	9.00 ± 0.15 (67/17)	8.78 ± 0.17 (59/15)	7.97 ± 0.19 (57/14)**

	Sham Control	3 W/kg	6 W/kg	9 W/kg
PND 7	15.26 ± 0.24 (79/20)**	14.63 ± 0.24 (67/17)	13.90 ± 0.29 (57/15)**	12.29 ± 0.36 (57/14)**
PND 14	29.82 ± 0.40 (79/20)**	29.78 ± 0.38 (67/17)	28.57 ± 0.41 (57/15)	25.66 ± 0.51 (57/14)**
PND 21	49.87 ± 0.73 (79/20)**	49.50 ± 0.54 (67/17)	49.03 ± 0.64 (57/15)	43.24 ± 0.87 (57/14)**
Adjusted Combined Live Pup Weight				
PND 1[b]	6.51 ± 0.10 (20)▲▲	6.60 ± 0.11 (17)	6.33 ± 0.11 (17)	5.99 ± 0.10 (16)▲▲
PND 4 (Preculling)	9.45 ± 0.13 (237/20)**	9.17 ± 0.15 (195/17)	8.51 ± 0.34 (178/17)*	7.90 ± 0.25 (182/16)**
PND 4 (Postculling)	9.52 ± 0.13 (160/20)**	9.30 ± 0.14 (136/17)	8.57 ± 0.33 (120/17)*	8.00 ± 0.25 (128/16)**
PND 7	15.79 ± 0.19 (160/20)**	14.97 ± 0.23 (136/17)	13.29 ± 0.69 (118/17)**	12.21 ± 0.58 (121/16)**
PND 14	30.70 ± 0.39 (160/20)**	30.33 ± 0.37 (136/17)	29.17 ± 0.43 (116/15)*	26.29 ± 0.46 (120/15)**
PND 21	51.57 ± 0.67 (160/20)**	51.07 ± 0.59 (136/17)	50.29 ± 0.68 (116/15)	44.31 ± 0.82 (120/15)**

▲▲Significantly different ($P \leq 0.01$) for PND 1 endpoint (statistical significance in the sham control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant pairwise comparison against the sham control group).
*Significantly different ($P \leq 0.05$) for PNDs after PND 1 endpoints (statistical significance in the sham control group column indicates a significant trend test; statistical significance in the treatment group column indicates a significant pairwise comparison against the sham control group).
**$P \leq 0.01$.
[a]Body weights in grams. Data are displayed as mean ± standard error. PND = postnatal day. Values listed as PND 1 refer to the total pup weight divided by the number of pups in litter at PND 1, and the statistical analysis performed is by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests. Values listed for all other days refer to individual pups, and the statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnet-Hsu adjustment method. Individual pup body weights first adjusted for live PND 1 litter size via the analysis of covariance.
[b]Values listed as PND 1 refer to the total pup weight divided by the number of pups in litter at PND 1.
[c]Number of dams.
[d]Number of pups/number of dams.

Postnatal Study

All rats survived to the end of the study (Table 35). In males, there were lower mean body weights in the 3 (5% to 7%) and 9 W/kg groups (13% to 15%) compared to the sham controls at all time points including terminal sacrifice (except day 1 for the 3 W/kg group) (Table 35 and Figure 9). In 6 W/kg males, mean body weights were lower (5% to 7%) at all time points except terminal sacrifice. Mean body weight gains were lower (6% to 14%) in all three male exposed groups compared to sham controls (data not presented). In females, mean body weights were lower on days 1, 8, 15, and 22 in the 9 W/kg group (7% to 12%), on days 1, 8, and 15 in the 6 W/kg group (1% to 2%), and on day 8 in the 3 W/kg group (4%). However, at terminal sacrifice, the mean body weights of all exposed female groups were similar to those of the sham controls. Mean body weight gains of all exposed female groups were similar to that of the sham controls. There were no notable clinical observations in any groups of either sex during the study.

Table 35. Mean Body Weights and Survival of Rats Exposed to CDMA-modulated Cell Phone RFR for 28 Days

Day	Sham Control		3 W/kg			6 W/kg			9 W/kg		
	Av. Wt. (g)	No. of Survivors	Av. Wt.(g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors	Av. Wt. (g)	Wt. (% of Controls)	No. of Survivors
Male											
1	60.8	10	58.4	95.9	10	58.0	95.3	10	52.9	87.0	10
8	94.9	10	89.8	94.6	10	88.8	93.6	10	80.5	84.9	10
15	144.5	10	136.1	94.1	10	134.2	92.9	10	123.6	85.5	10
22	195.3	10	182.8	93.6	10	184.6	94.5	10	169.2	86.6	10
29	248.7	10	231.1	92.9	10	236.4	95.0	10	215.0	86.4	10
Female											
1	55.9	10	53.5	95.8	10	55.7	99.7	10	49.5	88.6	10
8	83.1	10	79.5	95.6	10	81.8	98.3	10	73.4	88.3	10
15	119.8	10	114.4	95.5	10	118.5	98.9	10	107.9	90.1	10
22	146.5	10	144.6	98.7	10	148.7	101.5	10	136.9	93.4	10
30	166.5	10	162.7	97.7	10	171.7	103.1	10	159.0	95.5	10

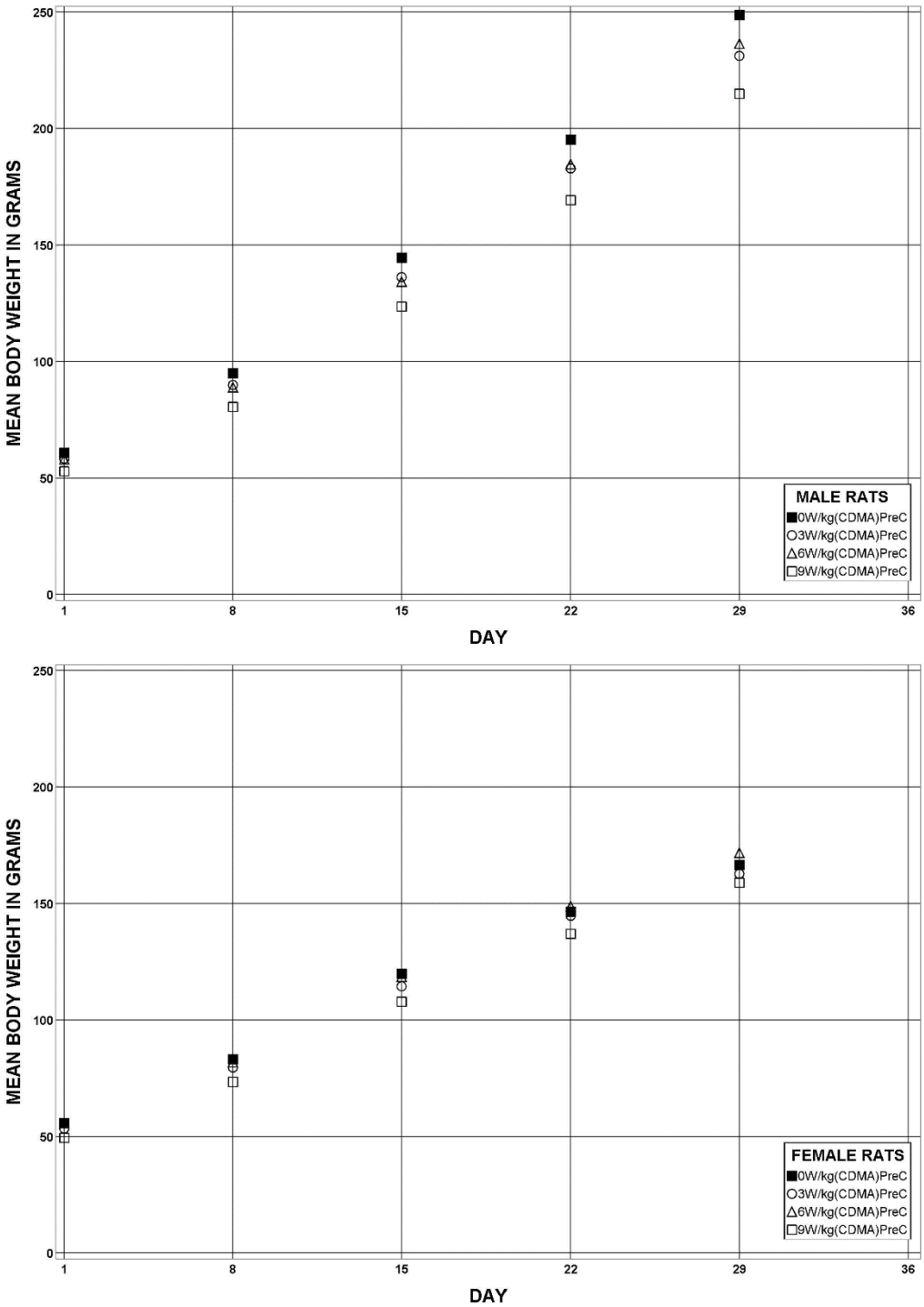

Figure 9. Growth Curves for Rats Exposed to CDMA-modulated Cell Phone RFR for 28 Days

Body temperatures were significantly, but sporadically, increased in F_0 females at several time points (Table 36). Body temperature in the F_1 groups were similar to those of the sham controls throughout the study (Table 36 and Table G-5).

Male body weights were lower in the 9 W/kg group at the end of the 28-day studies. There were a few statistically significant changes in organ weights at this exposure level that were considered secondary to the reduced body weights (Table G-6). There were no biologically significant changes in organ weights in females.

Kidney: There was a increased incidence of chronic progressive nephropathy in the 6 W/kg females compared to sham controls [sham control, 0/10; 3 W/kg, 2/10; 6 W/kg, 4/10*; 9 W/kg, 3/10; significantly different ($P \leq 0.05$) from the sham control group by the Fisher exact test]. The severity in all cases was minimal. This lesion was characterized by scattered tubular segments with basophilic epithelial cells with crowded nuclei, slightly thickened basement membranes, and occasional mononuclear inflammatory cells. Chronic progressive nephropathy was not considered to be related to treatment with CDMA-modulated cell phone RFR because there was no exposure-related response and it is a very common background lesion in rats.

Exposure Level Selection Rationale: Based on reduced maternal and pup weights, increased F_0 dam body temperature measurements at 9 W/kg in the 28-day studies, and increased body temperature in adult rats at ≥8 W/kg in the thermal pilot studies[146], the highest exposure level selected for the 2-year studies was 6 W/kg. In the thermal pilot studies and 28-day studies, exposure to 6 W/kg resulted in some increases in core body temperature, but these increases were less than 1°C. Therefore, 6 W/kg would provide an exposure adequate to challenge the animals without causing excessive heating or disruption of the thermoregulatory process. The lowest exposure level selected for the 2-year studies was 1.5 W/kg, which is close to the 1.6 W/kg maximum output limit for cell phone devices in the United States.

Table 36. Mean Body Temperatures of Rats Exposed to CDMA-modulated Cell Phone RFR for 28 Days[a]

	Sham Control		3 W/kg		6 W/kg		9 W/kg	
Day	**Temperature (°C)**	**No. Measured**	**Temperature (°C)**	**No. Measured**	**Temperature (°C)**	**No. Measured**	**Temperature (°C)**	**No. Measured**
F0 Female[b]								
GD 6	36.7 ± 0.1	10[c]	36.8 ± 0.1	9	36.6 ± 0.2	9	36.4 ± 0.2	8
GD 7	36.6 ± 0.1▲	10	36.3 ± 0.2	9	36.6 ± 0.1	9	37.2 ± 0.1*	8
GD 11	36.7 ± 0.2	10	36.4 ± 0.1	9	36.2 ± 0.2*	9	36.8 ± 0.2	8
GD 16	36.5 ± 0.1▲	10	36.5 ± 0.2	9	36.6 ± 0.1	9	37.2 ± 0.2*	8
GD 7–16[d]	36.6 ± 0.1	10	36.4 ± 0.1	9	36.4 ± 0.1	9	37.1 ± 0.1*	8
LD 1	37.7 ± 0.1	10	37.3 ± 0.2	9	37.7 ± 0.1	9	37.9 ± 0.2	8
LD 4	36.7 ± 0.1▲▲	10	37.0 ± 0.2	9	37.3 ± 0.3*	9	38.1 ± 0.2**	8
LD 7	36.8 ± 0.2▲	10	37.0 ± 0.1	9	37.4 ± 0.2	9	37.5 ± 0.3	8
LD 14	36.9 ± 0.2▲▲	10	37.0 ± 0.3	9	37.6 ± 0.1*	9	38.3 ± 0.3**	7
LD 7–14[d]	37.0 ± 0.1▲▲	10	37.1 ± 0.1	9	37.5 ± 0.1*	9	37.9 ± 0.2**	8
F1 Male[e]								
16	37.4 ± 0.2	4	37.0 ± 0.0	4	37.1 ± 0.0	4	37.2 ± 0.1	4
20	37.6 ± 0.1	4	37.2 ± 0.1	4	37.1 ± 0.1	4	37.5 ± 0.2	4
27	37.3 ± 0.2	4	36.8 ± 0.1	4	37.0 ± 0.1	4	37.4 ± 0.1	4
16–27[d]	37.5 ± 0.1	4	37.0 ± 0.0	4	37.1 ± 0.0	4	37.4 ± 0.1	4

	Sham Control		3 W/kg		6 W/kg		9 W/kg	
F1 Female[e]								
16	38.0 ± 0.3	4	37.2 ± 0.2*	3	37.4 ± 0.1	4	37.5 ± 0.1	4
20	38.1 ± 0.2▲	4	38.1 ± 0.2	4	37.5 ± 0.2	4	37.7 ± 0.2	4
27	37.9 ± 0.2	4	37.1 ± 0.1	4	37.9 ± 0.3	4	37.9 ± 0.2	4
16–27[d]	38.0 ± 0.2	4	37.5 ± 0.1	4[f]	37.6 ± 0.2	4	37.7 ± 0.1	4

▲Significant trend ($P \leq 0.05$).
▲▲$P \leq 0.01$.
*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Temperatures are given as mean ± standard error. GD = gestation day; LD = lactation day.
[b]Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.
[c]For F0 females, number measured refers to individual animals; for F1 pups, number measured refers to litters.
[d]Average of days.
[e]Statistical analysis for linear trends was performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method.
[f]There were three litters on day 16.

Two-year Study

Perinatal Exposure

No exposure-related effects were observed on pregnancy status, maternal survival, or the percent of pregnant animals that littered (Table 37). Maternal body weights during gestation were similar to those of the sham control group (Table 38). Body weight gains were generally unaffected across time intervals except in the 3 W/kg group at the GD 6 through 9 interval where weight gains were lower than that of the sham control group, but this was not considered to be exposure related.

Table 37. Summary of Disposition during Perinatal Exposure and F_1 Allocation in the Two-year Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Time-mated Females	56	56	56	56
Pregnant Females	52	50	48	49
Nonpregnant Females	4	6	8	7
Pregnant Dams Not Delivering	2	2	2	1
Died[a]	1	0	0	0
Littered	50	48	46	48
Pregnant/Mated Percentage[b]	92.9%	89.3%	85.7%	87.5%
Littered/Pregnant Percentage[b]	96.2%	96.0%	95.8%	98.0%
Litters Removed (Insufficient Size)	2	3	3	2
Litters Post Standardization	48	45	43	46
Weaned/Sex[c]	105	105	105	105

[a]One pregnant female died on GD 25 with pups in uterus.
[b]Number in the numerator as proportion of the number in the denominator was tested using the Fisher exact test for pairwise comparisons against the sham control group.
[c]Total number of weaned animals per sex from 35 litters.

Table 38. Mean Body Weights and Mean Body Weight Gains of F0 Female Rats during Gestation in the Two-year Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Gestation Day				
6	238.4 ± 1.4 (51)[b]	239.0 ± 1.3 (48)	239.9 ± 1.6 (46)	237.8 ± 1.6 (48)
9	256.2 ± 1.6 (51)	255.2 ± 1.4 (48)	254.5 ± 1.8 (46)	253.7 ± 1.7 (48)
12	270.5 ± 1.6 (51)	268.7 ± 1.4 (48)	268.8 ± 1.6 (46)	267.0 ± 1.7 (48)
15	290.0 ± 1.9 (51)	288.5 ± 1.6 (48)	289.3 ± 1.8 (46)	287.5 ± 2.0 (48)
18	332.7 ± 2.3 (51)	329.7 ± 2.1 (48)	329.1 ± 2.3 (46)	329.0 ± 2.5 (48)
21	380.2 ± 2.8 (51)	375.9 ± 3.0 (48)	375.1 ± 3.2 (46)	375.0 ± 3.1 (48)
Gestation Day Interval				
6–9	17.7 ± 0.8 (51)	16.1 ± 0.7 (48)	14.6 ± 1.0 (46)*	15.9 ± 0.7 (48)
9–12	14.3 ± 0.6 (51)	13.6 ± 0.5 (48)	14.3 ± 1.0 (46)	13.3 ± 0.6 (48)
12–15	19.6 ± 0.6 (51)	19.8 ± 0.5 (48)	20.5 ± 0.7 (46)	20.5 ± 0.5 (48)
15–18	42.7 ± 1.0 (51)	41.3 ± 1.1 (48)	39.8 ± 0.8 (46)	41.5 ± 0.8 (48)
18–21	47.5 ± 1.0 (51)	46.2 ± 1.2 (48)	46.0 ± 1.2 (46)	46.0 ± 1.0 (48)
6–21	141.7 ± 2.2 (51)	136.9 ± 2.8 (48)	135.2 ± 2.5 (46)	137.1 ± 2.3 (48)

*Significantly different ($P \leq 0.05$) from the sham control group.
[a]Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.
[b]Number of dams.

On PND 1, there were no effects on total litter size or live litter size (Table 39). However, beginning on PND 7, live litter size was decreased in the 6 W/kg groups compared to the sham control group. There was a higher incidence of pup mortality (found dead or missing and presumed cannibalized) between PNDs 4 and 21 in the 6 W/kg group that corresponded to an increase in average number of dead pups per litter and a reduced survival ratio (Table 40).

Table 39. Mean Number of Surviving F1 Male and Female Rats during Lactation in the Two-year Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Total Pups per Litter				
PND 1	12.76 ± 0.32 (50)[b]	12.42 ± 0.41 (48)	12.43 ± 0.39 (46)	12.94 ± 0.35 (48)
Live Pups per Litter				
PND 1	12.56 ± 0.40 (50)	12.33 ± 0.42 (48)	12.39 ± 0.41 (46)	12.94 ± 0.35 (48)
PND 4 (Preculling)	12.73 ± 0.30 (48)	12.72 ± 0.26 (46)	12.77 ± 0.31 (43)	12.87 ± 0.30 (46)
PND 4 (Postculling)	8.00 ± 0.00 (48)	8.00 ± 0.00 (45)	8.00 ± 0.00 (43)	8.00 ± 0.00 (46)
PND 7	8.00 ± 0.00 (48)▲▲	7.98 ± 0.02 (45)	7.98 ± 0.02 (43)	7.85 ± 0.06 (46)**
PND 10	8.00 ± 0.00 (48)▲▲	7.98 ± 0.02 (45)	7.98 ± 0.02 (43)	7.76 ± 0.08 (46)**
PND 14	8.00 ± 0.00 (48)▲▲	7.98 ± 0.02 (45)	7.98 ± 0.02 (43)	7.52 ± 0.11 (46)**
PND 17	8.00 ± 0.00 (48)▲▲	7.98 ± 0.02 (45)	7.98 ± 0.02 (43)	7.52 ± 0.11 (46)**
PND 21	8.00 ± 0.00 (48)▲▲	7.98 ± 0.02 (45)	7.98 ± 0.02 (43)	7.52 ± 0.11 (46)**
Live Males per Litter				
PND 1	6.20 ± 0.30 (50)	6.33 ± 0.30 (48)	6.07 ± 0.31 (46)	6.69 ± 0.30 (48)
PND 4 (Preculling)	6.33 ± 0.28 (48)	6.78 ± 0.24 (46)	6.26 ± 0.33 (43)	6.76 ± 0.29 (46)
PND 4 (Postculling)	3.96 ± 0.05 (48)	4.00 ± 0.06 (45)	3.95 ± 0.09 (43)	4.04 ± 0.05 (46)
Live Females per Litter				
PND 1	6.36 ± 0.28 (50)	6.00 ± 0.32 (48)	6.33 ± 0.36 (46)	6.25 ± 0.26 (48)
PND 4 (Preculling)	6.40 ± 0.25 (48)	5.93 ± 0.28 (46)	6.51 ± 0.35 (43)	6.11 ± 0.24 (46)
PND 4 (Postculling)	4.04 ± 0.05 (48)	4.00 ± 0.06 (45)	4.05 ± 0.09 (43)	3.96 ± 0.05 (46)

▲▲Significant trend ($P \leq 0.01$).
**Significantly different ($P \leq 0.01$) from the sham control group.
[a]All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests. Statistical significance in the sham control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant pairwise comparison against the sham control group.
[b]Number of dams.

Table 40. Offspring Mortality and Survival Ratio of Rats during Lactation in the Two-year Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR[a]

Pup Survival per Litter	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Total Dead PND 1–4[b]	19 (628/49)[c]	9 (592/46)	6 (570/43)	16 (621/46)
Total Dead PND 5–21	0 (348/48)	1 (360/45)	1 (344/43)	22 (368/46)
Dead/Litter PND 1–4	0.388 ± 0.193 (49)	0.196 ± 0.074 (46)	0.140 ± 0.063 (43)	0.348 ± 0.099 (46)
Dead/Litter PND 4–21	0.000 ± 0.000 (48)▲▲	0.022 ± 0.022 (45)	0.023 ± 0.023 (43)	0.478 ± 0.106 (46)**
Survival Ratio PND 1–4[d]	0.986 ± 0.005 (48)	0.991 ± 0.004 (46)	0.989 ± 0.005 (43)	0.975 ± 0.007 (46)
Survival Ratio PND 4–21[e]	1.000 ± 0.000 (48)▲▲	0.997 ± 0.003 (45)	0.997 ± 0.003 (43)	0.940 ± 0.013 (46)**

▲▲Significant trend (P ≤ 0.01).
**Significantly different (P ≤ 0.01) from the sham control group.
[a]All values shown as mean ± standard error; PND = postnatal day. Statistical analysis performed by Jonckheere's (trend) and Shirley's or Dunn's (pairwise) tests.
[b]Includes dead on PND 1. Survival information on PND 4 was not available for some non-acceptable litters, so these were excluded from the analysis.
[c]Number of pups/number of dams.
[d]Number of pups preculling on PND 4/total number of viable pups on PND 1 (does not include pups dead on PND 1).
[e]Number of pups alive on PND 21/number of pups at postculling on PND 4.

During the lactation period, maternal body weights and body weight gains (PND 1 through 21) in the 3 and 6 W/kg groups were significantly decreased (up to 3% and 7%, respectively) compared to sham controls (Table 41). At PND 1, male and female pup weights in the 6 W/kg groups were 5% to 6% less than those of the sham controls (Table 42). Male and female pup weights were also significantly decreased compared to the sham controls in the 3 W/kg groups at PND 4 and in the 6 W/kg groups at all time points. The lower weights occurred in similar magnitudes between the sexes with 5% to 6% decreases at PND 4 in the 3 W/kg group and up to 15% decreases in the 6 W/kg group at PND 7.

Table 41. Mean Body Weights and Mean Body Weight Gains of F0 Female Rats during Lactation in the Two-year Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Postnatal Day				
1	280.9 ± 2.0 (50)[b]	278.3 ± 1.7 (48)	278.2 ± 1.8 (45)	275.8 ± 2.2 (48)
4	289.7 ± 2.1 (48)▲▲	289.1 ± 2.0 (45)	286.4 ± 1.8 (43)	282.3 ± 2.0 (46)**
7	297.1 ± 2.2 (48)▲▲	297.4 ± 1.9 (45)	293.5 ± 1.8 (43)	285.3 ± 1.9 (45)**
14	314.4 ± 2.0 (48)▲▲	309.8 ± 1.9 (45)	306.6 ± 2.1 (43)*	293.5 ± 2.5 (46)**
17	313.9 ± 2.2 (48)▲▲	307.8 ± 1.9 (45)*	303.6 ± 2.1 (42)**	294.9 ± 2.4 (46)**
21	299.7 ± 2.2 (48)▲▲	293.8 ± 2.0 (45)*	291.2 ± 1.8 (43)**	277.6 ± 2.2 (46)**
Postnatal Day Interval				
1–4	9.2 ± 0.9 (48)▲▲	11.2 ± 1.0 (45)	7.5 ± 0.8 (42)	5.7 ± 1.0 (46)**
4–7	7.4 ± 1.5 (48)▲▲	8.2 ± 1.0 (45)	7.1 ± 1.0 (43)	3.4 ± 1.3 (45)*
7–14	17.4 ± 1.4 (48)▲▲	12.5 ± 1.0 (45)*	13.1 ± 1.5 (43)*	7.7 ± 1.4 (45)**
14–17	–0.6 ± 1.0 (48)	–2.0 ± 1.2 (45)	–2.6 ± 1.3 (42)	1.3 ± 1.0 (46)
17–21	–14.1 ± 1.3 (48)	–14.0 ± 1.4 (45)	–12.7 ± 1.5 (42)	–17.3 ± 1.5 (46)
1–21	19.3 ± 1.5 (48)▲▲	15.9 ± 1.7 (45)	12.2 ± 1.6 (42)**	1.0 ± 1.4 (46)**

▲▲Significant trend (P ≤ 0.01).
*Significantly different (P ≤ 0.05) from the sham control group.
**P ≤ 0.01.
[a]Body weights and body weight gains in grams. Data are displayed as mean ± standard error. Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.
[b]Number of dams.

Table 42. Adjusted Mean Body Weights of F1 Male and Female Rats during Lactation in the Two-year Perinatal and Postnatal Study of CDMA-modulated Cell Phone RFR[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adjusted Male Live Pup Weight				
PND 1[b]	7.19 ± 0.07 (49)▲▲[c]	7.02 ± 0.07 (47)	7.09 ± 0.06 (46)	6.78 ± 0.06 (48)▲▲
PND 4	10.87 ± 0.12 (190/48)**[d]	10.54 ± 0.15 (166/42)	10.35 ± 0.11 (169/43)**	9.58 ± 0.11 (178/44)**
PND 7	17.18 ± 0.19 (189/48)**	16.83 ± 0.22 (179/45)	16.82 ± 0.18 (166/42)	14.75 ± 0.25 (183/46)**
PND 14	35.20 ± 0.43 (181/46)**	33.89 ± 0.42 (179/45)	34.48 ± 0.32 (170/43)	30.81 ± 0.52 (176/46)**
PND 17	42.44 ± 0.49 (190/48)**	41.55 ± 0.50 (179/45)	41.88 ± 0.40 (170/43)	38.08 ± 0.60 (175/46)**
PND 21	58.46 ± 0.62 (190/48)**	57.30 ± 0.72 (179/45)	57.85 ± 0.55 (170/43)	52.63 ± 0.83 (176/46)**
Adjusted Female Live Pup Weight				
PND 1[b]	6.79 ± 0.06 (49)▲▲	6.65 ± 0.09 (47)	6.73 ± 0.06 (45)	6.44 ± 0.05 (48)▲▲
PND 4	10.43 ± 0.11 (194/48)**	10.16 ± 0.13 (172/43)	9.80 ± 0.12 (171/43)**	9.21 ± 0.11 (182/46)**
PND 7	16.45 ± 0.18 (194/48)**	16.16 ± 0.21 (180/45)	15.90 ± 0.20 (173/43)	14.12 ± 0.22 (174/45)**
PND 14	33.87 ± 0.39 (192/48)**	32.71 ± 0.38 (180/45)	32.91 ± 0.37 (169/42)	29.59 ± 0.50 (170/46)**
PND 17	40.80 ± 0.45 (194/48)**	39.99 ± 0.47 (180/45)	39.90 ± 0.42 (173/43)	36.58 ± 0.55 (170/46)**
PND 21	55.38 ± 0.53 (194/48)**	54.23 ± 0.64 (180/45)	54.37 ± 0.57 (173/43)	50.29 ± 0.70 (170/46)**
Adjusted Combined Live Pup Weight				
PND 1[b]	6.99 ± 0.06 (49)▲▲	6.83 ± 0.09 (48)	6.90 ± 0.06 (46)	6.62 ± 0.05 (48)▲▲
PND 4	10.65 ± 0.11 (384/48)**	10.36 ± 0.13 (338/44)	10.07 ± 0.11 (340/43)**	9.39 ± 0.10 (360/46)**
PND 7	16.80 ± 0.18 (383/48)**	16.50 ± 0.20 (359/45)	16.34 ± 0.18 (339/43)	14.45 ± 0.22 (357/46)**
PND 14	34.46 ± 0.40 (373/48)**	33.31 ± 0.38 (359/45)	33.69 ± 0.32 (339/43)	30.23 ± 0.48 (346/46)**
PND 17	41.60 ± 0.46 (384/48)**	40.78 ± 0.47 (359/45)	40.88 ± 0.39 (343/43)	37.35 ± 0.54 (345/46)**
PND 21	56.89 ± 0.56 (384/48)**	55.77 ± 0.65 (359/45)	56.10 ± 0.53 (343/43)	51.51 ± 0.71 (346/46)**

▲▲Significantly different ($P \leq 0.01$) for PND 1 endpoint (statistical significance in the sham control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant pairwise comparison against the sham control group).
**Significantly different ($P \leq 0.01$) for PNDs after PND 1 endpoints (statistical significance in the sham control group column indicates a significant trend test; statistical significance in a treatment group column indicates a significant pairwise comparison against the sham control group).
[a]Body weights in grams. Data are displayed as mean ± standard error. PND = postnatal day. Values listed as PND 1 refer to the total pup weight divided by the number of pups in litter at PND 1, and the statistical analysis performed is by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests. Values listed for all other days refer to individual pups, and the statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dose groups to sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method. Individual pup body weights first adjusted for live PND 1 litter size via the analysis of covariance.
[b]Values listed as PND 1 refer to the total pup weight divided by the number of pups in litter at PND 1.
[c]Number of dams.
[d]Number of pups/number of dams.

Postnatal Study

Survival

Estimates of 2-year survival probabilities for male and female rats are shown in Table 43 and in the Kaplan-Meier survival curves (Figure 10). In males, survival was greater in all exposed groups compared to sham controls, though it was statistically significant only in the 1.5 and 3 W/kg groups. Survival in the sham control group was 28% compared to 48%, 62%, and 48% in the 1.5, 3, and 6 W/kg groups, respectively. Decreased survival in the sham control group was largely attributed to the higher severity of chronic progressive nephropathy in the kidney. In females, there was a small, but statistically significant increase in survival in the 6 W/kg group. Survival in the sham control females was similar to that in the 1.5 and 3 W/kg groups.

Table 43. Survival of Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Animals initially in study	105	105	105	105
14-week interim evaluation[a]	15	15	15	15
Accidental death[b]	1	0	0	0
Moribund	44	24	13	6
Natural deaths	20	23	21	41
Animals surviving to study termination	25	43	56f	43
Percent probability of survival at end of study[c]	28	48	62	48
Mean survival (days)[d]	642	675	687	637
Survival analysis[e]	P = 0.070N	P = 0.005N	P < 0.001N	P = 0.072N
Female				
Animals initially in study	105	105	105	105
14-week interim evaluation[a]	15	15	15	15
Accidental death	1	0	0	0
Moribund	30	29	28	16
Natural deaths	11	15	12	13
Animals surviving to study termination	48[f]	46[g]	50	61
Percent probability of survival at end of study	54	50	56	68
Mean survival (days)	659	673	665	701
Survival analysis	P = 0.020N	P = 1.000	P = 0.841N	P = 0.037N

[a]Excluded from survival analysis.
[b]Censored in the survival analysis.
[c]Kaplan-Meier determinations.
[d]Mean of all deaths (uncensored, censored, and terminal euthanasia).
[e]The result of the life table trend test[161] is in the sham control column, and the results of the life table pairwise comparisons[160] with the sham controls are in the exposed group columns. A negative trend or lower mortality in an exposure group is indicated by N.
[f]Includes one animal that died during the last week of the study.
[g]Includes two animals that died during the last week of the study.

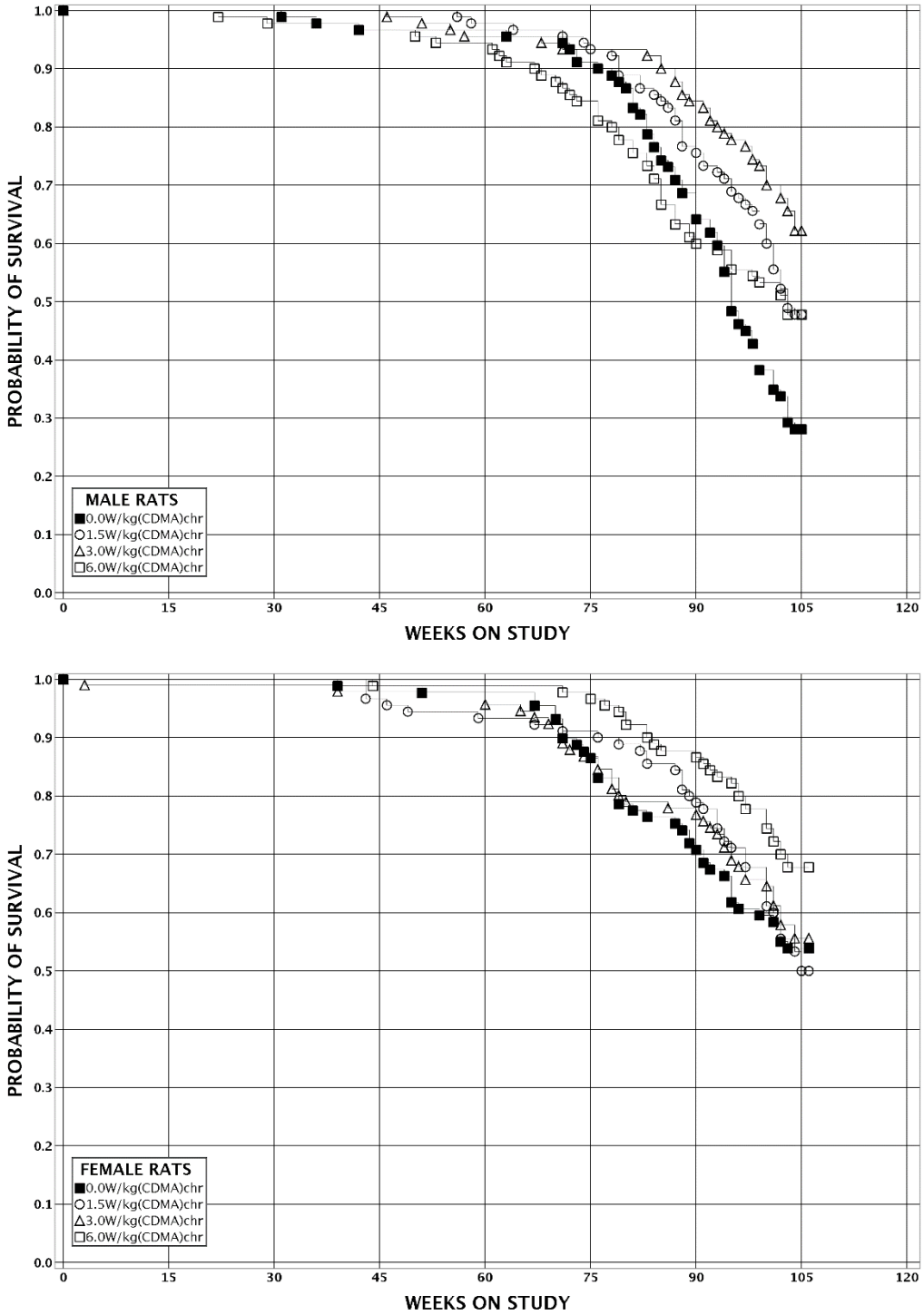

Figure 10**. Kaplan-Meier Survival Curves for Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years**

Body Weights and Clinical Observations

In 6 W/kg males, body weights were lower (4% to 9%) than those of the sham controls at all time points through day 457 (Figure 11 and Table 44; however, at the end of the study, the mean body weight was similar to that of the sham controls. In 1.5 and 3 W/kg males, mean body weights were significantly higher (compared to sham controls) at several time points, but at the end of the study, though the mean body weights were higher than in sham controls, the difference was not statistically significant. Mean body weights of exposed females were similar to those of the sham controls throughout the study (Figure 11 and Table 45). There were no clinical observations in males or females related to CDMA-modulated cell phone RFR exposure.

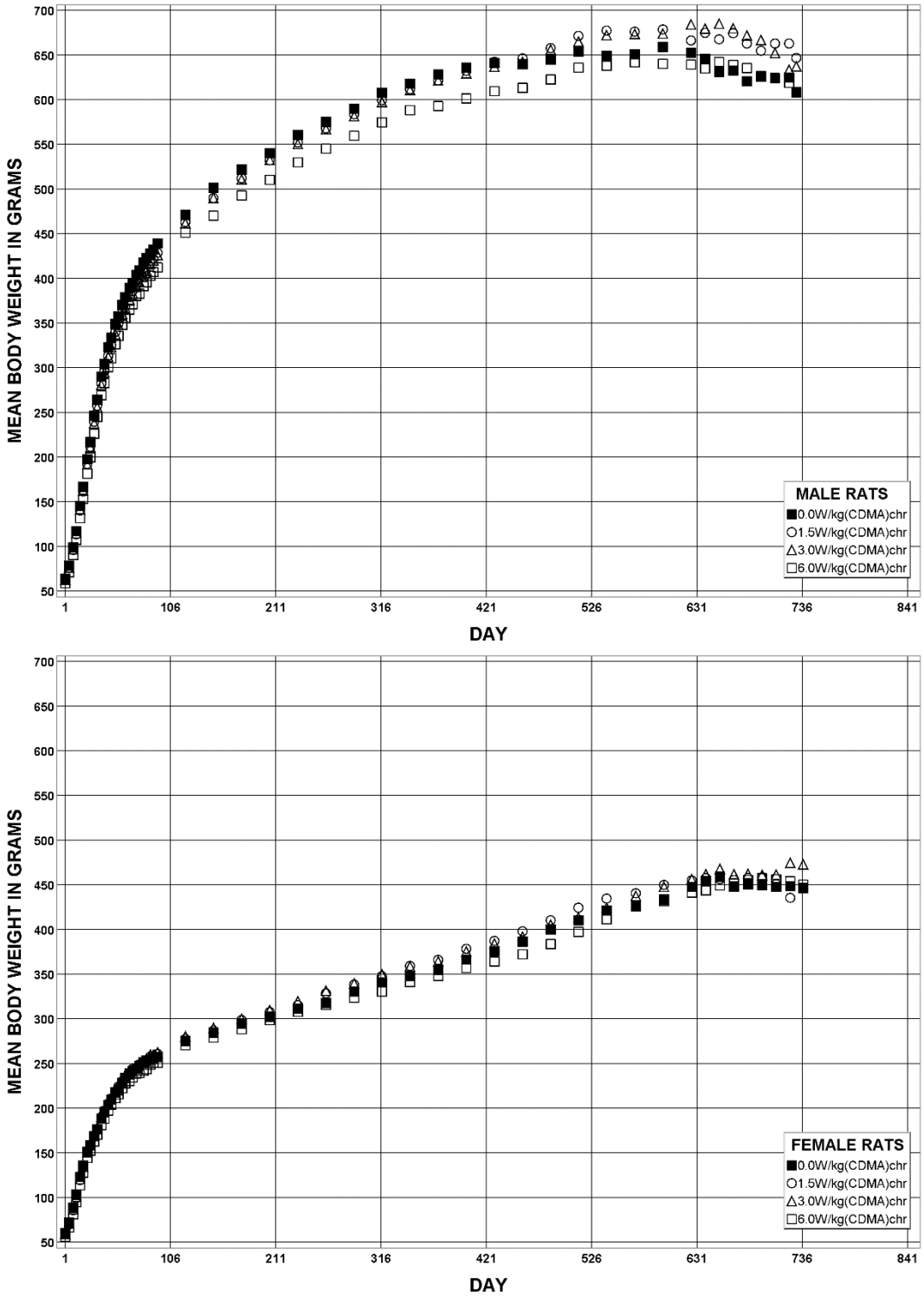

Figure 11. Growth Curves for Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

Table 44. Mean Body Weights and Survival of Male Rat Litters Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
Day	Av. Wt. (g)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters	Av. Wt. (g)	Wt. (% of Controls)	No. of Litters
1	64.3	35	62.7	97.5	35	63.0	98.1	35	59.2	92.1	35
5	78.7	35	76.0	96.6	35	76.5	97.3	35	71.4	90.8	35
9	99.0	35	96.2	97.2	35	96.8	97.7	35	90.5	91.5	35
12	117.6	35	113.7	96.7	35	114.9	97.7	35	107.7	91.6	35
16	145.5	35	140.4	96.5	35	141.5	97.3	35	132.0	90.7	35
19	167.0	35	161.7	96.8	35	162.4	97.3	35	153.5	92.0	35
23	197.8	35	192.2	97.2	35	192.0	97.1	35	181.5	91.8	35
26	217.1	35	211.6	97.4	35	211.0	97.2	35	200.1	92.2	35
30	246.5	35	239.5	97.2	35	237.8	96.5	35	226.5	91.9	35
33	264.3	35	257.4	97.4	35	255.7	96.7	35	245.2	92.8	35
37	290.4	35	282.6	97.3	35	280.9	96.7	35	269.5	92.8	35
40	304.5	35	296.4	97.3	35	294.9	96.9	35	283.2	93.0	35
44	322.9	35	313.4	97.0	35	312.6	96.8	35	301.0	93.2	35
47	333.8	35	324.6	97.2	35	323.1	96.8	35	310.9	93.1	35
51	349.0	35	340.3	97.5	35	337.7	96.8	35	326.6	93.6	35
54	357.6	35	349.4	97.7	35	347.6	97.2	35	335.7	93.9	35
58	370.2	35	361.8	97.8	35	360.2	97.3	35	348.3	94.1	35
61	379.1	35	368.0	97.1	35	367.5	96.9	35	356.0	93.9	35
65	389.6	35	379.1	97.3	35	376.3	96.6	35	365.1	93.7	35
68	395.2	35	386.3	97.7	35	383.4	97.0	35	371.3	94.0	35
72	404.0	35	392.9	97.2	35	391.9	97.0	35	380.8	94.3	35
75	409.0	35	399.7	97.7	35	396.1	96.8	35	383.3	93.7	35

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
79	417.9	35	406.8	97.3	35	404.1	96.7	35	391.5	93.7	35
82	422.7	35	411.3	97.3	35	408.6	96.7	35	395.8	93.6	35
86	427.7	35	417.1	97.5	35	416.3	97.3	35	403.0	94.2	35
89	432.4	35	422.0	97.6	35	420.4	97.2	35	407.3	94.2	35
93	439.3	35	428.6	97.6	35	426.2	97.0	35	412.5	93.9	35
121[a]	471.3	35	463.4	98.3	35	462.0	98.0	35	451.3	95.8	35
149	501.7	35	490.2	97.7	35	489.9	97.6	35	470.6	93.8	35
177	522.0	35	512.3	98.1	35	511.1	97.9	35	493.0	94.4	35
205	540.4	35	532.4	98.5	35	532.5	98.5	35	510.4	94.4	35
233	560.8	35	552.4	98.5	35	550.5	98.2	35	530.0	94.5	35
261	575.2	35	567.9	98.7	35	567.0	98.6	35	545.4	94.8	35
289	590.2	35	583.9	98.9	35	581.8	98.6	35	560.1	94.9	35
317	608.0	35	599.6	98.6	35	597.5	98.3	35	575.0	94.6	35
345	618.4	35	611.9	99.0	35	611.2	98.8	35	588.3	95.1	35
373	628.4	35	621.5	98.9	35	621.5	98.9	35	592.9	94.4	35
401	636.3	35	632.5	99.4	35	629.7	99.0	35	601.8	94.6	35
429	641.4	35	642.7	100.2	35	637.0	99.3	35	609.9	95.1	35
457	639.9	35	646.3	101.0	35	642.3	100.4	35	613.4	95.9	35
485	645.0	35	658.0	102.0	35	656.0	101.7	35	622.8	96.6	35
513	654.1	35	671.5	102.7	35	665.1	101.7	35	636.0	97.2	35
541	649.5	35	677.3	104.3	34	672.3	103.5	35	638.4	98.3	35
569	651.2	33	676.0	103.8	34	673.8	103.5	35	641.9	98.6	33
597	659.2	32	678.6	102.9	34	674.2	102.3	35	640.3	97.1	33
625	652.8	32	666.3	102.1	32	684.1	104.8	34	639.7	98.0	32
639	645.9	32	674.7	104.5	31	679.5	105.2	34	635.2	98.3	32

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
653	630.8	31	667.7	105.9	31	685.2	108.6	33	642.3	101.8	31
667	632.9	28	674.5	106.6	29	679.8	107.4	33	638.9	101.0	30
681	621.0	26	662.9	106.8	29	671.7	108.2	33	635.5	102.3	30
695	625.8	21	654.7	104.6	29	666.7	106.5	33	626.8	100.2	30
709	624.0	20	663.0	106.3	28	652.2	104.5	33	624.7	100.1	30
723	624.9	19	663.0	106.1	25	633.5	101.4	32	618.9	99.0	28
Mean for Weeks											
1–14	297.9	–	290.1	97.4	–	288.9	97.0	–	278.1	93.4	–
15–52	554.2	–	546.0	98.5	–	544.8	98.3	–	524.9	94.7	–
53–104	639.0	–	660.7	103.4	–	660.3	103.3	–	627.0	98.1	–

[a]Interim evaluation occurred during week 14.

Table 45. Mean Body Weights and Survival of Female Rat Litters Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
Day	**Av. Wt. (g)**	**No. of Litters**	**Av. Wt. (g)**	**Wt. (% of Controls)**	**No. of Litters**	**Av. Wt. (g)**	**Wt. (% of Controls)**	**No. of Litters**	**Av. Wt. (g)**	**Wt. (% of Controls)**	**No. of Litters**
1	60.4	35	59.3	98.2	35	59.6	98.7	35	55.9	92.6	35
5	72.1	35	70.2	97.4	35	71.4	99.0	35	66.6	92.5	35
9	88.7	35	85.8	96.7	35	87.1	98.2	35	81.7	92.1	35
12	103.3	35	99.6	96.5	35	101.1	97.9	35	95.2	92.1	35
16	123.2	35	119.7	97.1	35	120.7	97.9	35	113.9	92.4	35
19	136.3	35	133.7	98.1	35	134.8	98.9	35	128.1	94.0	35
23	151.5	35	150.7	99.4	35	150.8	99.5	35	144.8	95.6	35
26	158.5	35	159.3	100.5	35	160.0	100.9	35	152.8	96.4	35
30	169.1	35	168.4	99.6	35	169.9	100.5	35	162.8	96.3	35
33	176.4	35	177.2	100.4	35	177.7	100.7	35	170.6	96.7	35
37	188.7	35	190.2	100.8	35	190.2	100.8	35	181.4	96.1	35

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
40	195.8	35	197.3	100.8	35	198.0	101.1	35	188.6	96.3	35
44	203.7	35	205.3	100.8	35	206.3	101.3	35	197.6	97.0	35
47	209.9	35	210.9	100.5	35	211.8	100.9	35	204.3	97.4	35
51	218.1	35	218.4	100.1	35	221.3	101.5	35	211.5	97.0	35
54	220.1	35	224.4	102.0	35	225.9	102.6	35	215.8	98.0	35
58	228.6	35	230.4	100.8	35	232.9	101.9	35	222.6	97.4	35
61	233.9	35	234.6	100.3	35	237.4	101.5	35	228.0	97.5	35
65	239.0	35	238.7	99.9	35	238.1	99.6	35	230.5	96.5	35
68	241.7	35	244.0	100.9	35	242.3	100.2	35	234.6	97.1	35
72	244.9	35	244.9	100.0	35	246.2	100.5	35	239.2	97.7	35
75	247.6	35	249.2	100.7	35	248.9	100.5	35	239.5	96.7	35
79	251.6	35	252.4	100.3	35	251.2	99.9	35	242.1	96.2	35
82	253.5	35	253.3	99.9	35	253.3	99.9	35	243.7	96.1	35
86	254.4	35	257.8	101.4	35	260.0	102.2	35	249.0	97.9	35
89	256.2	35	258.9	101.0	35	259.6	101.3	35	251.0	97.9	35
93	257.6	35	260.8	101.3	35	262.8	102.0	35	251.2	97.5	35
121[a]	275.6	35	277.6	100.7	34	280.7	101.9	35	270.5	98.1	35
149	284.8	35	286.9	100.8	34	289.8	101.8	35	279.4	98.1	35
177	295.0	35	299.0	101.4	34	300.3	101.8	35	288.4	97.8	35
205	302.8	35	307.9	101.7	34	309.8	102.3	35	298.9	98.7	35
233	311.4	35	315.6	101.4	34	319.8	102.7	35	308.4	99.0	35
261	317.9	35	328.6	103.4	34	331.6	104.3	35	316.0	99.4	35
289	330.8	35	338.6	102.4	34	339.7	102.7	35	323.9	97.9	35
317	340.8	35	347.7	102.0	34	349.8	102.7	35	330.9	97.1	35
345	348.2	35	359.3	103.2	34	358.8	103.0	35	341.6	98.1	35
373	355.2	35	366.3	103.1	34	364.5	102.6	35	348.3	98.0	35
401	366.7	35	378.3	103.2	34	375.6	102.4	35	357.1	97.4	35

	Sham Control		1.5 W/kg			3 W/kg			6 W/kg		
429	375.9	35	387.4	103.1	34	384.5	102.3	35	364.5	97.0	35
457	386.6	35	397.9	102.9	34	392.2	101.5	35	372.5	96.4	35
485	400.1	35	410.2	102.5	34	405.5	101.3	35	384.1	96.0	35
513	410.3	35	424.4	103.4	34	413.6	100.8	34	397.6	96.9	35
541	421.4	35	434.8	103.2	34	424.4	100.7	34	411.7	97.7	35
570	426.0	35	440.6	103.4	34	437.5	102.7	33	426.8	100.2	35
598	433.7	35	450.1	103.8	34	447.8	103.2	33	432.1	99.6	35
626	447.7	34	455.1	101.6	34	456.5	102.0	33	441.3	98.6	35
640	454.9	33	457.7	100.6	34	462.0	101.6	32	444.2	97.7	35
654	459.5	33	456.4	99.3	34	467.8	101.8	32	449.6	97.8	34
668	448.5	31	455.5	101.6	33	461.9	103.0	32	452.3	100.9	34
682	450.7	31	457.7	101.5	33	462.2	102.5	31	455.6	101.1	33
696	450.0	31	458.6	101.9	33	461.2	102.5	31	457.6	101.7	33
710	447.9	30	450.7	100.6	33	461.7	103.1	31	456.6	102.0	33
724	448.8	30	435.4	97.0	32	474.9	105.8	29	454.2	101.2	32
Mean for Weeks											
1–14	192.0	–	192.4	100.2	–	193.3	100.7	–	185.3	96.5	–
15–52	311.9	–	317.9	101.9	–	320.0	102.6	–	306.4	98.2	–
53–104	422.6	–	430.4	101.8	–	432.6	102.4	–	418.0	98.9	–

[a]Interim evaluation occurred during week 14.

Fourteen-week Interim Evaluation

There were no changes to the hematology or clinical chemistry variables attributable to CDMA exposure (Table F-2).

In 6 W/kg males, there were significantly lower absolute left and right kidney weights (13% to 15%) and absolute (14%) and relative liver weights compared to the sham controls (Table G-8). In females, the absolute left and right kidney weights were significantly lower (13% to 15%) in the 6 W/kg group, and the left absolute kidney weights were significantly lower in the 1.5 and 3 W/kg groups (10% and 8%, respectively). The mean relative kidney weights were not similarly decreased, which was likely due to the decrease in mean body weight of the 6 W/kg group compared to the sham controls. These changes did not correlate with any histopathologic findings. There were no CDMA exposure-related effects on reproductive organ weights, testis spermatid concentrations, caudal epididymal sperm concentrations, or sperm motility in males (Table H-2). Due to the frequency of poor slide quality, the estrous cycle in females was not evaluated.

In males, the incidences of cardiomyopathy of the right ventricle in all exposed groups were increased compared to that in sham controls but the increases were not statistically significant, and the severities were minimal in all cases (Table 46). There were marginally increased incidences of cardiomyopathy of the heart in 3 and 6 W/kg females, but they were not statistically significant. Cardiomyopathy is a common spontaneous disease in rats that typically has no clinical manifestations. It is characterized by degeneration and necrosis of myofibers with a mild inflammatory response of macrophages and lymphocytes with occasional neutrophils. In later stages of the disease, fibrosis may be prominent.

Table 46. Incidences of Non-neoplastic Lesions of the Heart at the 14-week Interim Evaluation in Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 w/kg
Male				
Number Examined Microscopically	10	10	10	10
Cardiomyopathy (excluding right ventricle)[a]	2 (1.5)[b]	0	3 (1.0)	6 (1.0)
Ventricle Right, Cardiomyopathy	1 (1.0)	5 (1.0)	4 (1.0)	4 (1.0)
Cardiomyopathy, All Sites	3 (1.3)	5 (1.0)	6 (1.0)	6 (1.0)
Female				
Number Examined Microscopically	10	10	10	10
Cardiomyopathy (excluding right ventricle)	0	0	2 (1.0)	2 (1.0)
Ventricle Right, Cardiomyopathy	0	0	1 (1.0)	1 (1.0)
Cardiomyopathy, All Sites	0	0	3 (1.0)	3 (1.0)

[a]Number of animals with lesion.
[b]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.

Pathology and Statistical Analyses

This section describes the statistically significant or biologically noteworthy changes in the incidences of neoplasms and non-neoplastic lesions of the heart, brain, pituitary gland (pars distalis), liver, adrenal medulla, prostate gland, kidney and other organs, pancreas, mammary gland, adrenal cortex, and thymus. Summaries of the incidences of neoplasms and non-neoplastic lesions, statistical analyses of primary neoplasms that occurred with an incidence of at least 5% in at least one animal group, and historical incidences for the neoplasms mentioned in this section are presented in Appendix C for male rats and Appendix D for female rats.

Heart: Malignant schwannomas were observed in all exposed male groups (Table 47, Table C-1, and Table C-2). No schwannomas were observed in sham controls. The incidence in the 6 W/kg group was significant, as was the positive trend. The 6 W/kg incidence slightly exceeded the historical control range for all exposure routes (Table C-3). Endocardial Schwann cell hyperplasia, a putative preneoplastic Schwann cell lesion, was seen in three 6 W/kg males, resulting in a significant positive trend (P = 0.044; Table 47 and Table C-7).

In females, there were two malignant schwannomas each in the 1.5 and 6 W/kg groups (Table 47 and Table D-1). Neither of these incidences nor the positive trend were statistically significant. These incidences were also within the historical control range for all routes of exposure (Table D-3). A single incidence of endocardial Schwann cell hyperplasia was diagnosed in each of the three exposure groups, but there were none in the sham control group (Table 47 and Table D-7).

The malignant schwannomas and Schwann cell hyperplasias in males and females were morphologically similar to those seen in the rats exposed to GSM-modulated cell phone RFR.

Cardiomyopathy of the right ventricular free wall was seen in all male and female groups, including the sham controls (Table 47, Table C-7, and Table D-7). In males and females, the incidences in exposed groups were increased compared to the sham controls; the increased incidence in 6 W/kg males was statistically significant. There was also a slight elevation in the severity of this non-neoplastic lesion in 6 W/kg males, but there was no similar elevation in severity in females. Cardiomyopathy was diagnosed separately in the right ventricle because the study pathologist observed an unusual predilection for, and a slightly different morphology in the right ventricle as compared to the left ventricular free wall and the interventricular septum. An increased incidence was found in the right ventricle in the 6 W/kg males compared to the sham controls. Cardiomyopathy in the CDMA-exposed rats was morphologically identical to that described previously for the GSM-exposed rats.

Table 47. Incidences of Malignant Schwannoma and Neoplasms and Non-neoplastic Lesions of the Heart in Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Heart[a]	90	90	90	90
Cardiomyopathy[b]	79 (1.9)[c]	84 (1.9)	83 (1.8)	85 (1.3)
Ventricle Right, Cardiomyopathy	54 (1.1)	45 (1.2)	62 (1.3)	74* (1.7)
Endocardium, Hyperplasia, Schwann Cell	0	0	0	3 (2.0)
Malignant Schwannoma[d]				
Overall rate[e]	0/90 (0%)	2/90 (2%)	3/90 (3%)	6/90 (7%)
Litters rate[f]	0/35 (0%)	2/35 (6%)	3/35 (9%)	6/35 (17%)
Adjusted rate[g]	0.0%	2.7%	3.8%	8.8%
Terminal rate[h]	0/25 (0%)	2/43 (5%)	2/56 (4%)	3/43 (7%)
First incidence (days)	–j	730 (T)	642	488
Rao-Scott adjusted Poly-3 test[i]	P = 0.011	P = 0.273	P = 0.175	P = 0.030
All Organs: Malignant Schwannoma[k]				
Overall rate	3/90 (3%)	4/90 (4%)	4/90 (4%)	8/90 (9%)
Litters rate	3/35 (9%)	4/35 (11%)	4/35 (11%)	7/35 (20%)
Adjusted rate	4.5%	5.4%	5.1%	11.6%
Terminal rate	1/25 (4%)	2/43 (5%)	2/56 (4%)	4/43 (9%)
First incidence (days)	555	573	619	153
Rao-Scott adjusted Poly-3 test	P = 0.075	P = 0.551	P = 0.582	P = 0.136
Female				
Heart	90	90	90	90
Cardiomyopathy	40 (1.1)	43 (1.1)	33 (1.2)	45 (1.1)
Ventricle Right, Cardiomyopathy	4 (1.0)	7 (1.0)	9 (1.0)	9 (1.0)
Endocardium, Hyperplasia, Schwann Cell	0	1 (3.0)	1 (1.0)	1 (1.0)
Malignant Schwannoma[l]				
Overall rate	0/90 (0%)	2/90 (2%)	0/90 (0%)	2/90 (2%)
Litters rate	0/35 (0%)	2/34 (6%)	0/35 (0%)	2/35 (6%)
Adjusted rate	0.0%	2.7%	0.0%	2.5%
Terminal rate	0/48 (0%)	1/45 (2%)	0/50 (0%)	2/61 (3%)
First incidence (days)	–	649	–	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.343	P = 0.317	–[m]	P = 0.342
All Organs: Malignant Schwannoma[n]				
Overall rate	4/90 (4%)	2/90 (2%)	2/90 (2%)	4/90 (4%)
Litters rate	3/35 (9%)	2/34 (6%)	2/35 (6%)	4/35 (11%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adjusted rate	5.7%	2.7%	2.8%	5.0%
Terminal rate	2/48 (4%)	1/45 (2%)	2/50 (4%)	4/61 (7%)
First incidence (days)	489	649	737 (T)	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.561	P = 0.346N	P = 0.354N	P = 0.577N

*Significantly different (P ≤ 0.05) from the sham control group by the Rao-Scott adjusted Poly-3 test.
(T) = terminal euthanasia.
[a]Number of animals with tissue examined microscopically.
[b]Number of animals with lesion.
[c]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.
[d]Historical control incidence for other 2-year studies (all routes): 1/50, 1/50, 0/50.
[e]Number of animals with neoplasm per number of animals necropsied.
[f]Number of litters with animals with neoplasm per number of litters necropsied.
[g]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[h]Observed incidence at terminal euthanasia.
[i]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A lower incidence in an exposure group is indicated by N.
[j]Not applicable; no neoplasms in animal group.
[k]Historical control incidence for other 2-year studies (all routes): 1/50, 2/50, 0/50.
[l]Historical control incidence for other 2-year studies (all routes): 0/49, 0/50, 0/50.
[m]Value of statistic cannot be computed.
[n]Historical control incidence for other 2-year studies (all routes): 0/50, 2/50, 2/50.

Brain: In males, there three malignant gliomas in the 6 W/kg group, resulting in a significant positive trend (Table 48 and Table C-1). In females, malignant glioma occurred in three 1.5 W/kg animals; no malignant gliomas were observed in the other exposed groups or in the sham controls (Table 48 and Table D-1). There was no significant positive trend for this neoplasm in females, and the increased incidence was not significant. Glial cell hyperplasia occurred in most exposed groups of males and females, but none of the incidences were significantly increased (Table 48, Table C-7, and Table D-7). The proliferative glial cell lesions were morphologically similar to those seen in the rats exposed to GSM-modulated RFR.

Table 48. Incidences of Neoplasms and Non-neoplastic Lesions of the Brain in Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	90	90	90	90
Glial Cell, Hyperplasia[a]	0	2 (1.5)[b]	0	2 (2.5)
Glioma Malignantc				
Overall rate[d]	0/90 (0%)	0/90 (0%)	0/90 (0%)	3/90 (3%)
Litters rate[e]	0/35 (0%)	0/35 (0%)	0/35 (0%)	3/35 (9%)
Adjusted rate[f]	0.0%	0.0%	0.0%	4.5%
Terminal rate[g]	0/25 (0%)	0/43 (0%)	0/56 (0%)	3/43 (7%)
First incidence (days)	–[i]	–	–	730 (T)
Rao-Scott adjusted Poly-3 test[h]	P = 0.044	–[j]	–	P = 0.221

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Female				
Number Examined Microscopically	90	90	90	90
Glial Cell, Hyperplasia	0	0	1 (2.0)	1 (2.0)
Glioma Malignant[k]				
Overall rate	0/90 (0%)	3/90 (3%)	0/90 (0%)	0/90 (0%)
Litters rate	0/35 (0%)	3/34 (9%)	0/35 (0%)	0/35 (0%)
Adjusted rate	0.0%	4.1%	0.0%	0.0%
Terminal rate	0/48 (0%)	2/45 (4%)	0/50 (0%)	0/61 (0%)
First incidence (days)	–	550	–	–
Rao-Scott adjusted Poly-3 test	P = 0.384N	P = 0.236	–	–

(T) = terminal euthanasia.
[a]Number of animals with lesion.
[b]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.
[c]Historical control incidence for other 2-year studies (all routes): 0/50, 2/50.
[d]Number of animals with neoplasm per number of animals with brain examined microscopically.
[e]Number of litters with animals with neoplasm per number of litters with brain examined microscopically.
fPoly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[g]Observed incidence at terminal euthanasia.
[h]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend is indicated by N.
[i]Not applicable; no neoplasms in animal group.
[j]Value of statistic cannot be computed.
[k]Historical control incidence for other 2-year studies (all routes): 0/50, 1/50.

Pituitary Gland (Pars Distalis): In males, there were increased incidences of adenoma in the 1.5 and 3 W/kg groups, but only the 3 W/kg incidence was significant (Table 49, Table C-1, and Table C-2). No carcinomas occurred in males and there was no significant increase in the incidence or severity of hyperplasia (Table 49, Table C-1, and Table C-7). In females exposed to 3 W/kg, there were significantly decreased incidences of adenoma and adenoma or carcinoma (combined) (Table 49, Table D-1, and Table D-2). There were no increased incidences of carcinoma or hyperplasia in females (Table 49, Table D-1, and Table D-8). In the males, there was a significantly increased incidence of cyst in the 1.5 W/kg group (Table 49 and Table C-7). In the females, there was a significantly decreased incidence of cyst in the 6 W/kg group (Table 49 and Table D-8). Pituitary gland cysts are considered developmental anomalies and are fairly common, so the toxicologic significance of these changes is unclear.

Liver: In males, there were increased incidences of hepatocellular adenoma in the 1.5 and 3 W/kg groups compared to sham controls (Table 50 and Table C-1). There were also hepatocellular carcinomas, one each in the 3 and 6 W/kg groups. The incidences of hepatocellular adenoma or carcinoma (combined) were increased in all exposed groups, but none of the incidences were significant. In 6 W/kg females, there was a decreased incidence of hepatocellular adenoma with a significant negative trend (Table 50, Table D-1, and Table D-2). The NTP historical control incidence of hepatocellular adenoma in male Hsd:Sprague Dawley® SD® rats by all routes of exposure is 1/240, and no hepatocellular carcinomas have been seen in this strain of rat in the NTP historical control data. In females, the historical control incidence of

hepatocellular adenoma in NTP studies by all routes of exposure is 11/240, and as with males, no hepatocellular carcinomas have been seen. In males, the incidences of mixed cell focus were increased in all exposed groups, but only the incidence in the 1.5 W/kg group was significant (Table 50 and Table C-7).

Table 49. Incidences of Neoplasms and Non-neoplastic Lesions of the Pituitary Gland (Pars Distalis) in Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	89	90	90	90
Cyst[a]	5	15*	7	6
Hyperplasia	32 (2.4)[b]	32 (2.4)	34 (2.5)	27 (2.2)
Adenoma, Multiple	0	0	1	2
Adenoma (includes multiple)[c]				
Overall rate[d]	17/89 (19%)	25/90 (28%)	34/90 (38%)	13/90 (14%)
Litters rate[e]	13/35 (37%)	18/35 (51%)	24/35 (69%)	12/35 (34%)
Adjusted rate[f]	24.9%	32.7%	41.8%	19.0%
Terminal rate[g]	5/25 (20%)	16/43 (37%)	22/56 (39%)	6/43 (14%)
First incidence (days)	527	605	471	567
Rao-Scott adjusted Poly-3 test[h]	P = 0.226N	P = 0.208	P = 0.030	P = 0.273N
Female				
Number Examined Microscopically	90	89	89	90
Cyst	7▲	5	3	1*
Hyperplasia	20 (2.5)	22 (2.2)	26 (1.9)	22 (2.8)
Adenoma, Multiple	1	1	0	0
Adenoma (includes multiple)[i]				
Overall rate	43/90 (48%)	41/89 (46%)	30/89 (34%)	40/90 (44%)
Litters rate	28/35 (80%)	26/34 (76%)	21/35 (60%)	25/35 (71%)
Adjusted rate	57.1%	52.9%	39.5%	49.0%
Terminal rate	28/48 (58%)	20/45 (44%)	16/50 (32%)	31/61 (51%)
First incidence (days)	464	578	493	626
Rao-Scott adjusted Poly-3 test	P = 0.156N	P = 0.360N	P = 0.026N	P = 0.204N
Carcinoma[j]	1	1	1	0
Adenoma or Carcinoma[i]				
Overall rate	44/90 (49%)	42/89 (47%)	31/89 (35%)	40/90 (44%)
Litters rate	29/35 (83%)	26/34 (76%)	21/35 (60%)	25/35 (71%)
Adjusted rate	57.9%	54.1%	40.7%	49.0%
Terminal rate	28/48 (58%)	20/45 (44%)	16/50 (32%)	31/61 (51%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
First incidence (days)	464	578	493	626
Rao-Scott adjusted Poly-3 test	P = 0.131N	P = 0.381N	P = 0.030N	P = 0.180N

▲Significant trend (P ≤ 0.05) by the Rao-Scott adjusted Poly-3 test.
*Significantly different (P ≤ 0.05) from the sham control group by the Rao-Scott adjusted Poly-3 test.
[a]Number of animals with lesion.
[b]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.
[c]Historical control incidence for other 2-year studies (all routes): 14/50, 5/50, 11/50.
[d]Number of animals with neoplasm per number of animals with pituitary gland examined microscopically.
[e]Number of litters with animals with neoplasm per number of litters with pituitary gland examined microscopically.
[f]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[g]Observed incidence at terminal euthanasia.
[h]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
[i]Historical control incidence for other 2-year studies (all routes): 19/50, 18/50, 18/50.
[j]Historical control incidence for other 2-year studies (all routes): 0/50, 0/50, 0/50.

Table 50. Incidences of Neoplasms and Non-neoplastic Lesions of the Liver in Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Male				
Number Examined Microscopically	90	90	89	88
Mixed Cell Focus[a]	32	51*	47	37
Hepatocellular Adenoma[b]				
Overall rate[c]	0/90 (0%)	2/90 (2%)	4/89 (4%)	0/88 (0%)
Litters rate[d]	0/35 (0%)	2/35 (6%)	4/35 (11%)	0/35 (0%)
Adjusted rate[e]	0.0%	2.7%	5.1%	0.0%
Terminal rate[f]	0/25 (0%)	2/43 (5%)	4/56 (7%)	0/43 (0%)
First incidence (days)	–[h]	730 (T)	730 (T)	–
Rao-Scott adjusted Poly-3 test[g]	P = 0.556N	P = 0.310	P = 0.132	–[i]
Hepatocellular Carcinoma[j]	0	0	1	1
Hepatocellular Adenoma or Carcinoma[b]				
Overall rate	0/90 (0%)	2/90 (2%)	4/89 (4%)	1/88 (1%)
Litters rate	0/35 (0%)	2/35 (6%)	4/35 (11%)	1/35 (3%)
Adjusted rate	0.0%	2.7%	5.1%	1.5%
Terminal rate	0/25 (0%)	2/43 (5%)	4/56 (7%)	0/43 (0%)
First incidence (days)	–	730 (T)	730 (T)	594
Rao-Scott adjusted Poly-3 test	P = 0.416	P = 0.281	P = 0.113	P = 0.475
Female				
Hepatocellular Adenoma[k]				
Overall rate	7/90 (8%)	2/90 (2%)	2/90 (2%)	1/90 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Litters rate	6/35 (17%)	2/34 (6%)	2/35 (6%)	1/35 (3%)
Adjusted rate	10.1%	2.7%	2.8%	1.3%
Terminal rate	6/48 (13%)	1/45 (2%)	2/50 (4%)	1/61 (2%)
First incidence (days)	707	493	737 (T)	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.042N	P = 0.118N	P = 0.125N	P = 0.052N
Hepatocellular Carcinoma[j]	0	0	0	1

*Significantly different (P ≤ 0.05) from the sham control group by the Rao-Scott adjusted Poly-3 test.
(T) = terminal euthanasia.
[a]Number of animals with lesion.
[b]Historical control incidence for other 2-year studies (all routes): 0/50, 1/50, 0/50.
[c]Number of animals with neoplasm per number of animals with liver examined microscopically.
[d]Number of litters with animals with neoplasm per number of litters with liver examined microscopically.
[e]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[f]Observed incidence at terminal euthanasia.
[g]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
[h]Not applicable; no neoplasms in animal group.
[i]Value of statistic cannot be computed.
[j]Historical control incidence for other 2-year studies (all routes): 0/50, 0/50, 0/50.
[k]Historical control incidence for other 2-year studies (all routes): 0/50, 2/50, 2/50.

Adrenal Medulla: In females, there were increased incidences of benign pheochromocytoma and benign, malignant, or complex pheochromocytoma (combined) in all exposed groups, but only the incidence of benign, malignant, or complex pheochromocytoma (combined) at 1.5 W/kg was significant (Table 51, Table D-1, and Table D-2). There were increased incidences of malignant pheochromocytoma in the 1.5 and 3 W/kg groups compared to sham controls. There were increased incidences of hyperplasia in all exposed female groups, but the incidences were not significant (Table 51 and Table D-8).

Prostate Gland: The incidences of epithelial hyperplasia were increased in all exposed groups compared to the sham controls (Table C-7), and the severity increased slightly. Only the incidence in the 6 W/kg group was significant (sham control, 5/90; 1.5 W/kg, 11/90; 3 W/kg, 9/90; 6 W/kg, 15/85). Epithelial hyperplasia of the prostatic epithelium was characterized as infoldings or papillary projections of epithelial cells into the lumen of a prostatic gland. The cells did not fill or distend the gland, and there was no atypia and no mitotic figures.

Table 51. Incidences of Neoplasms and Non-neoplastic Lesions of the Adrenal Medulla in Female Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Number Examined Microscopically	86	89	87	88
Hyperplasia[a]	13 (1.5)[b]	20 (1.7)	20 (1.3)	18 (1.9)
Benign Pheochromocytoma[c]				
Overall rate[d]	1/86 (1%)	7/89 (8%)	3/87 (3%)	4/88 (5%)
Litters rate[e]	1/35 (3%)	7/34 (21%)	3/35 (9%)	4/35 (11%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adjusted rate[f]	1.5%	9.6%	4.4%	5.2%
Terminal rate[g]	1/45 (2%)	5/44 (11%)	3/48 (6%)	4/60 (7%)
First incidence (days)	737 (T)	464	737 (T)	737 (T)
Rao-Scott adjusted Poly-3 test[h]	P = 0.466	P = 0.059	P = 0.322	P = 0.248
Malignant Pheochromocytoma[i]	0	2	1	0
Complex Pheochromocytoma[j]	0	0	1	0
Benign, Malignant, or Complex Pheochromocytoma[k]				
Overall rate	1/86 (1%)	9/89 (10%)	5/87 (6%)	4/88 (5%)
Litters rate	1/35 (3%)	9/34 (26%)	5/35 (14%)	4/35 (11%)
Adjusted rate	1.5%	12.3%	7.2%	5.2%
Terminal rate	1/45 (2%)	7/44 (16%)	4/48 (8%)	4/60 (7%)
First incidence (days)	737 (T)	464	652	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.546	P = 0.022	P = 0.126	P = 0.242

(T) = terminal euthanasia.
[a]Number of animals with lesion.
[b]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.
[c]Historical control incidence for other 2-year studies (all routes): 3/50, 0/50, 0/49.
[d]Number of animals with neoplasm per number of animals with adrenal medulla examined microscopically.
[e]Number of litters with animals with neoplasm per number of litters with adrenal medulla examined microscopically.
[f]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[g]Observed incidence at terminal euthanasia.
[h]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation.
[i]Historical control incidence for other 2-year studies (all routes): 2/50, 0/50, 0/49.
[j]Historical control incidence for other 2-year studies (all routes): 0/50, 0/50, 0/49.
[k]Historical control incidence for other 2-year studies (all routes): 5/50, 0/50, 0/49.

Kidney and Other Organs: The severity of chronic progressive nephropathy was lower in all exposed male groups compared to the sham controls (Table 52). The chronic progressive nephropathy was morphologically similar to that seen in the rats exposed to GSM-modulated RFR. There were decreased incidences in a number of lesions in other organs in exposed male groups, some statistically significant, that were thought to be secondary to the chronic progressive nephropathy, either directly or indirectly (Table 52 and Table C-7). These lesions included hyperplasia of the parathyroid gland; mineral in the blood vessels in the colon, liver, mesentery, pancreas, salivary glands, brain, heart, kidney, skeletal muscle, glandular stomach, spleen, and aorta; mineral in the heart, salivary gland, and stomach; fibrous osteodystrophy of bone; polyarteritis nodosa (chronic active inflammation of the blood vessels) of the epididymis, testis, cecum, liver, pancreas, glandular stomach, and thymus; germ cell degeneration of the testis; edema, erosion, epithelial regeneration, acute inflammation, and ulcer of the cecum; epithelial regeneration of the colon; epithelial regeneration and acute inflammation of the rectum; red pulp atrophy and white pulp atrophy of the spleen; and exfoliated germ cell and hypospermia of the epididymis.

Other Lesions: In males, there were significantly decreased incidences of adenoma and adenoma or carcinoma (combined) of the pancreas in the 6 W/kg group, although the incidences in 1.5 and 3 W/kg groups were slightly increased compared to the sham controls (Table C-1 and Table C-2). One male in the 3 W/kg group had a carcinoma but no adenoma.

In females, there were significantly decreased incidences of adenoma (sham control, 8/90; 1.5 W/kg, 4/90; 3 W/kg, 1/90; 6 W/kg, 2/90) and adenoma or carcinoma (combined) (16/90, 12/90, 7/90, 6/90) of the mammary gland (Table D-1 and Table D-2).

In males, the incidences of adrenal cortex hypertrophy were increased in all exposed groups compared to the sham controls, and the incidence in the 3 W/kg group was significant (35/90, 42/90, 55/90, 44/89; P = 0.013) (Table C-7). In 6 W/kg males, there were significantly increased incidences of lung congestion (13/90, 13/90, 11/90, 33/90) and thymic hemorrhage (2/88, 2/85, 2/87, 20/82).

Genetic Toxicology

Twenty tissue samples obtained from animals at the 14-week interim evaluation in the 2-year study were evaluated for DNA damage using the comet assay (two sexes, two cell phone RFR modulations, five tissues). Results are based on the standard 100-cell scoring approach in use at the time these data were collected; data obtained using a 150-cell scoring approach, recommended in a recently adopted international guideline for the in vivo comet assay, are noted here for the few instances where results differed between the two methods. The complete 100-cell and 150-cell data are presented in Appendix E data tables. A significant increase in DNA damage (% comet tail DNA) was observed in hippocampus cells of male rats exposed to the CDMA modulation (Table E-1). Although the levels of DNA damage in hippocampus cells were also increased in an exposure-related fashion using the 150-cell scoring approach, the increases were not statistically significant (Table E-3). An exposure-related increase (trend test P = 0.004) in DNA damage was seen in the cells of the frontal cortex of male rats exposed to the CDMA modulation (Table E-1); however, no individual exposure groups were significantly elevated over the sham control group and the result was therefore judged to be equivocal. For male rat blood leukocytes exposed to either the CDMA or GSM modulation (Table E-1 and Table E-2), results from scoring 100 cells were negative; however, these leukocyte samples showed equivocal responses with the 150-cell method due to a significant trend test (P = 0.012) or pairwise test (P = 0.021) for CDMA- and GSM-exposed rats, respectively (Table E-3 and Table E-4). No statistically significant increases in the % comet tail DNA were observed in any of the female rat samples scored with the 100-cell approach (Table E-5 and Table E-6). The 150-cell scoring approach yielded a significant trend test (P = 0.013) in peripheral blood leukocytes of female rats exposed to the CDMA modulation, but these results were driven by data from a single animal (Table E-7).

In contrast to what was seen in the mice[197], a high degree of interanimal variability was observed in the % comet tail DNA values in rats within a treatment group, and this level of variability reduced the statistical power to detect increases in DNA migration, although the magnitudes of the increases observed in some rats suggested these were treatment-related effects. To rule out any influence from technical artifacts or protocol features, % tail DNA values and percent hedgehogs were correlated to the position of slides in the electrophoresis chambers, the interval from exposure cessation to tissue collection, and the date of slide preparation; no patterns

emerged for any of these variables and the level of DNA damage observed. The possibility that the longer interval from exposure cessation to tissue collection for the female rats may have been a factor in the absence of any detectable exposure-related increases in DNA damage cannot be ruled out due to the increased opportunity for DNA repair during this interval.

Table 52. Incidences of Non-neoplastic Lesions Associated with the Decreased Severity of Chronic Progressive Nephropathy of the Kidney in Male Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Kidney[a]	90	90	90	87
Nephropathy, Chronic Progressive[b]	88 (3.7)[c]	90 (3.3)	90 (3.0)	8 6(2.3)
Aorta	90	90	90	90
Mineral	30 (2.1)	8** (2.8)	6** (2.2)	2** (1.5)
Bone	90	90	90	90
Fibrous Osteodystrophy	46 (1.4)	20** (1.7)	15** (1.6)	5** (1.6)
Brain	90	90	90	90
Mineral	5 (1.0)	3 (1.0)	4 (1.0)	4 (1.0)
Epididymis	90	90	90	90
Artery, Inflammation, Chronic Active	2 (2.5)	3 (3.0)	3 (2.3)	3 (2.7)
Exfoliated Germ Cell	51 (1.9)	33** (1.7)	33** (1.7)	17** (1.5)
Hypospermia	28 (3.4)	24 (3.1)	13** (3.7)	13* (3.0)
Heart	90	90	90	90
Artery, Mineral	20 (2.5)	7* (2.1)	2** (2.0)	1** (2.0)
Intestine Large, Cecum	75	76	74	68
Artery, Inflammation, Chronic Active	20 (2.1)	8* (1.9)	7* (1.9)	2** (2.5)
Edema	11 (2.0)	0**	0**	0**
Epithelium, Regeneration	14 (2.4)	1** (2.0)	0**	1** (2.0)
Erosion	10 (2.5)	1* (3.0)	1* (4.0)	1* (2.0)
Inflammation, Acute	10 (2.8)	1** (2.0)	0**	1* (2.0)
Ulcer	6 (2.3)	0*	0*	0
Intestine Large, Colon	81	83	82	76
Artery, Mineral	2 (2.0)	0	0	0
Epithelium, Regeneration	5 (2.6)	0	0	0
Intestine Large, Rectum	83	81	80	76
Epithelium, Regeneration	3 (2.3)	0	0	0
Inflammation, Acute	2 (2.5)	0	0	0
Kidney	90	90	90	87

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Artery, Mineral	2 (2.0)	0	0	0
Liver	90	90	89	88
Artery, Inflammation, Chronic Active	2 (3.5)	1 (2.0)	0	0
Artery, Mineral	1 (1.0)	1 (1.0)	0	0
Mesentery	39	19	17	6
Artery, Mineral	21 (2.1)	5* (2.0)	2** (2.5)	0*
Pancreas	90	88	87	78
Artery, Inflammation, Chronic Active	48 (2.3)	28** (2.0)	23** (2.0)	5** (2.2)
Artery, Mineral	11 (1.8)	2* (2.5)	0**	0**
Parathyroid Gland	83	83	83	82
Hyperplasia	51 (2.5)	35* (2.5)	32** (2.0)	17** (1.8)
Salivary Glands	90	90	90	86
Artery, Mineral	2 (2.5)	1 (2.0)	1 (2.0)	0
Skeletal Muscle	90	90	90	90
Mineral	2 (1.0)	0	1 (1.0)	0
Spleen	90	90	90	85
Arteriole, Mineral	1 (2.0)	0	0	0
Red Pulp, Atrophy	26 (2.2)	14* (1.9)	12** (2.1)	13* (2.0)
White Pulp, Atrophy	30 (2.1)	11** (2.3)	10** (2.4)	24 (1.9)
Stomach, Glandular	86	86	85	78
Artery, Inflammation, Chronic Active	3 (2.3)	0	0	0
Mineral	31 (2.5)	9** (3.1)	6** (2.7)	1** (2.0)
Testis	90	89	90	90
Artery, Inflammation, Chronic Active	52 (2.9)	37** (2.8)	30** (2.5)	12** (3.1)
Germ Cell, Degeneration	51 (2.3)	37* (2.6)	31** (2.2)	24** (2.1)
Thymus	88	85	87	82
Artery, Inflammation, Chronic Active	6 (2.7)	3 (2.3)	2 (1.5)	1 (2.0)

*Significantly different ($P \leq 0.05$) from the sham control group by the Rao-Scott adjusted Poly-3 test.
**$P \leq 0.01$.
[a]Number of animals with tissue examined microscopically.
[b]Number of animals with lesion.
[c]Average severity grade of lesions in affected animals: 1 = minimal, 2 = mild, 3 = moderate, 4 = marked.

The possibility that the longer interval from exposure cessation to tissue collection for the female rats may have been a factor in the absence of any detectable exposure-related increases in DNA damage cannot be ruled out due to the increased opportunity for DNA repair during this interval.

Similar to what was seen in the mice, no significant effects on the frequency of micronucleated red blood cells or the % PCEs were observed in rats of either sex exposed to either modulation of cell phone RFR (Table E-9).

Discussion

The U.S. Food and Drug Administration nominated the radio frequency radiation (RFR) emissions of wireless communication devices for toxicology and carcinogenicity testing based on several factors. Current exposure guidelines are based on protection from acute injury from thermal effects, and little was known about the potential for health effects of long-term exposure. Epidemiology and toxicology studies have not definitively demonstrated an association between cell phone RFR exposure and any specific health problems in humans; however, the results of these studies are mixed and further complicated by confounding factors (including potential recall biases of the study participants that could impact the assessment of exposure). For epidemiology studies, exposures in the general population may not have occurred for a long enough period of time to accommodate the long latency period for some types of cancers in humans. Studies in laboratory animals have been complicated by limitations that researchers have faced in conducting robust studies designed to characterize the toxicity and carcinogenicity of RFR used by cell phones.

To improve on prior methods of exposing laboratory animals to RFR, the National Toxicology Program (NTP) collaborated with technical experts from the National Institute of Standards and Technology (Boulder, CO) and the Foundation for Research on Information Technologies in Society (Zurich, Switzerland) to design, construct, and validate a novel system of exposing rodents to RFR. The exposure system was designed to expose unrestrained, individually housed animals to a statistically uniform field of RFR at frequencies (900 MHz in rats and 1,900 MHz in mice) and with modulations [Global System for Mobile Communications (GSM) or Code Division Multiple Access (CDMA)] used in cellular communication devices. The exposure facility was installed at IIT Research Institute (Chicago, IL), where all animal studies were conducted following system testing and RFR exposure validation. The design and performance characteristics for the exposure system are reported fully in Capstick et al.[148], and detailed tissue-specific RFR exposure modeling at these frequencies is presented in Gong et al.[147].

Studies assessing the effects of GSM- and CDMA-modulated cell phone RFR on body temperatures of rats, including exposure in dams, were previously reported in Wyde et al.[146]. Following these initial studies, the NTP conducted 28-day and 2-year studies to characterize the potential toxicity and carcinogenicity of whole-body exposure to RFR in rats beginning in utero with exposures continuing throughout gestation and lactation and for an additional 28 days, 14 weeks, or 2 years. Due to the exposure and use pattern of cell phones by pregnant women and women of childbearing age, exposures were initiated in utero.

Hsd:Sprague Dawley® SD® rats received whole-body exposure to GSM- or CDMA-modulated cell phone RFR (900 MHz) for 9 hours and 10 minutes a day over an 18-hour exposure period starting during gestation and continuing throughout lactation and for an additional 28 days, 14 weeks, or 2 years. Exposures for the GSM- and CDMA-modulated cell phone RFR studies were conducted in parallel with a common non-exposed (sham) control group that was housed in an identical chamber that differed from the RFR exposure chambers only in the absence of RFR emission during the exposure periods.

The absorption of nonionizing RFR transfers energy to biological tissues through a process that results in some degree of heating of the exposed tissue. It has been well established that exposure

to excessive levels of RFR can cause damage to tissues through overt thermal changes for which the body is unable to compensate. However, exposure to such high levels of RFR is not reflective of the scenario of human exposure from cell phones and other wireless communication devices, which involves episodic exposure to much lower power levels. To characterize the thermal effects of RFR in male and female Hsd:Sprague Dawley® SD® rats, a series of short-term pilot studies was conducted. The goal of these studies was to further investigate the overt thermal effect of RFR, characterize the impact of body size and pregnancy status on body temperature following exposure to RFR, and to identify adequately challenging exposure levels [specific absorption rates (SARs)] of RFR below those that raised body temperature by more than 1°C[146].

In general, the studies by Wyde et al.[146] demonstrated a significant SAR-dependent increase in body temperature that was greater in larger, older rats than in smaller, young rats. Exposures to 10 W/kg RFR or greater (both modulations) for up to 5 days induced excessively high body temperatures compared to sham controls, leading to mortality in many cases and increased resorptions in pregnant rats at 12 W/kg. These data suggest that exposure at these levels resulted in the potential exceedance of the thermoregulatory capacity in rats. In these studies, body temperatures were higher with increasing SAR compared to sham controls at exposures of 6 W/kg or greater for both modulations. Male rats were more sensitive than females to RFR-induced rises in body temperature compared to sham controls. In the current 28-day studies, body temperature was higher in the F0 dams compared to sham controls during perinatal exposure at 6 and 9 W/kg. These findings were consistent with the effects observed in the thermal pilot studies in pregnant dams at similar SARs[146]. No increases in body temperature were observed in the F1 offspring at 6 or 9 W/kg.

Based on temperature changes (>1°C) in adult rats at 8 W/kg or greater in the thermal pilot studies[146], increased body temperature in F0 dams at 9 W/kg, and decreased F1 pup survival at 9 W/kg in the current 28-day studies, the highest exposure level selected for the 2-year studies was 6 W/kg. This exposure level was selected to provide an exposure considered adequate to challenge the animals without causing disruption of the thermoregulatory process. The lowest whole-body exposure level selected for the 2-year studies in rats, 1.5 W/kg, is close to the current Federal Communications Commission guidelines for localized exposure of 1.6 W/kg for cellular communication devices in the United States. The localized (1.6 W/kg) and whole-body exposure limits (0.08 W/kg) are based on protection from acute injury induced by thermal effects of RFR[198]. While core body temperature is a good general surrogate for the heating effects of RFR, it must be noted that core body temperature does not address the potential for localized heating in certain tissues at exposures that do not induce higher core temperature compared to control animals. This concept is further illustrated in the relative tissue SAR modeling studies of Gong et al.[147], which demonstrates a differing distribution of energy absorption in various tissues. These differences are based on the size, shape, nature of surrounding tissues, and the dielectric properties of the tissue. Due to logistical constraints, core body temperature measurements were not collected in the 2-year studies as they were in the 28-day studies.

Significant biological effects occurred during the perinatal exposure period in dams (F0) and their offspring (F1) that were associated with exposure to RFR in both the 28-day and 2-year studies, regardless of modulation (GSM or CDMA). In the F0 RFR-exposed dams, lower body weights and body weight gains compared to sham controls were observed during the gestation and lactation periods. In both the 28-day and 2-year studies, lower body weight gains late in gestation (gestation day 15 through 21) were observed at 9 and 6 W/kg, respectively, which may

be related to the lower pup weights on postnatal day (PND) 1. During the lactation period, maternal body weight gains were decreased and body weights in the 6 and 9 W/kg groups were lower in both the 28-day and 2-year studies compared to sham controls. In general, body weights were lower with increasing SAR.

In the F1 offspring, body weights were lower than those of the sham controls on PND 1 and throughout the lactation period. Males appeared to be slightly more susceptible to the effects of RFR on body weight. The lack of further decreases in body weight over the course of the 2-year studies suggests that the RFR-mediated effects on body weight in the F1 offspring may be specific to the perinatal period. There was no effect on live litter size throughout lactation; however, in the 28-day GSM study, there was a significantly lower survival ratio at 9 W/kg compared to sham controls early in the lactation period (PND 1 to 4) prior to culling litters to a standard size. Additionally, there was significantly greater pup mortality during the lactation period (PND 4 to 21) in the 2-year CDMA study at 6 W/kg than in sham controls, which was not observed in the 28-day CDMA study at the same exposure level.

The results from the perinatal portions of these studies indicate that RFR at these exposures could impact normal development. However, the occurrence of early pup deaths and slowed pup weight gain with RFR exposure compared to sham controls could also be secondary to effects on the dams. For example, changes in maternal behavior or capacity to properly nourish their pups may have contributed to these effects as the magnitude of the lower pup body weights appeared to increase during early lactation and then decrease as the pups aged and required less maternal care. Unfortunately, behavioral abnormalities could not be directly observed in the current studies because the design of the chambers prohibited observation during the 18-hour daily RFR exposure periods. Further research would be required to elucidate the mechanism by which RFR induces these effects in pups.

At the end of the 2-year studies, survival was significantly greater in all groups of exposed male rats in the GSM study (50% to 68%) and at 3 and 6 W/kg in the CDMA study (48% to 62%) compared to the male sham control group (28%). In the male sham control group, survival declined more rapidly after week 75 than in all exposed groups, reflecting a higher rate of moribund sacrifices. The resulting 28% survival rate in sham control males was lower than the range observed in the historical controls (40% to 60%); however, survival of the GSM- and CDMA-exposed groups (48% to 68%) was similar to the historical control range. When including control groups of rats from additional studies that have not yet been reported by the NTP, survival in the male control groups is highly variable, ranging from 24% to 72%[196]. The differences in survival in male rats may reflect the inherent variability in survival observed among the control groups in the Hsd:Sprague Dawley® SD® male rat. The duration of NTP 2-year rodent cancer studies is near the average life span of the Hsd:Sprague Dawley® SD® rat, and slight variations in the mortality rate can result in large apparent differences when only considering the 2-year time point. In female rats, survival in the sham control and exposed groups was within the historical control range (42% to 60%), except for the CDMA 6 W/kg group (68%) which exceeded the concurrent sham controls (54%) and the historical control range.

The higher mortality in the sham control males compared to the exposed males was largely attributed to a high severity of chronic progressive nephropathy in the kidneys that resulted in moribund removal of a large number of male rats from the studies. Chronic progressive

nephropathy is a common cause of death in Sprague Dawley rats as well as other rat strains, and typically occurs to some degree in many aged rats, although with greater severity in males. Of note, the severity of chronic progressive nephropathy in males decreased with increasing SAR exposure to either modulation when compared to the sham control group.

Advanced chronic progressive nephropathy can result in markedly impaired renal function and a host of secondary lesions in other organs. In males, a broad spectrum of non-neoplastic lesions considered to be secondary to chronic progressive nephropathy (including parathyroid gland hyperplasia, mineralization in multiple tissues, and fibrous osteodystrophy of bone) was seen in all groups (sham control and exposed), with the highest incidences in the sham control group and decreasing with increasing SAR exposure. Chronic progressive nephropathy can also cause an increased incidence of polyarteritis nodosa, a spontaneous vascular disease that most commonly affects medium-sized arteries in the mesentery, pancreas, kidney, pancreaticoduodenal artery, and testis, although arteries in most other organs can also be affected[199]. In the current studies, there were a number of organs with arterial inflammation consistent with polyarteritis nodosa. The incidence of these vascular lesions was greater in the sham control group than in exposed groups, correlating to the high severity of chronic progressive nephropathy in the sham control group. The apparent SAR-dependent effects in males and the slight effects (decreased incidence of nephropathy) observed in females suggest that the decrease in chronic progressive nephropathy may have been related to RFR exposure. Whether this reflects a direct effect of RFR on possible suppression of inflammatory processes through stimulation of stress response pathways[200], or is secondary to a possible reduction in feed intake leading to a reduction in the severity of nephropathy[201; 202], remains to be established. The heart, brain, and adrenal gland were target organs for RFR exposure in males. The heart and brain were affected by both GSM- and CDMA-modulated RFR, whereas the adrenal medulla was a target only for GSM-modulated RFR exposure.

In the heart of male rats, exposure to RFR for 2 years resulted in a statistically significant positive trend in the incidences of cardiac schwannoma for both GSM and CDMA modulations. The incidence at 6 W/kg for CDMA was statistically significantly increased compared to sham controls. Additionally, cardiac Schwann cell hyperplasia occurred in groups of male rats exposed to both GSM- and CDMA-modulated cell phone RFR. These hyperplastic lesions are relevant to the evaluation of neoplasms because Schwann cell hyperplasia in the heart may progress to cardiac schwannoma[203; 204]. No cardiac schwannomas or Schwann cell hyperplasia were observed in the sham control male rats. The 5.5% and 6.6% incidences of malignant schwannoma observed in the 6 W/kg GSM- and CDMA-modulated cell phone RFR groups, respectively, exceed the highest rate observed in a single historical control group (2%) of completed peer reviewed studies[l]. The absence of these lesions in sham control males in the current studies was not considered to be related to the shorter longevity in controls. The 28% survival rate of the sham control male rats in the current studies was relatively low compared to other recent NTP studies in Hsd:Sprague Dawley® SD® rats[196]. However, while most (13/19) of the malignant schwannomas were observed at terminal necropsy, some (6/19) were observed at 70 to 94 weeks, a time when survival in the sham control male rats was greater than 65%. Based on the rarity of these lesions, the presence of preneoplastic Schwann cell hyperplasia, and the

[l]The historical control rate reported in Wyde et al.[158]was 9/699 (range 0% to 6%) because control groups from NTP studies using the Hsd:Sprague Dawley® SD® rat model that had not yet been peer reviewed were included.

higher incidence of these malignant tumors in the highest exposure group with both modulations, malignant schwannomas in the heart were considered to result from exposure to RFR. This was the basis for the conclusion of clear evidence for carcinogenic activity for both GSM- and CDMA-modulated cell phone RFR in male rats.

In females, there were two occurrences of schwannoma in the heart in the groups exposed to 3 W/kg GSM-modulated RFR or 1.5 or 6 W/kg CDMA-modulated RFR. Single occurrences of Schwann cell hyperplasia were also observed in each of the CDMA exposed groups of females. With consideration of the significant increase observed for both modulations in males, this was an uncertain finding in female rats that may have been related to RFR exposures and was considered equivocal evidence of carcinogenic activity for both GSM- and CDMA-modulated cell phone RFR.

Schwannomas occurred in organs other than the heart in both male and female rats, but the incidences of these extra-cardiac schwannomas were not affected by RFR exposures. These results suggest that the proliferative effects of RFR on Schwann cells was specific to the heart. It is possible that these heart-specific effects are a result of higher SAR exposures in the heart compared to some of the other tissues based on the dielectric properties of the heart, which can affect tissue-specific RFR absorption[147]. A recent study by Falcioni et al.[205] reported low incidences of cardiac schwannoma in male rats exposed to whole-body GSM-modulated RFR at 5, 25, or 50 V/m, 1,800 MHz for 19 hours a day. The incidence of schwannoma at a constant 50 V/m (0.1 W/kg estimated SAR) was statistically significant in male rats.

In the current studies, RFR exposure also induced higher incidences and severity of cardiomyopathy of the right ventricular free wall in male rats exposed to either GSM- or CDMA-modulated cell phone RFR and in female rats exposed to GSM-modulated RFR. The effect of RFR on cardiomyopathy appears to be specific to the right ventricle in these groups because incidences of cardiomyopathy in the whole heart were unchanged in males and CDMA females and lower in the GSM females compared to sham controls. Higher incidences of cardiomyopathy in the right ventricle of male rats exposed to GSM-modulated cell phone RFR compared to sham controls were also observed at the 14-week interim evaluation. For males exposed to CDMA-modulated cell phone RFR for 14 weeks, higher incidences of cardiomyopathy were observed relative to sham controls across the whole heart, not specifically in the right ventricle. Cardiomyopathy is a common spontaneous disease in rats that typically has no clinical manifestations. The observation of this lesion at the 14-week interim evaluation in 3 and 6 W/Kg GSM males and the relatively higher modeled organ-specific SAR for heart for both males and females compared to other organs[147] further suggest that the heart is a specific target organ for RFR.

In the brain, incidences of malignant gliomas were observed in RFR-exposed male rats, whereas none were observed in any of the sham controls. Malignant gliomas are poorly differentiated neoplasms of glial cells in the central nervous system. They may arise from or differentiate into astrocytes, oligodendrocytes, microglial cells, or ependymal cells. Similar to the occurrence of cardiac schwannomas, glial cell lesions in the brain were observed only in RFR-exposed male rats and not in any of the sham controls. The findings of malignant gliomas in the brain of male rats were not as robust as those observed for cardiac schwannomas, however the differences between these responses may be related to a relatively low predicted absorption rate (SAR) in the cerebral hemisphere in relation to other tissues based on the SAR estimates modeled by Gong et

al.[147]. Malignant gliomas were observed at all SARs in the GSM study, and at 6 W/kg in the CDMA study, although not in an exposure-related manner, or with statistical significance. However, glial cell hyperplasia, a potentially preneoplastic lesion that may progress to a malignant glioma, was also observed at all SARs in the GSM study and at 1.5 and 6 W/kg in the CDMA study. No malignant gliomas or glial cell hyperplasias were observed in any of the sham control males. These data support an association between the observed incidences of malignant glioma in male rats and RFR exposure. Unlike the cardiac schwannomas, nearly all (10/11) of the malignant gliomas in males were observed at 101 weeks or later and more than half (6/10) of the glial cell hyperplasias were observed at terminal necropsy. Therefore, the occurrence in exposed males but not sham control males may in part reflect differences in survival, where sham control animals did not survive long enough to develop a tumor. However, taken together, these tumors were also considered to be related to RFR exposure.

In females, there were occurrences of malignant glioma of the brain at 1.5 and 6 W/kg in the CDMA and GSM studies, respectively, and single instances of glial cell hyperplasia at 3 W/kg in the GSM study and at 3 and 6 W/kg in the CDMA study. None were observed in any of the female sham controls. The occurrence of malignant gliomas was judged to be equivocal evidence of carcinogenic activity of CDMA-modulated RFR in female rats.

The schwannomas observed in the heart and the malignant gliomas observed in the brain arise from a similar functional cell type. Schwannomas are tumors of Schwann cell origin occurring in the peripheral nervous system (PNS). Schwann cells are classified as glial cells of the PNS. They are similar to glial cells in the brain in that they are specialized non-neuronal supportive cells whose functions include maintaining homeostasis, forming myelin, and providing support and protection for neurons of the PNS. In the central nervous system (CNS), glial cells include astrocytes, oligodendrogliocytes, microglial cells, and ependymal cells. In the PNS, Schwann cells produce myelin and are analogous to oligodendrocytes of the CNS. Generally, glial neoplasms in the rat are aggressive, poorly differentiated, and usually classified as malignant.

Granular cell tumors, which arise within the meninges from a different type of cell than the schwannomas and malignant gliomas, were also observed in the brain in all GSM-exposed male groups. The combined incidences of granular cell tumors (3% to 4%) were higher than the concurrent sham control (1%) but not the historical control range (0% to 4%[196]), and they were not statistically different from the sham control incidences.

The granular cell tumors were not considered to be biologically related to the malignant gliomas because these lesions arise from different tissues. Consequently, the carcinogenic activity of RFR with respect to the granular cell tumors was considered independently from the malignant gliomas. While the higher incidences in the exposed groups suggest an association with exposure, the occurrence of this lesion in a sham control animal combined with the low magnitude of the increase in exposed rats and the lack of statistical significance reduces the confidence that this effect is attributable to the GSM cell phone RFR exposure. Therefore, the higher incidences of granular cell tumors of the meninges in the brain of males exposed to GSM-modulated RFR may or may not have been related to RFR exposure.

In the adrenal medulla, there were higher combined incidences of benign, malignant, or complex pheochromocytoma in GSM-exposed males and CDMA-exposed females. In GSM-exposed males, there were higher incidences of benign, malignant, or complex pheochromocytoma

(combined) in the 1.5 and 3 W/kg groups compared to sham controls. The incidence in the 1.5 W/kg group (27%) exceeded the historical control incidence (20.1%), while the incidence in the 3 W/kg group (31%) exceeded both the historical control incidence and range (16% to 28%). The incidence at 6 W/kg was marginally higher than in sham controls, but it did not exceed the historical control range. The higher incidences of benign, malignant, or complex pheochromocytoma (combined) that were observed in GSM-exposed males were considered to be related to RFR exposure. In CDMA-exposed females, there were higher incidences of benign pheochromocytoma and benign, malignant, or complex pheochromocytoma (combined) in the 1.5 W/kg group compared to sham controls. The higher incidence (8% vs. 1% in sham controls) of benign pheochromocytoma occurred only at the lowest exposure and was outside the historical control range (0% to 6%). No effect was observed in the incidences of hyperplasia in any of the exposed female groups. The isolated increased incidences of these neoplasms in the low-exposure female group reduces the confidence that this effect is attributable to the RFR exposure. Therefore, the higher incidence of benign, malignant, or complex pheochromocytoma (combined) in females was judged to be equivocal evidence of carcinogenic activity of CDMA-modulated RFR.

The increased incidences of pheochromocytoma in the adrenal medulla in the 2-year studies may suggest a stress response; however, not enough endpoints were evaluated in the current studies to elucidate the role of stress, if any, in these findings. Other parameters that would provide a clearer insight into whether these effects were related to a stress response, such as measurements of feed consumption, activity, or serum stress hormones, were not evaluated in the current studies. Further studies of RFR should include the evaluation of stress as a potential mechanism for these effects.

Incidences of hepatocellular adenoma or carcinoma (combined) in the liver were observed in all groups of male rats exposed to CDMA-modulated RFR; none were observed in the sham control group. Hepatocellular adenomas and carcinomas appear to be rare in the Hsd:Sprague Dawley® SD® rat (1/150 and 0/150, respectively, in other NTP studies), and the combined incidence at 3 W/kg (4%) in CDMA RFR-exposed male rats exceeded the highest incidence seen in single studies in the historical controls (2%). However, the increased incidence in only the 3 W/kg group reduces the confidence that this is attributable to the RFR exposure, so this increase may or may not have been related to RFR exposure.

The incidences of adenoma or carcinoma (combined) in pancreatic islets were higher in all groups of male rats exposed to GSM-modulated cell phone RFR compared to the sham controls; however, only the incidence at 1.5 W/kg was significantly increased compared to sham controls. The incidences in all groups of GSM-exposed rats (19% to 30%) exceeded the range in the historical controls (4% to 16%). The lack of an exposure-response gradient reduced the confidence that this was attributable to RFR exposure. Therefore, the higher incidences of neoplasms of the pancreatic islets may or may not have been related to RFR exposure.

The incidences of adenoma of the pituitary gland (pars distalis) were increased in most groups of male rats exposed to either GSM- or CDMA-modulated cell phone RFR, compared to the sham controls. In the GSM study, the incidences in all groups of exposed male rats exceeded the incidence in the historical controls. However, the incidences of adenoma were similar between all exposed groups regardless of SAR. In the CDMA study, there was a significantly higher incidence of adenoma in the 3 W/kg male group compared to sham controls with a similar

(nonsignificant) response at 1.5 W/kg. The incidence in the 6 W/kg group was lower than in sham controls. There were no differences in the incidences of hyperplasia in any of the GSM- or CDMA-exposed male groups when compared to sham controls. Based on these findings, the higher incidences of pituitary gland (pars distalis) adenoma in RFR-exposed males may or may not have been related to exposure to RFR.

In GSM-exposed males, there were higher incidences of prostate gland adenoma (7%) and adenoma or carcinoma (combined) (8%) in the 3 W/kg group versus the sham control (2%). Although these incidences were not statistically significant compared to sham controls, these neoplasms have not been observed in historical controls (0/150). These neoplasms are considered rare in Hsd:Sprague Dawley® SD® rats. Preneoplastic lesions of epithelial hyperplasia were higher in the exposed groups, but the incidences were not statistically higher compared to sham controls. The higher incidence of prostate gland adenoma or carcinoma (combined) in the 3 W/kg group may have been related to GSM-modulated cell phone RFR exposure, although the lack of an exposure response reduces the confidence that the increased incidence is attributable to exposure, therefore, this was considered an equivocal finding. No effect in prostate gland neoplasms was observed in the CDMA study; however, prostate gland epithelial hyperplasia was higher compared to sham controls in all CDMA groups, with a significant increase in the 6 W/kg group versus sham controls and a statistically significant positive trend across exposure groups.

Subsets of male and female rats from the 2-year studies were examined at 14-week interim evaluations for biomarkers of genotoxicity. Chromosomal damage was evaluated using the peripheral blood erythrocyte micronucleus (MN) assay, and DNA damage was evaluated in the frontal cortex, hippocampus, cerebellum, liver, and peripheral blood using the comet assay. Results of the MN assays were negative, but higher levels of DNA damage were observed in some tissues of male rats (hippocampus and frontal cortex in the CDMA modulation). In general, results of the comet assay suggested that CDMA induced more effects than GSM, and male rats showed greater sensitivity than female rats. However, the difference between the response in males and females may have been related to the longer interval between cessation of exposure and tissue sampling that occurred for the females, potentially allowing for an increased amount of DNA repair to take place.

There were several instances in which one or two animals within a group of RFR-exposed rats showed high levels of DNA damage compared to the rest of the animals within the exposure group, while levels of DNA damage in sham control animals tended to be more tightly clustered. Interanimal variation in response to RFR might be due to the genetic heterogeneity of these Hsd:Sprague Dawley® SD® rats, which are maintained as an outbred stock. However, tissues from a single rat rarely tracked together with regard to the extent of DNA damage (i.e., there was never a case in which all of the tissues from one rat had the highest mean percent comet tail DNA). This observation suggests intertissue variability in response, as has also been seen with chemicals[195]. Although the markedly higher levels of DNA damage observed in some rats were suggestive of an exposure-related effect, the high degree of interanimal variation within an exposure group resulted in nonsignificant statistical tests in most instances (for example, male rat cerebellum exposed to CDMA and female rat peripheral blood exposed to CDMA).

Unlike ionizing radiation or ultraviolet radiation, RFR is not sufficiently energetic, by several orders of magnitude, to directly damage macromolecules[4], and little is known about the mechanisms by which RFR could induce DNA damage in the absence of thermal effects.

Proposed mechanisms include, for example, induction of oxygen radicals and interference with DNA repair mechanisms[54; 206].

No histopathologic assessments of cytotoxicity (apoptosis and necrosis) were conducted in the brain or liver tissues that were examined for DNA damage, which leaves open the possibility that apoptosis or necrosis may have confounded the comet assay results. However, this seems unlikely as brain sections from other groups of rats at the 14-week interim evaluations and in the 2-year studies did undergo histopathologic assessment and no significant findings were reported. Unlike ionizing radiation or ultraviolet radiation, RFR is not sufficiently energetic, by several orders of magnitude, to directly damage macromolecules[4], and little is known about the mechanism by which RFR could induce DNA damage in the absence of a thermal effect. Proposed mechanisms include, for example, induction of oxygen radicals and interference with DNA repair mechanisms[54; 206].

The primary effects of RFR in rats included perinatal effects on body weights and body weight gains in the F0 dams and the F1 offspring, higher body temperatures in the F0 dams, and higher incidences of neoplasms of the heart (malignant schwannoma), brain (malignant glioma), and adrenal gland (pheochromocytoma) in males. There were also several histopathologic lesions that occurred in single or lower exposure groups, lacked a clear exposure response, or occurred at incidences that did not differ from those observed in control groups in past NTP studies. With reduced confidence that these increased incidences were attributable to the RFR exposure, these uncertain findings may or may not have been related to RFR exposure. The greater survival in RFR-exposed males compared to sham controls was attributed to decreased chronic progressive nephropathy, which may be a result of RFR exposure. Many of these findings occurred with both modulations suggesting the modulations had minimal, if any impact on the responses to the 900 MHz cell phone RFR exposure.

With a few exceptions, male rats seemed more sensitive to effects of RFR than females. In pilot studies of effects of RFR on body temperature in young and aged rats, temperatures were generally higher in aged males than females at SARs of 10 W/kg and above with both GSM and CDMA modulation[146]. The higher incidences of right ventricular cardiomyopathy compared to sham controls observed at 14 weeks and 2 years also occurred to a greater extent in males than in females. Heart schwannomas were related to exposure in RFR-exposed males, but the incidences in females may or may not have been related to exposure. More instances of marginally higher incidences in lesions compared to sham controls in other tissues that may have been related to RFR exposure were observed in males [brain (granular cell tumors), pituitary gland, prostate gland, and pancreas] than in females (brain and adrenal gland). Finally, reduced chronic progressive nephropathy in RFR-exposed male rats compared to sham controls in the 2-year studies was the likely basis for the higher survival rate of RFR-exposed males (in particular in the GSM exposure groups). Reasons for these sex differences remain to be explored.

Some epidemiology studies have suggested an association between cell phone radio frequency radiation exposure and potential health effects[95; 96; 124; 131; 207-210]. Based on available studies, a working group of the International Agency for Research on Cancer[93] classified radiofrequency electromagnetic fields as possibly carcinogenic to humans. Of particular concern was the possible association (limited evidence) with brain glioma and acoustic neuroma (vestibular schwannoma) in the region of the head that is most exposed to RFR when a wireless phone is used at the ear. The malignant schwannomas of the heart observed in male rats in the current

studies and the malignant gliomas observed in the brain of male rats, arise from the same cell type as the acoustic neuromas (vestibular schwannomas) observed in humans, though in a different location. This lends credence to the possible association of these tumors with cellular phone use. The cellular origin of malignant gliomas in the rat brain is unclear, but they do arise from glial cells (support cells in the brain), as do human glioblastomas, so it is possible that such an association exists for these tumors as well. However, the interpretation of these findings with respect to specific risks to humans from cellular telephone use is beyond the scope of the current studies. Further efforts to characterize the molecular basis by which RFR elicits its effects in rats, and a more complete assessment of the exposure conditions in the current studies in relation to exposures to humans from cellular telephone technologies should provide context to aid understanding of the implications of the current findings to human health.

Conclusions

GSM-modulated RFR

Under the conditions of this 2-year whole-body exposure study, there was clear evidence of carcinogenic activity[m] of GSM-modulated cell phone RFR at 900 MHz in male Hsd:Sprague Dawley® SD® rats based on the incidences of malignant schwannoma of the heart. The incidences of malignant glioma of the brain and benign, malignant, or complex pheochromocytoma (combined) of the adrenal medulla were also related to RFR exposure. The incidences of benign or malignant granular cell tumors of the brain, adenoma or carcinoma (combined) of the prostate gland, adenoma of the pars distalis of the pituitary gland, and pancreatic islet cell adenoma or carcinoma (combined) may have been related to RFR exposure. There was equivocal evidence of carcinogenic activity of GSM-modulated cell phone RFR at 900 MHz in female Hsd:Sprague Dawley® SD® rats based on the incidences of schwannomas of the heart.

Increases in non-neoplastic lesions of the heart, brain, and prostate gland in male rats, and of the heart, thyroid gland, and adrenal gland in female rats occurred with exposures to GSM-modulated RFR at 900 MHz.

CDMA-modulated RFR

Under the conditions of this 2-year whole-body exposure study, there was clear evidence of carcinogenic activity of CDMA-modulated cell phone RFR at 900 MHz in male Hsd:Sprague Dawley® SD® rats based on the incidences of malignant schwannoma of the heart. The incidences of malignant glioma of the brain were also related to RFR exposure. The incidences of adenoma of the pars distalis of the pituitary gland and adenoma or carcinoma (combined) of the liver may have been related to RFR exposure. There was equivocal evidence of carcinogenic activity of CDMA-modulated cell phone RFR at 900 MHz in female Hsd:Sprague Dawley® SD® rats based on the incidences of malignant schwannoma of the heart, malignant glioma of the brain, and benign, malignant, or complex pheochromocytoma (combined) of the adrenal medulla. Increases in non-neoplastic lesions of the heart, brain, and prostate gland in male rats, and of the brain in female rats occurred with exposures to CDMA-modulated RFR at 900 MHz.

[m]See Explanation of Levels of Evidence of Carcinogenic Activity. See summary of the peer review panel comments and the public discussion on this Technical Report appears in Appendix L.

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Appendix A. Summary of Lesions in Male Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

Tables

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Table A-1. Summary of the Incidence of Neoplasms in Male Rats Exposed to GSM-modulated Cell Phone RFR for Two Years[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
Fourteen-week interim evaluation	15	15	15	15
Early deaths				
Accidental deaths	1	–	–	1
Moribund	44	24	19	13
Natural deaths	20	21	21	16
Survivors				
Terminal euthanasia	25	45	50	60
Animals examined microscopically	100	100	100	100
Systems Examined at 14 Weeks with No Neoplasms Observed				
Alimentary System				
Cardiovascular System				
Endocrine System				
General Body System				
Genital System				
Hematopoietic System				
Integumentary System				
Musculoskeletal System				
Nervous System				
Respiratory System				
Special Senses System				
Urinary System				
Two-year Study				
Alimentary System				
Esophagus	(90)	(89)	(90)	(90)
Intestine large, cecum	(75)	(75)	(79)	(80)
Intestine large, colon	(81)	(83)	(81)	(82)
Serosa, pheochromocytoma malignant, metastatic, adrenal medulla	–	–	1 (1%)	–
Intestine large, rectum	(83)	(81)	(85)	(87)
Intestine small, duodenum	(81)	(82)	(79)	(79)
Adenocarcinoma	–	1 (1%)	–	1 (1%)
Intestine small, ileum	(78)	(76)	(78)	(76)
Intestine small, jejunum	(73)	(76)	(70)	(76)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adenocarcinoma	2 (3%)	1 (1%)	1 (1%)	1 (1%)
Liver	(90)	(90)	(90)	(90)
Cholangioma	–	1 (1%)	–	–
Hepatocellular adenoma	–	1 (1%)	–	2 (2%)
Hepatocellular carcinoma	–	–	1 (1%)	–
Pheochromocytoma malignant, metastatic, adrenal medulla	–	–	1 (1%)	–
Serosa, carcinoma, metastatic, intestine small, jejunum	–	–	–	1 (1%)
Mesentery	(39)	(19)	(17)	(7)
Pheochromocytoma malignant, metastatic, adrenal medulla	–	–	1 (6%)	–
Oral mucosa	(0)	(2)	(0)	(2)
Squamous cell carcinoma	–	1 (50%)	–	1 (50%)
Pancreas	(90)	(89)	(88)	(86)
Adenoma	13 (14%)	17 (19%)	14 (16%)	12 (14%)
Adenoma, multiple	5 (6%)	4 (4%)	4 (5%)	4 (5%)
Carcinoma	1 (1%)	1 (1%)	2 (2%)	–
Carcinoma, metastatic, intestine small, jejunum	–	–	–	1 (1%)
Pheochromocytoma malignant, metastatic, adrenal medulla	–	–	1 (1%)	–
Salivary glands	(90)	(90)	(90)	(90)
Schwannoma malignant	–	1 (1%)	–	–
Sublingual gland, schwannoma malignant, metastatic, uncertain primary site	1 (1%)	–	–	–
Stomach, forestomach	(90)	(90)	(90)	(90)
Sarcoma	–	–	–	1 (1%)
Squamous cell carcinoma	–	–	–	1 (1%)
Squamous cell papilloma	1 (1%)	–	–	–
Stomach, glandular	(86)	(88)	(87)	(86)
Tooth	(0)	(1)	(1)	(0)
Odontoma	–	–	1 (100%)	–
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Chemodectoma malignant	–	1 (1%)	–	–
Blood vessel	(1)	(2)	(1)	(0)
Heart	(90)	(90)	(90)	(90)
Carcinoma, metastatic, adrenal cortex	–	–	–	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Chemodectoma malignant	–	1 (1%)	–	–
Endocardium, schwannoma malignant	–	1 (1%)	1 (1%)	2 (2%)
Myocardium, schwannoma malignant	–	1 (1%)	–	3 (3%)
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(88)
Adenoma	1 (1%)	4 (4%)	2 (2%)	1 (1%)
Carcinoma	–	1 (1%)	–	1 (1%)
Adrenal medulla	(88)	(90)	(89)	(87)
Pheochromocytoma benign	8 (9%)	21 (23%)	19 (21%)	14 (16%)
Pheochromocytoma benign, multiple	1 (1%)	–	2 (2%)	–
Pheochromocytoma, complex	1 (1%)	–	–	–
Pheochromocytoma, malignant	1 (1%)	1 (1%)	4 (4%)	–
Bilateral, pheochromocytoma benign	1 (1%)	2 (2%)	4 (4%)	–
Bilateral, pheochromocytoma malignant	–	1 (1%)	–	–
Islets, pancreatic	(90)	(89)	(86)	(85)
Adenoma	5 (6%)	12 (13%)	9 (10%)	10 (12%)
Adenoma, multiple	–	2 (2%)	1 (1%)	1 (1%)
Carcinoma	8 (9%)	13 (15%)	10 (12%)	5 (6%)
Carcinoma, multiple	–	2 (2%)	–	–
Parathyroid gland	(83)	(87)	(87)	(81)
Adenoma	1 (1%)	1 (1%)	–	1 (1%)
Pituitary gland	(89)	(90)	(90)	(90)
Sarcoma, metastatic, eye	–	–	1 (1%)	–
Schwannoma malignant, metastatic, Harderian gland	–	–	–	1 (1%)
Schwannoma malignant, metastatic, trigeminal ganglion	–	–	1 (1%)	–
Pars distalis, adenoma	17 (19%)	28 (31%)	25 (28%)	26 (29%)
Pars distalis, adenoma, multiple	–	–	1 (1%)	–
Thyroid gland	(89)	(89)	(89)	(87)
Chemodectoma malignant, metastatic, tissue NOS	–	1 (1%)	–	
Bilateral, C-cell, adenoma	–	–	1 (1%)	1 (1%)
C-cell, adenoma	8 (9%)	9 (10%)	7 (8%)	7 (8%)
C-cell, carcinoma	2 (2%)	2 (2%)	2 (2%)	3 (3%)
Follicular cell, adenoma	–	1 (1%)	1 (1%)	–
Follicular cell, adenoma, multiple	–	1 (1%)	–	–
Follicular cell, carcinoma	–	–	–	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
General Body System				
Tissue NOS	(3)	(4)	(4)	(5)
Chemodectoma malignant	–	1 (25%)	–	–
Abdominal, fat, hemangiosarcoma	–	1 (25%)	–	–
Abdominal, fat, pheochromocytoma malignant, metastatic, adrenal medulla	–	–	1 (25%)	–
Mediastinum, chemodectoma benign	–	–	–	1 (20%)
Mediastinum, schwannoma malignant	1 (33%)	–	–	–
Genital System				
Bulbourethral gland	(1)	(0)	(0)	(0)
Coagulating gland	(0)	(0)	(0)	(1)
Ductus deferens	(1)	(0)	(0)	(0)
Epididymis	(90)	(90)	(90)	(90)
Penis	(0)	(0)	(0)	(1)
Preputial gland	(88)	(90)	(90)	(90)
Squamous cell carcinoma	–	–	–	1 (1%)
Prostate	(90)	(90)	(90)	(90)
Adenoma	2 (2%)	2 (2%)	6 (7%)	3 (3%)
Carcinoma	–	–	1 (1%)	–
Seminal vesicle	(90)	(89)	(89)	(90)
Testis	(90)	(90)	(90)	(90)
Interstitial cell, adenoma	2 (2%)	2 (2%)	3 (3%)	2 (2%)
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Hemangioma	–	–	–	1 (1%)
Lymph node	(25)	(22)	(18)	(12)
Mediastinal, pheochromocytoma malignant, metastatic, adrenal medulla	–	–	1 (6%)	–
Pancreatic, pheochromocytoma malignant, metastatic, adrenal medulla	–	–	1 (6%)	–
Lymph node, mandibular	(89)	(90)	(89)	(90)
Schwannoma malignant, metastatic, salivary glands	–	1 (1%)	–	–
Lymph node, mesenteric	(90)	(89)	(86)	(89)
Carcinoma, metastatic, intestine small, jejunum	–	–	–	1 (1%)
Hemangiosarcoma	–	–	–	1 (1%)
Spleen	(90)	(90)	(89)	(90)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Hemangiosarcoma	3 (3%)	–	–	1 (1%)
Capsule, carcinoma, metastatic, intestine small, jejunum	–	–	–	1 (1%)
Thymus	(88)	(86)	(88)	(86)
Alveolar/bronchiolar carcinoma, metastatic, lung	–	1 (1%)	–	–
Thymoma benign	–	2 (2%)	1 (1%)	–
Thymoma malignant	–	–	–	1 (1%)
Integumentary System				
Mammary gland	(82)	(76)	(82)	(82)
Adenocarcinoma	–	–	1 (1%)	–
Fibroadenoma	2 (2%)	3 (4%)	4 (5%)	2 (2%)
Skin	(90)	(90)	(90)	(90)
Basal cell adenoma	1 (1%)	1 (1%)	–	1 (1%)
Basal cell carcinoma	–	–	1 (1%)	–
Keratoacanthoma	5 (6%)	3 (3%)	9 (10%)	2 (2%)
Neural crest tumor	–	–	1 (1%)	–
Sarcoma	–	–	1 (1%)	–
Squamous cell papilloma	2 (2%)	–	–	–
Sebaceous gland, adenoma	1 (1%)	–	–	–
Subcutaneous tissue, fibroma	2 (2%)	5 (6%)	7 (8%)	5 (6%)
Subcutaneous tissue, fibrosarcoma	1 (1%)	3 (3%)	1 (1%)	2 (2%)
Subcutaneous tissue, hemangiopericytoma	–	–	1 (1%)	–
Subcutaneous tissue, hibernoma	–	–	–	1 (1%)
Subcutaneous tissue, lipoma	2 (2%)	3 (3%)	1 (1%)	2 (2%)
Subcutaneous tissue, malignant fibrous histiocytoma	–	–	1 (1%)	–
Subcutaneous tissue, myxosarcoma	–	–	1 (1%)	–
Subcutaneous tissue, sarcoma	2 (2%)	1 (1%)	–	1 (1%)
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Skeletal muscle	(90)	(90)	(90)	(90)
Diaphragm, carcinoma, metastatic, intestine small, jejunum	–	–	–	1 (1%)
Nervous System				
Brain	(90)	(90)	(90)	(90)
Glioma malignant	–	3 (3%)	3 (3%)	2 (2%)
Sarcoma, metastatic, eye	–	–	1 (1%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Schwannoma malignant, metastatic, trigeminal ganglion	–	–	1 (1%)	–
Meninges, granular cell tumor benign	1 (1%)	3 (3%)	3 (3%)	3 (3%)
Meninges, granular cell tumor malignant	–	–	1 (1%)	–
Nerve trigeminal	(84)	(88)	(87)	(88)
Schwannoma malignant, metastatic, trigeminal ganglion	–	–	1 (1%)	–
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Peripheral nerve, tibial	(88)	(89)	(90)	(88)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Trigeminal ganglion	(75)	(73)	(77)	(77)
Sarcoma, metastatic, eye	–	–	1 (1%)	–
Schwannoma malignant	–	–	1 (1%)	–
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Alveolar/bronchiolar adenoma	1 (1%)	1 (1%)	–	–
Alveolar/bronchiolar carcinoma	–	1 (1%)	–	–
Carcinoma, metastatic, uncertain primary site	–	1 (1%)	–	–
Sarcoma, metastatic, kidney	–	–	–	1 (1%)
Schwannoma malignant, metastatic, salivary glands	–	1 (1%)	–	–
Nose	(89)	(90)	(90)	(89)
Sarcoma, metastatic, eye	–	–	1 (1%)	–
Schwannoma malignant, metastatic, trigeminal ganglion	–	–	1 (1%)	–
Schwannoma malignant, metastatic, uncertain primary site	1 (1%)	–	–	–
Trachea	(90)	(88)	(87)	(86)
Chemodectoma malignant, metastatic, tissue NOS	–	1 (1%)	–	–
Special Senses System				
Eye	(85)	(86)	(87)	(83)
Choroid, schwannoma malignant	–	–	1 (1%)	–
Retrobulbar, sarcoma	–	–	1 (1%)	–
Retrobulbar, schwannoma malignant, metastatic, trigeminal ganglion	–	–	1 (1%)	–
Retrobulbar, schwannoma malignant, metastatic, uncertain primary site	2 (2%)	–	1 (1%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Harderian gland	(90)	(90)	(90)	(90)
Schwannoma malignant	–	–	1 (1%)	2 (2%)
Schwannoma malignant, metastatic, trigeminal ganglion	–	–	1 (1%)	–
Schwannoma malignant, metastatic, uncertain primary site	2 (2%)	–	1 (1%)	–
Lacrimal gland	(2)	(1)	(2)	(2)
Urinary System				
Kidney	(90)	(90)	(90)	(90)
Hemangioma	–	1 (1%)	–	–
Lipoma	1 (1%)	1 (1%)	–	–
Oncocytoma benign	1 (1%)	–	1 (1%)	–
Sarcoma	–	–	1 (1%)	1 (1%)
Bilateral, renal tubule, adenoma	–	–	–	1 (1%)
Bilateral, renal tubule, adenoma, multiple	1 (1%)	–	–	–
Bilateral, renal tubule, carcinoma	1 (1%)	–	–	1 (1%)
Renal tubule, adenoma	1 (1%)	1 (1%)	3 (3%)	1 (1%)
Renal tubule, adenoma, multiple	1 (1%)	–	–	–
Ureter	(0)	(1)	(0)	(0)
Urethra	(0)	(0)	(1)	(0)
Transitional epithelium, papilloma	–	–	1 (100%)	–
Urinary bladder	(89)	(89)	(86)	(85)
Carcinoma, metastatic, prostate	–	–	1 (1%)	–
Systemic Lesions				
Multiple organs[b]	(90)	(90)	(90)	(90)
Leukemia mononuclear	–	–	–	1 (1%)
Lymphoma malignant	2 (2%)	4 (4%)	4 (4%)	2 (2%)
Mesothelioma malignant	2 (2%)	1 (1%)	6 (7%)	1 (1%)
Neoplasm Summary				
Total animals with primary neoplasms[c]				
Two-year study	56	73	78	71
Total primary neoplasms				
Two-year study	114	176	179	141
Total animals with benign neoplasms				
Two-year study	49	68	71	57
Total benign neoplasms				
Two-year study	87	132	131	104

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Total animals with malignant neoplasms				
Two-year study	24[d]	36	38	34
Total malignant neoplasms				
Two-year study	27	44	47	37
Total animals with metastatic neoplasms				
Two-year study	2	4	5	3
Total metastatic neoplasms				
Two-year study	6	6	20	8
Total animals with malignant neoplasms, uncertain primary site				
Two-year study	2	1	1	–
Total animals with uncertain neoplasms-benign or malignant				
Two-year study	–	–	1	–
Total uncertain neoplasms				
Two-year study	–	–	1	–

[a]Number of animals examined microscopically at the site and the number of animals with neoplasm.
[b]Number of animals with any tissue examined microscopically.
[c]Primary neoplasms: all neoplasms except metastatic neoplasms.
[d]Includes one animal that had a neoplasm of unknown primary origin.

Table A-2. Statistical Analysis of Primary Neoplasms in Male Rats Exposed to GSM-modulated Cell Phone RFR for Two Years[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adrenal Medulla: Benign Pheochromocytoma				
Overall rate[a]	10/88 (11%)	23/90 (26%)	25/89 (28%)	14/87 (16%)
Rate per litters[b]	8/35 (23%)	19/35 (54%)	21/35 (60%)	12/35 (34%)
Adjusted rate[c]	15.2%	29.9%	31.7%	18.3%
Terminal rate[d]	3/23 (13%)	13/45 (29%)	14/49 (29%)	11/59 (19%)
First incidence (days)	510	599	526	631
Rao-Scott adjusted Poly-3 test[e]	P = 0.472N	P = 0.030	P = 0.017	P = 0.384
Litter C-A/Fisher's test[f]	P = 0.365	P = 0.007	P = 0.002	P = 0.214
Adrenal Medulla: Benign, Complex, or Malignant Pheochromocytoma				
Overall rate	11/88 (13%)	24/90 (27%)	28/89 (31%)	14/87 (16%)
Rate per litters	9/35 (26%)	19/35 (54%)	23/35 (66%)	12/35 (34%)
Adjusted rate	16.7%	31.1%	35.3%	18.3%
Terminal rate	3/23 (13%)	13/45 (29%)	15/49 (31%)	11/59 (19%)
First incidence (days)	510	599	526	631
Rao-Scott adjusted Poly-3 test	P = 0.409N	P = 0.035	P = 0.010	P = 0.472

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Litter C-A/Fisher's test	P = 0.420	P = 0.014	P < 0.001	P = 0.301
Heart: Malignant Schwannoma				
Overall rate	0/90 (0%)	2/90 (2%)	1/90 (1%)	5/90 (6%)
Rate per litters	0/35 (0%)	2/35 (6%)	1/35 (3%)	5/35 (14%)
Adjusted rate	0%	2.7%	1.3%	6.4%
Terminal rate	0/25 (0%)	2/45 (4%)	1/50 (2%)	3/60 (5%)
First incidence (days)	–[g]	730 (T)	730 (T)	582
Rao-Scott adjusted Poly-3 test	P = 0.041	P = 0.297	P = 0.540	P = 0.080
Litter C-A/Fisher's test	P = 0.013	P = 0.246	P = 0.500	P = 0.027
Mammary Gland: Fibroma, Fibroadenoma, Adenoma, or Carcinoma				
Overall rate	2/90 (2%)	3/90 (3%)	5/90 (6%)	2/90 (2%)
Rate per litters	2/35 (6%)	3/35 (9%)	5/35 (14%)	2/35 (6%)
Adjusted rate	3.0%	4.0%	6.3%	2.6%
Terminal rate	1/25 (4%)	0/45 (0%)	3/50 (6%)	1/60 (2%)
First incidence (days)	440	320	454	544
Rao-Scott adjusted Poly-3 test	P = 0.504N	P = 0.536	P = 0.289	P = 0.604N
Litter C-A/Fisher's test	P = 0.581	P = 0.500	P = 0.214	P = 0.693
Pancreas: Adenoma				
Overall rate	18/90 (20%)	21/89 (24%)	18/88 (20%)	16/86 (19%)
Rate per litters	16/35 (46%)	17/35 (49%)	13/34 (38%)	13/35 (37%)
Adjusted rate	26.8%	27.6%	23.5%	21.3%
Terminal rate	9/25 (36%)	13/45 (29%)	15/50 (30%)	14/60 (23%)
First incidence (days)	580	614	716	674
Rao-Scott adjusted Poly-3 test	P = 0.216N	P = 0.523	P = 0.396N	P = 0.292N
Litter C-A/Fisher's test	P = 0.204N	P = 0.500	P = 0.350N	P = 0.314N
Pancreas: Adenoma or Carcinoma				
Overall rate	18/90 (20%)	22/89 (25%)	19/88 (22%)	16/86 (19%)
Rate per litters	16/35 (46%)	18/35 (51%)	14/34 (41%)	13/35 (37%)
Adjusted rate	26.8%	28.9%	24.7%	21.3%
Terminal rate	9/25 (36%)	13/45 (29%)	15/50 (30%)	14/60 (23%)
First incidence (days)	580	614	677	674
Rao-Scott adjusted Poly-3 test	P = 0.201N	P = 0.459	P = 0.457N	P = 0.288N
Litter C-A/Fisher's test	P = 0.189N	P = 0.406	P = 0.446N	P = 0.314N
Pancreatic Islets: Adenoma				
Overall rate	5/90 (6%)	14/89 (16%)	10/86 (12%)	11/85 (13%)
Rate per litters	5/35 (14%)	12/35 (34%)	9/34 (26%)	11/35 (31%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adjusted rate	7.6%	18.5%	13.2%	14.8%
Terminal rate	2/25 (8%)	10/45 (22%)	9/50 (18%)	11/60 (18%)
First incidence (days)	624	531	677	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.282	P = 0.051	P = 0.204	P = 0.140
Litter C-A/Fisher's test	P = 0.141	P = 0.046	P = 0.169	P = 0.077
Pancreatic Islets: Carcinoma				
Overall rate	8/90 (9%)	15/89 (17%)	10/86 (12%)	5/85 (6%)
Rate per litters	8/35 (23%)	12/35 (34%)	10/34 (29%)	4/35 (11%)
Adjusted rate	12.0%	19.7%	13.1%	6.7%
Terminal rate	3/25 (12%)	7/45 (16%)	8/50 (16%)	4/60 (7%)
First incidence (days)	663	531	537	544
Rao-Scott adjusted Poly-3 test	P = 0.088N	P = 0.173	P = 0.517	P = 0.220N
Litter C-A/Fisher's test	P = 0.083N	P = 0.214	P = 0.365	P = 0.171N
Pancreatic Islets: Adenoma or Carcinoma				
Overall rate	13/90 (14%)	27/89 (30%)	19/86 (22%)	16/85 (19%)
Rate per litters	12/35 (34%)	19/35 (54%)	17/34 (50%)	14/35 (40%)
Adjusted rate	19.4%	35.2%	24.8%	21.3%
Terminal rate	5/25 (20%)	16/45 (36%)	16/50 (32%)	15/60 (25%)
First incidence (days)	624	531	537	544
Rao-Scott adjusted Poly-3 test	P = 0.344N	P = 0.032	P = 0.282	P = 0.462
Litter C-A/Fisher's test	P = 0.518	P = 0.074	P = 0.140	P = 0.402
Pituitary Gland (Pars Distalis): Adenoma				
Overall rate	17/89 (19%)	28/90 (31%)	26/90 (29%)	26/90 (29%)
Rate per litters	13/35 (37%)	23/35 (66%)	19/35 (54%)	22/35 (63%)
Adjusted rate	24.9%	35.2%	32.2%	32.4%
Terminal rate	5/25 (20%)	15/45 (33%)	17/50 (34%)	19/60 (32%)
First incidence (days)	527	309	537	384
Rao-Scott adjusted Poly-3 test	P = 0.301	P = 0.126	P = 0.216	P = 0.210
Litter C-A/Fisher's test	P = 0.064	P = 0.015	P = 0.115	P = 0.028
Prostate Gland: Adenoma				
Overall rate	2/90 (2%)	2/90 (2%)	6/90 (7%)	3/90 (3%)
Rate per litters	2/35 (6%)	2/35 (6%)	5/35 (14%)	3/35 (9%)
Adjusted rate	3.0%	2.7%	7.7%	3.9%
Terminal rate	1/25 (4%)	0/45 (0%)	6/50 (12%)	3/60 (5%)
First incidence (days)	642	591	730 (T)	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.419	P = 0.625N	P = 0.224	P = 0.566

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Litter C-A/Fisher's test	P = 0.342	P = 0.693	P = 0.214	P = 0.500
Prostate Gland: Adenoma or Carcinoma				
Overall rate	2/90 (2%)	2/90 (2%)	7/90 (8%)	3/90 (3%)
Rate per litters	2/35 (6%)	2/35 (6%)	6/35 (17%)	3/35 (9%)
Adjusted rate	3.0%	2.7%	9.0%	3.9%
Terminal rate	1/25 (4%)	0/45 (0%)	6/50 (12%)	3/60 (5%)
First incidence (days)	642	591	717	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.412	P = 0.626N	P = 0.161	P = 0.566
Litter C-A/Fisher's test	P = 0.329	P = 0.693	P = 0.130	P = 0.500
Skin: Keratoacanthoma				
Overall rate	5/90 (6%)	3/90 (3%)	9/90 (10%)	2/90 (2%)
Rate per litters	5/35 (14%)	3/35 (9%)	7/35 (20%)	2/35 (6%)
Adjusted rate	7.4%	4.0%	11.6%	2.6%
Terminal rate	0/25 (0%)	2/45 (4%)	9/50 (18%)	2/60 (3%)
First incidence (days)	552	694	730 (T)	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.271N	P = 0.332N	P = 0.322	P = 0.208N
Litter C-A/Fisher's test	P = 0.256N	P = 0.355N	P = 0.376	P = 0.214N
Skin: Squamous Cell Papilloma or Keratoacanthoma				
Overall rate	7/90 (8%)	3/90 (3%)	9/90 (10%)	2/90 (2%)
Rate per litters	7/35 (20%)	3/35 (9%)	7/35 (20%)	2/35 (6%)
Adjusted rate	10.4%	4.0%	11.6%	2.6%
Terminal rate	1/25 (4%)	2/45 (4%)	9/50 (18%)	2/60 (3%)
First incidence (days)	552	694	730 (T)	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.145N	P = 0.164N	P = 0.520	P = 0.088N
Litter C-A/Fisher's test	P = 0.113N	P = 0.153N	P = 0.617	P = 0.075N
Skin: Squamous Cell Papilloma, Keratoacanthoma, Basal Cell Adenoma, or Basal Cell Carcinoma				
Overall rate	8/90 (9%)	4/90 (4%)	10/90 (11%)	3/90 (3%)
Rate per litters	8/35 (23%)	4/35 (11%)	8/35 (23%)	3/35 (9%)
Adjusted rate	11.9%	5.3%	12.8%	3.9%
Terminal rate	2/25 (8%)	3/45 (7%)	10/50 (20%)	2/60 (3%)
First incidence (days)	552	694	730 (T)	694
Rao-Scott adjusted Poly-3 test	P = 0.145N	P = 0.163N	P = 0.526	P = 0.090N
Litter C-A/Fisher's test	P = 0.132N	P = 0.171N	P = 0.612	P = 0.094N
Skin (Subcutaneous Tissue): Fibroma				
Overall rate	2/90 (2%)	5/90 (6%)	7/90 (8%)	5/90 (6%)
Rate per litters	2/35 (6%)	5/35 (14%)	7/35 (20%)	5/35 (14%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adjusted rate	3.1%	6.7%	8.9%	6.4%
Terminal rate	2/25 (8%)	3/45 (7%)	4/50 (8%)	2/60 (3%)
First incidence (days)	730 (T)	673	501	443
Rao-Scott adjusted Poly-3 test	P = 0.306	P = 0.256	P = 0.129	P = 0.277
Litter C-A/Fisher's test	P = 0.214	P = 0.214	P = 0.075	P = 0.214
Skin (Subcutaneous Tissue): Fibroma, Fibrosarcoma, Sarcoma, Myxosarcoma, or Malignant Fibrous Histiocytoma				
Overall rate	5/90 (6%)	9/90 (10%)	10/90 (11%)	8/90 (9%)
Rate per litters	5/35 (14%)	9/35 (26%)	10/35 (29%)	8/35 (23%)
Adjusted rate	7.5%	11.7%	12.6%	10.0%
Terminal rate	2/25 (8%)	4/45 (9%)	5/50 (10%)	2/60 (3%)
First incidence (days)	567	291	501	443
Rao-Scott adjusted Poly-3 test	P = 0.428	P = 0.268	P = 0.218	P = 0.383
Litter C-A/Fisher's test	P = 0.293	P = 0.185	P = 0.122	P = 0.270
Skin (Subcutaneous Tissue): Fibroma, Fibrosarcoma, Sarcoma, Myxosarcoma, Malignant Fibrous Histiocytoma, or Hemangiopericytoma				
Overall rate	5/90 (6%)	9/90 (10%)	11/90 (12%)	8/90 (9%)
Rate per litters	5/35 (14%)	9/35 (26%)	11/35 (31%)	8/35 (23%)
Adjusted rate	7.5%	11.7%	13.8%	10.0%
Terminal rate	2/25 (8%)	4/45 (9%)	6/50 (12%)	2/60 (3%)
First incidence (days)	567	291	501	443
Rao-Scott adjusted Poly-3 test	P = 0.421	P = 0.268	P = 0.159	P = 0.383
Litter C-A/Fisher's test	P = 0.284	P = 0.185	P = 0.077	P = 0.270
Thyroid Gland (C-cell): Adenoma				
Overall rate	8/89 (9%)	9/89 (10%)	8/89 (9%)	8/87 (9%)
Rate per litters	7/35 (20%)	8/35 (23%)	8/34 (24%)	8/35 (23%)
Adjusted rate	12.1%	12.0%	10.3%	10.6%
Terminal rate	6/25 (24%)	8/45 (18%)	6/50 (12%)	8/60 (13%)
First incidence (days)	498	576	636	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.416N	P = 0.581N	P = 0.460N	P = 0.481N
Litter C-A/Fisher's test	P = 0.456	P = 0.500	P = 0.474	P = 0.500
Thyroid Gland (C-cell): Adenoma or Carcinoma				
Overall rate	10/89 (11%)	11/89 (12%)	10/89 (11%)	11/87 (13%)
Rate per litters	8/35 (23%)	8/35 (23%)	9/34 (26%)	11/35 (31%)
Adjusted rate	14.9%	14.6%	12.9%	14.5%
Terminal rate	6/25 (24%)	8/45 (18%)	8/50 (16%)	11/60 (18%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
First incidence (days)	498	576	636	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.512N	P = 0.566N	P = 0.457N	P = 0.561N
Litter C-A/Fisher's test	P = 0.214	P = 0.612	P = 0.472	P = 0.296
All Organs: Malignant Schwannoma				
Overall rate	3/90 (3%)	3/90 (3%)	5/90 (6%)	7/90 (8%)
Rate per litters	3/35 (9%)	3/35 (9%)	5/35 (14%)	7/35 (20%)
Adjusted rate	4.5%	4.0%	6.4%	8.9%
Terminal rate	1/25 (4%)	2/45 (4%)	3/50 (6%)	4/60 (7%)
First incidence (days)	555	720	661	582
Rao-Scott adjusted Poly-3 test	P = 0.133	P = 0.577N	P = 0.435	P = 0.238
Litter C-A/Fisher's test	P = 0.073	P = 0.663	P = 0.355	P = 0.153
All Organs: Malignant Mesothelioma				
Overall rate	2/90 (2%)	1/90 (1%)	6/90 (7%)	1/90 (1%)
Rate per litters	2/35 (6%)	1/35 (3%)	6/35 (17%)	1/35 (3%)
Adjusted rate	3.0%	1.3%	7.6%	1.3%
Terminal rate	1/25 (4%)	0/45 (0%)	1/50 (2%)	1/60 (2%)
First incidence (days)	645	705	550	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.485N	P = 0.454N	P = 0.228	P = 0.446N
Litter C-A/Fisher's test	P = 0.544N	P = 0.500N	P = 0.130	P = 0.500N
All Organs: Benign Neoplasms				
Overall rate	49/90 (54%)	68/90 (76%)	71/90 (79%)	57/90 (63%)
Rate per litters	26/35 (74%)	33/35 (94%)	35/35 (100%)	32/35 (91%)
Adjusted rate	65.3%	80.3%	83.5%	69.2%
Terminal rate	18/25 (72%)	35/45 (78%)	44/50 (88%)	43/60 (72%)
First incidence (days)	440	309	454	384
Rao-Scott adjusted Poly-3 test	P = 0.519	P = 0.032	P = 0.010	P = 0.370
Litter C-A/Fisher's test	P = 0.035	P = 0.023	P < 0.001	P = 0.055
All Organs: Malignant Neoplasms				
Overall rate	24/90 (27%)	36/90 (40%)	38/90 (42%)	35/90 (38%)
Rate per litters	18/35 (51%)	24/35 (69%)	25/35 (71%)	26/35 (74%)
Adjusted rate	33.2%	44.9%	45.8%	41.3%
Terminal rate	5/25 (20%)	15/45 (33%)	17/50 (34%)	23/60 (38%)
First incidence (days)	212	291	537	472
Rao-Scott adjusted Poly-3 test	P = 0.276	P = 0.100	P = 0.081	P = 0.197
Litter C-A/Fisher's test	P = 0.042	P = 0.111	P = 0.070	P = 0.041
All Organs: Benign or Malignant Neoplasms				

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Overall rate	57/90 (63%)	73/90 (81%)	78/90 (87%)	71/90 (79%)
Rate per litters	29/35 (83%)	33/35 (94%)	35/35 (100%)	35/35 (100%)
Adjusted rate	72.8%	84.5%	89.3%	83.0%
Terminal rate	20/25 (80%)	36/45 (80%)	44/50 (88%)	50/60 (83%)
First incidence (days)	212	291	454	384
Rao-Scott adjusted Poly-3 test	P = 0.108	P = 0.061	P = 0.009	P = 0.096
Litter C-A/Fisher's test	P = 0.003	P = 0.130	P = 0.012	P = 0.012

(T) = terminal euthanasia.
[a]Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for adrenal gland, heart, pancreas, pancreatic islets, pituitary gland, prostate gland, and thyroid gland; for other tissues, denominator is number of animals necropsied.
[b]Number of litters with tumor-bearing animals/number of litters examined at site.
[c]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[d]Observed incidence at terminal euthanasia.
[e]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
[f]The Litter Cochran-Armitage and Fishers exact tests directly compare the litter incidence rates.
[g]Not applicable; no neoplasms in animal group.

Table A-3. Historical Incidence of Malignant Schwannoma in Control Male Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Heart: Malignant Schwannoma	All Organs: Malignant Schwannoma
Historical Incidence: All Studies		
p-Chloro-α,α,α-trifluorotoluene (January 2011)	0/50	1/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	1/50	2/50
Indole-3-carbinol (March 2007)	1/50	0/50
Radiofrequency radiation (September 2012)	0/90	3/90
Overall Historical Incidence		
Total (%)	2/240 (0.8%)	6/240 (2.5%)
Mean ± standard deviation	0.6% ± 1.1%	2.3% ± 1.8%
Range	0%–2%	0%–4%

[a]Data as of November 2017.

Table A-4. Historical Incidence of Brain Neoplasms in Control Male Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Malignant Glioma	Benign Granular Cell Tumor	Malignant Granular Cell Tumor	Benign or Malignant Granular Cell Tumor
Historical Incidence: All Studies				
p-Chloro-α,α,α-trifluorotoluene (January 2011)	2/50	0/50	0/50	0/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	0/50	2/50	0/50	2/50
Radiofrequency radiation (September 2012)	0/90	1/90	0/90	1/90
Overall Historical Incidence:				
Total (%)	2/190 (1.1%)	3/190 (1.6%)	0/190	3/190 (1.6%)
Mean ± standard deviation	1.3% ± 2.3%	1.7% ± 2.1%	–	1.7% ± 2.1%
Range	0%–4%	0%–4%	–	0%–4%

[a]Data as of November 2017; due to differences in sectioning method, indole-3-carbinol was excluded from evaluation of brain lesions.

Table A-5. Historical Incidence of Adrenal Medulla Neoplasms in Control Male Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Benign Pheochromocytoma	Malignant Pheochromocytoma	Complex Pheochromocytoma	Benign, Malignant, or Complex Pheochromocytoma
Historical Incidence: All Studies				
p-Chloro-α,α,α-trifluorotoluene (January 2011)	12/50	2/50	0/50	14/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	9/50	3/50	0/50	12/50
Indole-3-carbinol (March 2007)	5/50	2/50	1/50	8/50
Radiofrequency radiation (September 2012)	10/88	1/88	1/88	11/88
Overall Historical Incidence				
Total (%)	36/238 (15.1%)	8/238 (3.4%)	2/238 (0.8%)	45/238 (18.9%)
Mean ± standard deviation	15.8% ± 6.5%	3.8% ± 2.0%	0.8% ± 1.0%	20.1% ± 7.1%
Range	10%–24%	1%–6%	0%–2%	13%–28%

[a]Data as of November 2017.

Table A-6. Historical Incidence of Prostate Gland Neoplasms in Control Male Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Adenoma	Carcinoma	Adenoma or Carcinoma
Historical Incidence: All Studies			
p-Chloro-α,α,α-trifluorotoluene (January 2011)	0/50	0/50	0/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	0/50	0/50	0/50
Indole-3-carbinol (March 2007)	0/50	0/50	0/50
Radiofrequency radiation (September 2012)	2/90	0/90	2/90
Overall Historical Incidence			
Total (%)	2/240 (0.8%)	0/240	2/240 (0.8%)
Mean ± standard deviation	0.6% ± 1.1%	–	0.6% ± 1.1%
Range	0%–2%	–	0%–2%

[a]Data as of November 2017.

Table A-7. Historical Incidence of Pituitary Gland (Pars Distalis) Adenoma in Control Male Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Incidence in Controls
Historical Incidence: All Studies	
p-Chloro-α,α,α-trifluorotoluene (January 2011)	11/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	14/50
Indole-3-carbinol (March 2007)	5/50
Radiofrequency radiation (September 2012)	17/89
Overall Historical Incidence	
Total (%)	47/239 (19.7%)
Mean ± standard deviation	19.8% ± 7.5%
Range	10%–28%

[a]Data as of November 2017.

Table A-8. Historical Incidence of Pancreatic Islet Neoplasms in Control Male Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Adenoma	Carcinoma	Adenoma or Carcinoma
Historical Incidence: All Studies			
p-Chloro-α,α,α-trifluorotoluene (January 2011)	8/50	0/50	8/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	3/50	0/50	3/50

Study (Study Start)	Adenoma	Carcinoma	Adenoma or Carcinoma
Indole-3-carbinol (March 2007)	2/50	0/50	2/50
Radiofrequency radiation (September 2012)	5/90	8/90	13/90
Overall Historical Incidence			
Total (%)	18/240 (7.5%)	8/240 (3.3%)	26/240 (10.8%)
Mean ± standard deviation	7.9% ± 5.5%	2.2% ± 4.4%	10.1% ± 6.0%
Range	4%–16%	0%–9%	4%–16%

[a]Data as of November 2017.

Table A-9. Summary of the Incidence of Non-neoplastic Lesions in Male Rats Exposed to GSM-modulated Cell Phone RFR for Two Years[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
Fourteen-week interim evaluation	15	15	15	15
Early deaths				
Accidental deaths	1	–	–	1
Moribund	44	24	19	13
Natural deaths	20	21	21	16
Survivors				
Terminal euthanasia	25	45	50	60
Animals examined microscopically	100	100	100	100
Fourteen-week Interim Evaluation				
Alimentary System				
Esophagus	(10)	(10)	(10)	(10)
Intestine large, cecum	(10)	(10)	(10)	(10)
Intestine large, colon	(10)	(10)	(10)	(10)
Intestine large, rectum	(10)	(10)	(10)	(10)
Lymphoid tissue, hyperplasia	1 (10%)	1 (10%)	–	–
Intestine small, duodenum	(10)	(10)	(10)	(10)
Intestine small, ileum	(10)	(10)	(10)	(10)
Intestine small, jejunum	(10)	(10)	(10)	(10)
Liver	(10)	(10)	(10)	(10)
Infiltration cellular, mixed cell	1 (10%)	–	3 (30%)	3 (30%)
Pancreas	(10)	(10)	(10)	(10)
Salivary glands	(10)	(10)	(10)	(10)
Stomach, forestomach	(10)	(10)	(10)	(10)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Stomach, glandular	(10)	(10)	(10)	(10)
Cardiovascular System				
Aorta	(10)	(10)	(10)	(10)
Heart	(10)	(10)	(10)	(10)
Cardiomyopathy	2 (20%)	4 (40%)	3 (30%)	4 (40%)
Ventricle right, cardiomyopathy	1 (10%)	1 (10%)	5 (50%)	5 (50%)
Endocrine System				
Adrenal cortex	(10)	(10)	(10)	(10)
Adrenal medulla	(10)	(10)	(10)	(10)
Islets, pancreatic	(10)	(10)	(10)	(10)
Parathyroid gland	(9)	(10)	(10)	(8)
Pituitary gland	(10)	(10)	(10)	(10)
Pars distalis, cyst	–	1 (10%)	2 (20%)	2 (20%)
Rathke's cleft, cyst	–	1 (10%)	3 (30%)	3 (30%)
Thyroid gland	(10)	(10)	(10)	(10)
Ectopic thymus	–	1 (10%)	1 (10%)	2 (20%)
Genital System				
Epididymis	(10)	(10)	(10)	(10)
Preputial gland	(10)	(10)	(10)	(10)
Inflammation, chronic active	7 (70%)	3 (30%)	1 (10%)	4 (40%)
Prostate	(10)	(10)	(10)	(10)
Inflammation, chronic active	–	1 (10%)	1 (10%)	–
Seminal vesicle	(10)	(10)	(10)	(10)
Testis	(10)	(10)	(10)	(10)
Germ cell, degeneration	–	–	–	1 (10%)
Hematopoietic System				
Bone marrow	(10)	(10)	(10)	(10)
Lymph node, mandibular	(10)	(10)	(10)	(10)
Hemorrhage	2 (20%)	2 (20%)	2 (20%)	1 (10%)
Hyperplasia, lymphocyte	–	–	–	4 (40%)
Proliferation, plasma cell	–	–	–	2 (20%)
Lymph node, mesenteric	(10)	(10)	(10)	(10)
Hemorrhage	–	2 (20%)	–	–
Spleen	(10)	(10)	(10)	(10)
Thymus	(10)	(10)	(10)	(10)
Hemorrhage	5 (50%)	1 (10%)	2 (20%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Musculoskeletal System				
Bone	(10)	(10)	(10)	(10)
Tibia, fracture, chronic	–	–	–	1 (10%)
Skeletal muscle	(10)	(10)	(10)	(10)
Respiratory System				
Lung	(10)	(10)	(10)	(10)
Congestion	–	–	1 (10%)	–
Inflammation, chronic active	1 (10%)	1 (10%)	–	–
Nose	(10)	(10)	(10)	(10)
Trachea	(10)	(10)	(10)	(10)
Special Senses System				
Eye	(10)	(10)	(10)	(10)
Retina, developmental malformation	–	–	–	1 (10%)
Harderian gland	(10)	(10)	(10)	(10)
Inflammation, chronic active	–	3 (30%)	2 (20%)	2 (20%)
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Congestion	–	–	1 (10%)	–
Nephropathy, chronic progressive	9 (90%)	8 (80%)	8 (80%)	7 (70%)
Pelvis, dilation	–	–	1 (10%)	–
Renal tubule, dilation	–	1 (10%)	–	–
Urinary bladder	(10)	(10)	(10)	(10)
Systems Examined with No Lesions Observed				
General Body System				
Integumentary System				
Nervous System				
Two-year Study				
Alimentary System				
Esophagus	(90)	(89)	(90)	(90)
Dilation	2 (2%)	–	–	–
Hyperplasia	1 (1%)	–	–	–
Arteriole, inflammation, chronic active	–	1 (1%)	–	–
Artery, inflammation, chronic active	–	–	1 (1%)	–
Intestine large, cecum	(75)	(75)	(79)	(80)
Edema	11 (15%)	1 (1%)	–	4 (5%)
Erosion	10 (13%)		–	3 (4%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Inflammation, acute	10 (13%)	1 (1%)	–	2 (3%)
Inflammation, chronic active	1 (1%)	–	–	–
Ulcer	6 (8%)	–	–	–
Artery, inflammation, chronic active	20 (27%)	9 (12%)	5 (6%)	6 (8%)
Artery, mineral	1 (1%)	–	–	–
Epithelium, erosion	–	–	–	1 (1%)
Epithelium, regeneration	14 (19%)	–	–	2 (3%)
Intestine large, colon	(81)	(83)	(81)	(82)
Edema	–	1 (1%)		1 (1%)
Erosion	1 (1%)	–	–	1 (1%)
Inflammation, acute	1 (1%)	–	–	–
Ulcer	1 (1%)	–	–	–
Artery, inflammation, chronic active	12 (15%)	5 (6%)	5 (6%)	5 (6%)
Artery, mineral	2 (2%)	–	–	–
Epithelium, regeneration	5 (6%)	–	–	2 (2%)
Intestine large, rectum	(83)	(81)	(85)	(87)
Cyst	–	–	1 (1%)	–
Edema	1 (1%)	–	–	1 (1%)
Erosion	1 (1%)	–	–	–
Hyperplasia, lymphocyte	1 (1%)	1 (1%)	–	–
Inflammation, acute	2 (2%)	–	–	–
Artery, inflammation, chronic active	4 (5%)	7 (9%)	4 (5%)	2 (2%)
Epithelium, regeneration	3 (4%)	–	–	–
Intestine small, duodenum	(81)	(82)	(79)	(79)
Dilation	–	–	1 (1%)	–
Erosion	1 (1%)	–	–	–
Ulcer	1 (1%)	–	–	–
Intestine small, ileum	(78)	(76)	(78)	(76)
Artery, inflammation, chronic active	2 (3%)	1 (1%)		
Epithelium, regeneration	1 (1%)	–	–	–
Intestine small, jejunum	(73)	(76)	(70)	(76)
Dilation	–	–	1 (1%)	–
Liver	(90)	(90)	(90)	(90)
Angiectasis	1 (1%)	–	1 (1%)	–
Basophilic focus	1 (1%)	1 (1%)	–	–
Clear cell focus	8 (9%)	7 (8%)	22 (24%)	16 (18%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Eosinophilic focus	12 (13%)	5 (6%)	2 (2%)	8 (9%)
Extramedullary hematopoiesis	5 (6%)	4 (4%)	1 (1%)	4 (4%)
Hepatodiaphragmatic nodule	1 (1%)	–	2 (2%)	2 (2%)
Infiltration cellular, mixed cell	3 (3%)	2 (2%)	–	5 (6%)
Mixed cell focus	32 (36%)	45 (50%)	50 (56%)	58 (64%)
Artery, inflammation, chronic active	2 (2%)	5 (6%)	1 (1%)	–
Artery, mineral	1 (1%)	–	–	–
Artery, thrombus	–	1 (1%)	–	–
Bile duct, cyst	3 (3%)	3 (3%)	2 (2%)	–
Bile duct, fibrosis	–	–	–	1 (1%)
Bile duct, hyperplasia	41 (46%)	35 (39%)	37 (41%)	33 (37%)
Centrilobular, hepatocyte, hypertrophy		1 (1%)	–	–
Hepatocyte, degeneration	1 (1%)	–	–	–
Hepatocyte, degeneration, cystic	–	–	1 (1%)	–
Hepatocyte, necrosis	5 (6%)	6 (7%)	8 (9%)	1 (1%)
Hepatocyte, vacuolation, cytoplasmic	6 (7%)	4 (4%)	9 (10%)	3 (3%)
Kupffer cell, pigment	1 (1%)	1 (1%)		1 (1%)
Periductal, cholangiofibrosis	2 (2%)	1 (1%)	1 (1%)	1 (1%)
Mesentery	(39)	(19)	(17)	(7)
Hemorrhage	1 (3%)	–	–	–
Inflammation, chronic	2 (5%)	–	–	–
Inflammation, chronic active	–	1 (5%)	–	–
Necrosis	2 (5%)	3 (16%)	1 (6%)	–
Neovascularization	1 (3%)	1 (5%)	–	–
Arteriole, inflammation, chronic active	–	1 (5%)	–	–
Artery, inflammation, chronic active	32 (82%)	12 (63%)	14 (82%)	5 (71%)
Artery, mineral	21 (54%)	4 (21%)	5 (29%)	2 (29%)
Vein, degeneration	1 (3%)	–	–	–
Vein, inflammation, chronic active	1 (3%)	1 (5%)	–	1 (14%)
Oral mucosa	(0)	(2)	(0)	(2)
Hyperplasia	–	–	–	1 (50%)
Ulcer	–	1 (50%)	–	–
Pancreas	(90)	(89)	(88)	(86)
Cyst	1 (1%)	–	–	–
Inflammation, chronic active	–	–	–	1 (1%)
Thrombus	1 (1%)	–	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Acinus, atrophy	13 (14%)	16 (18%)	10 (11%)	11 (13%)
Acinus, hyperplasia	63 (70%)	58 (65%)	44 (50%)	32 (37%)
Artery, inflammation, chronic active	48 (53%)	28 (31%)	26 (30%)	14 (16%)
Artery, mineral	11 (12%)	3 (3%)	3 (3%)	1 (1%)
Salivary glands	(90)	(90)	(90)	(90)
Inflammation, chronic active	–	1 (1%)	–	–
Artery, inflammation, chronic active	11 (12%)	7 (8%)	3 (3%)	1 (1%)
Artery, mineral	2 (2%)	–	–	–
Duct, parotid gland, dilation	5 (6%)	3 (3%)	1 (1%)	4 (4%)
Duct, parotid gland, inflammation, acute	1 (1%)	–	–	–
Parotid gland, atrophy	18 (20%)	16 (18%)	14 (16%)	14 (16%)
Parotid gland, inflammation, acute	2 (2%)	7 (8%)	3 (3%)	1 (1%)
Parotid gland, vacuolation, cytoplasmic	1 (1%)	–	–	–
Sublingual gland, inflammation, acute	–	–	1 (1%)	–
Submandibular gland, atrophy	–	1 (1%)	1 (1%)	–
Stomach, forestomach	(90)	(90)	(90)	(90)
Cyst	–	1 (1%)	–	–
Edema	5 (6%)	11 (12%)	3 (3%)	2 (2%)
Erosion	–	1 (1%)	1 (1%)	
Fibrosis	–	–	–	1 (1%)
Inflammation, acute	1 (1%)	1 (1%)	–	–
Inflammation, chronic active	7 (8%)	14 (16%)	5 (6%)	6 (7%)
Mineral	1 (1%)	–	1 (1%)	–
Necrosis	–	–	–	1 (1%)
Ulcer	6 (7%)	8 (9%)	3 (3%)	2 (2%)
Artery, inflammation, chronic active		4 (4%)	3 (3%)	–
Epithelium, degeneration	–	1 (1%)	–	–
Epithelium, hyperplasia	11 (12%)	21 (23%)	12 (13%)	11 (12%)
Epithelium, hyperplasia, atypical	1 (1%)	–	–	–
Epithelium, hyperplasia, basal cell	–	–	1 (1%)	–
Stomach, glandular	(86)	(88)	(87)	(86)
Erosion	3 (3%)	–	2 (2%)	1 (1%)
Hemorrhage	–	–	1 (1%)	–
Inflammation, granulomatous	–	1 (1%)	–	–
Inflammation, acute	1 (1%)	–	1 (1%)	–
Inflammation, chronic active	1 (1%)	3 (3%)	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Mineral	31 (36%)	7 (8%)	8 (9%)	4 (5%)
Ulcer	–	–	1 (1%)	–
Artery, inflammation, chronic active	3 (3%)	2 (2%)	2 (2%)	1 (1%)
Tooth	(0)	(1)	(1)	(0)
Dysplasia	–	1 (100%)	–	–
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Aneurysm	–	1 (1%)	–	–
Dilation	–	1 (1%)	3 (3%)	–
Mineral	30 (33%)	7 (8%)	12 (13%)	6 (7%)
Blood vessel	(1)	(2)	(1)	(0)
Mineral	1 (100%)	–	–	–
Pulmonary artery, inflammation, chronic active	–	1 (50%)	1 (100%)	–
Pulmonary artery, necrosis	–	1 (50%)	1 (100%)	–
Heart	(90)	(90)	(90)	(90)
Cardiomyopathy	79 (88%)	82 (91%)	78 (87%)	79 (88%)
Congestion	1 (1%)	–	–	–
Hemorrhage		–	–	1 (1%)
Thrombus	1 (1%)	–	–	–
Artery, infiltration cellular, histiocyte	–	–	1 (1%)	–
Artery, inflammation, chronic active	–	5 (6%)	4 (4%)	2 (2%)
Artery, mineral	20 (22%)	7 (8%)	3 (3%)	2 (2%)
Artery, necrosis	–	1 (1%)	–	–
Atrium, dilation	3 (3%)	–	1 (1%)	–
Atrium, thrombus	1 (1%)	1 (1%)	–	–
Atrium, myocardium, hypertrophy	1 (1%)	–	–	–
Endocardium, hyperplasia, Schwann cell	–	1 (1%)	–	2 (2%)
Myocardium, mineral	9 (10%)	2 (2%)	4 (4%)	1 (1%)
Myocardium, necrosis	1 (1%)	–	–	–
Valve, inflammation, chronic active	1 (1%)	–	–	–
Ventricle right, cardiomyopathy	54 (60%)	62 (69%)	72 (80%)	74 (82%)
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(88)
Accessory adrenal cortical nodule	6 (7%)	7 (8%)	6 (7%)	4 (5%)
Angiectasis	–	–	1 (1%)	2 (2%)
Atrophy	–	–	1 (1%)	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Congestion	–	–	1 (1%)	–
Degeneration	3 (3%)	–	–	–
Degeneration, cystic	–	–	2 (2%)	2 (2%)
Hemorrhage	–	–	1 (1%)	–
Hyperplasia	47 (52%)	46 (51%)	46 (51%)	45 (51%)
Hypertrophy	35 (39%)	43 (48%)	50 (56%)	54 (61%)
Necrosis	5 (6%)	3 (3%)	4 (4%)	–
Thrombus	2 (2%)	1 (1%)	–	–
Vacuolation, cytoplasmic	20 (22%)	32 (36%)	25 (28%)	22 (25%)
Adrenal medulla	(88)	(90)	(89)	(87)
Degeneration, cystic	–	1 (1%)	–	–
Hyperplasia	42 (48%)	24 (27%)	26 (29%)	35 (40%)
Thrombus	1 (1%)	–	–	–
Islets, pancreatic	(90)	(89)	(86)	(85)
Hyperplasia	12 (13%)	5 (6%)	5 (6%)	7 (8%)
Parathyroid gland	(83)	(87)	(87)	(81)
Cyst	–	–	–	1 (1%)
Hyperplasia	51 (61%)	35 (40%)	46 (53%)	28 (35%)
Hyperplasia, focal	–	–	2 (2%)	1 (1%)
Pituitary gland	(89)	(90)	(90)	(90)
Necrosis	–	–	1 (1%)	–
Craniopharyngeal duct, cyst	1 (1%)	–	–	–
Pars distalis, cyst	5 (6%)	9 (10%)	15 (17%)	16 (18%)
Pars distalis, hyperplasia	32 (36%)	34 (38%)	35 (39%)	32 (36%)
Pars distalis, necrosis	–	–	–	1 (1%)
Pars intermedia, angiectasis	1 (1%)	–	–	–
Pars intermedia, cyst	6 (7%)	5 (6%)	9 (10%)	6 (7%)
Pars intermedia, hyperplasia	1 (1%)	1 (1%)	2 (2%)	2 (2%)
Pars nervosa, cyst	–	1 (1%)	–	–
Pars nervosa, developmental malformation	–	–	1 (1%)	1 (1%)
Pars nervosa, infiltration cellular, mixed cell	–	–	–	1 (1%)
Thyroid gland	(89)	(89)	(89)	(87)
Congestion	–	–	–	1 (1%)
Ectopic thymus	–	–	1 (1%)	–
C-cell, hyperplasia	16 (18%)	24 (27%)	18 (20%)	14 (16%)
Follicle, cyst	–	1 (1%)	1 (1%)	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Follicle, hyperplasia, cystic	1 (1%)	–	–	–
Follicular cell, hyperplasia	–	1 (1%)	–	–
Follicular cell, hypertrophy	–	1 (1%)	–	1 (1%)
General Body System				
Tissues NOS	(3)	(4)	(4)	(5)
Inflammation, chronic active	–	1 (25%)	–	–
Abdominal, fat, hemorrhage	1 (33%)	–	–	–
Abdominal, fat, inflammation, chronic active	–	1 (25%)	–	–
Fat, necrosis	2 (67%)	–	2 (50%)	3 (60%)
Mediastinum, inflammation, chronic active	–	–	1 (25%)	1 (20%)
Genital System				
Bulbourethral gland	(1)	(0)	(0)	(0)
Coagulating gland	(0)	(0)	(0)	(1)
Inflammation, chronic active	–	–	–	1 (100%)
Ductus deferens	(1)	(0)	(0)	(0)
Granuloma	1 (100%)	–	–	–
Epididymis	(90)	(90)	(90)	(90)
Exfoliated germ cell	51 (57%)	26 (29%)	29 (32%)	15 (17%)
Granuloma sperm	1 (1%)	–	–	–
Hypospermia	28 (31%)	20 (22%)	23 (26%)	8 (9%)
Inflammation, acute	–	1 (1%)	–	–
Inflammation, chronic active	–	1 (1%)	–	–
Artery, inflammation, chronic active	2 (2%)	2 (2%)	1 (1%)	2 (2%)
Penis	(0)	(0)	(0)	(1)
Preputial gland	(88)	(90)	(90)	(90)
Atrophy	1 (1%)	–	–	–
Hyperplasia	1 (1%)	–	–	–
Inflammation, suppurative		1 (1%)	3 (3%)	–
Inflammation, granulomatous	1 (1%)	–	–	–
Inflammation, acute	1 (1%)	–	–	–
Inflammation, chronic active	46 (52%)	48 (53%)	54 (60%)	52 (58%)
Artery, inflammation, chronic active	1 (1%)	–	–	1 (1%)
Duct, dilation	51 (58%)	53 (59%)	49 (54%)	51 (57%)
Duct, hyperplasia	–	1 (1%)	–	1 (1%)
Duct, mineral	–	–	–	1 (1%)
Prostate	(90)	(90)	(90)	(90)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Decreased secretory fluid	4 (4%)	6 (7%)	6 (7%)	2 (2%)
Hemorrhage	1 (1%)	–	–	–
Inflammation cellular, mononuclear cell	1 (1%)	–	–	1 (1%)
Inflammation, suppurative	–	1 (1%)	1 (1%)	–
Inflammation, acute	7 (8%)	3 (3%)	6 (7%)	4 (4%)
Inflammation, chronic active	6 (7%)	15 (17%)	9 (10%)	13 (14%)
Artery, inflammation, chronic active	1 (1%)	1 (1%)	1 (1%)	–
Epithelium, hyperplasia	5 (6%)	13 (14%)	11 (12%)	11 (12%)
Seminal vesicle	(90)	(89)	(89)	(90)
Decreased secretory fluid	35 (39%)	18 (20%)	22 (25%)	11 (12%)
Degeneration	–	–	–	1 (1%)
Hemorrhage	1 (1%)	–	–	–
Inflammation, acute	4 (4%)	–	3 (3%)	1 (1%)
Inflammation, chronic	–	–	1 (1%)	–
Inflammation, chronic active	1 (1%)	4 (4%)	1 (1%)	1 (1%)
Artery, inflammation, chronic active	1 (1%)	1 (1%)	–	–
Epithelium, hyperplasia	1 (1%)	–	–	–
Testis	(90)	(90)	(90)	(90)
Cyst	1 (1%)	–	–	–
Edema	–	2 (2%)	3 (3%)	2 (2%)
Inflammation, chronic active	2 (2%)	–	–	–
Pigment	1 (1%)	–	–	–
Artery, inflammation, chronic active	52 (58%)	40 (44%)	37 (41%)	20 (22%)
Germ cell, degeneration	51 (57%)	35 (39%)	42 (47%)	20 (22%)
Germinal epithelium, mineral	–	1 (1%)	–	–
Interstitial cell, hyperplasia	1 (1%)	2 (2%)	–	4 (4%)
Rete testis, dilation	1 (1%)	–	–	–
Seminiferous tubule, dilation	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Tunic, hemorrhage	–	1 (1%)	–	–
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Fibrosis	–	–	–	1 (1%)
Hemorrhage	–	1 (1%)	3 (3%)	–
Hypercellularity	15 (17%)	42 (47%)	32 (36%)	23 (26%)
Lymph node	(25)	(22)	(18)	(12)
Artery, mediastinal, inflammation, chronic active	–	1 (5%)	–	1 (8%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Artery, mediastinal, mineral	–	1 (5%)	–	–
Bronchial, erythrophagocytosis	–	–	3 (17%)	–
Iliac, erythrophagocytosis	2 (8%)	2 (9%)	1 (6%)	–
Iliac, hyperplasia, lymphocyte	2 (8%)	–	–	1 (8%)
Iliac, infiltration cellular, histiocyte	2 (8%)	–	1 (6%)	
Iliac, pigment	–	–	1 (6%)	–
Iliac, proliferation, plasma cell	3 (12%)	1 (5%)	1 (6%)	2 (17%)
Iliac, lymphatic sinus, ectasia	5 (20%)	2 (9%)	–	1 (8%)
Lumbar, erythrophagocytosis	2 (8%)	1 (5%)	1 (6%)	1 (8%)
Lumbar, hemorrhage	–	1 (5%)	–	–
Lumbar, hyperplasia, lymphocyte	–	1 (5%)	–	1 (8%)
Lumbar, proliferation, plasma cell	–	1 (5%)	–	1 (8%)
Lumbar, lymphatic sinus, ectasia	–	2 (9%)	1 (6%)	–
Lymphatic sinus, mediastinal, ectasia	1 (4%)	1 (5%)	–	1 (8%)
Lymphatic sinus, renal, ectasia	–	3 (14%)	1 (6%)	1 (8%)
Mediastinal, congestion	–	–	2 (11%)	–
Mediastinal, erythrophagocytosis	6 (24%)	5 (23%)	5 (28%)	6 (50%)
Mediastinal, hemorrhage	1 (4%)	1 (5%)	–	–
Mediastinal, infiltration cellular, histiocyte	–	1 (5%)	–	–
Pancreatic, erythrophagocytosis	3 (12%)	1 (5%)	–	2 (17%)
Pancreatic, hemorrhage	1 (4%)	–	–	–
Pancreatic, hyperplasia, lymphocyte	1 (4%)	–	–	–
Pancreatic, proliferation, plasma cell	–	–	1 (6%)	–
Renal, erythrophagocytosis	8 (32%)	4 (18%)	3 (17%)	1 (8%)
Renal, hemorrhage	–	1 (5%)	–	–
Renal, hyperplasia, lymphocyte	–	1 (5%)	–	1 (8%)
Renal, proliferation, plasma cell	2 (8%)	–	–	1 (8%)
Lymph node, mandibular	(89)	(90)	(89)	(90)
Atrophy, lymphoid	–	1 (1%)	–	–
Congestion	–	–	–	3 (3%)
Erythrophagocytosis	–	2 (2%)	4 (4%)	3 (3%)
Hemorrhage	–	1 (1%)	–	–
Hyperplasia, lymphocyte	41 (46%)	50 (56%)	54 (61%)	57 (63%)
Infiltration cellular, histiocyte	–	–	–	1 (1%)
Infiltration cellular, polymorphonuclear	2 (2%)	–	–	–
Inflammation, suppurative	–	–	–	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Inflammation, chronic active	–	–	–	1 (1%)
Pigment	–	–	1 (1%)	1 (1%)
Proliferation, plasma cell	49 (55%)	67 (74%)	69 (78%)	68 (76%)
Lymphatic sinus, ectasia	16 (18%)	12 (13%)	20 (22%)	16 (18%)
Lymph node, mesenteric	(90)	(89)	(86)	(89)
Atrophy	–	1 (1%)	–	–
Depletion cellular	–	–	1 (1%)	–
Erythrophagocytosis	17 (19%)	7 (8%)	7 (8%)	8 (9%)
Hyperplasia, lymphocyte	2 (2%)	–	1 (1%)	4 (4%)
Infiltration cellular, histiocyte	1 (1%)	–	1 (1%)	–
Infiltration cellular, polymorphonuclear	2 (2%)	–	–	–
Proliferation, plasma cell	–	–	–	1 (1%)
Artery, inflammation, chronic active	–	–	1 (1%)	–
Lymphatic sinus, ectasia	–	3 (3%)	2 (2%)	1 (1%)
Lymphocyte, depletion	2 (2%)	–	–	–
Spleen	(90)	(90)	(89)	(90)
Congestion	–	–	–	1 (1%)
Developmental malformation	1 (1%)	–	–	–
Erythrophagocytosis	–	–	–	1 (1%)
Extramedullary hematopoiesis	45 (50%)	58 (64%)	56 (63%)	64 (71%)
Hemorrhage	–	–	2 (2%)	–
Hyperplasia, lymphocyte	5 (6%)	2 (2%)	2 (2%)	
Hyperplasia, plasma cell	–	–	1 (1%)	2 (2%)
Pigment	57 (63%)	62 (69%)	74 (83%)	74 (82%)
Arteriole, mineral	1 (1%)	–	–	–
Artery, inflammation, chronic active	–	1 (1%)	4 (4%)	1 (1%)
Artery, mineral	–	–	1 (1%)	–
Capsule, fibrosis	–	1 (1%)	–	–
Red pulp, atrophy	26 (29%)	10 (11%)	10 (11%)	3 (3%)
White pulp, atrophy	30 (33%)	16 (18%)	13 (15%)	11 (12%)
Thymus	(88)	(86)	(88)	(86)
Atrophy	79 (90%)	71 (83%)	75 (85%)	78 (91%)
Congestion	–	–	–	1 (1%)
Cyst	10 (11%)	10 (12%)	9 (10%)	10 (12%)
Ectopic parathyroid gland	6 (7%)	1 (1%)	2 (2%)	3 (3%)
Ectopic thyroid	1 (1%)	4 (5%)	–	2 (2%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Hemorrhage	2 (2%)	2 (2%)	2 (2%)	2 (2%)
Hyperplasia, epithelial	2 (2%)	4 (5%)	2 (2%)	2 (2%)
Thrombus	–	–	2 (2%)	–
Artery, inflammation, chronic active	6 (7%)	3 (3%)	2 (2%)	1 (1%)
Integumentary System				
Mammary gland	(82)	(76)	(82)	(82)
Atrophy	1 (1%)	–	2 (2%)	–
Galactocele	1 (1%)	–	–	–
Hyperplasia	–	2 (3%)	5 (6%)	2 (2%)
Inflammation, granulomatous	–	1 (1%)	–	–
Artery, inflammation, chronic active	–	1 (1%)	–	–
Duct, dilation	3 (4%)	13 (17%)	3 (4%)	13 (16%)
Skin	(90)	(90)	(90)	(90)
Cyst epithelial inclusion	3 (3%)	6 (7%)	8 (9%)	10 (11%)
Cyst epithelial inclusion, multifocal	–	–	–	1 (1%)
Hyperkeratosis	–	1 (1%)	2 (2%)	–
Inflammation, chronic	–	–	1 (1%)	–
Inflammation, chronic active	1 (1%)	1 (1%)	2 (2%)	1 (1%)
Ulcer	2 (2%)	3 (3%)	2 (2%)	–
Artery, subcutaneous tissue, inflammation, chronic active	1 (1%)	–	–	–
Epidermis, hyperplasia	1 (1%)	2 (2%)	2 (2%)	1 (1%)
Hair follicle, atrophy	–	1 (1%)	1 (1%)	
Hair follicle, dilation	–	–	–	1 (1%)
Lip, subcutaneous tissue, foreign body	–	–	–	1 (1%)
Lip, subcutaneous tissue, inflammation, chronic active	–	–	–	1 (1%)
Prepuce, cyst epithelial inclusion	–	1 (1%)	–	–
Subcutaneous tissue, degeneration	–	1 (1%)	–	–
Subcutaneous tissue, fibrosis	–	–	–	1 (1%)
Subcutaneous tissue, inflammation, suppurative	1 (1%)	–	–	–
Subcutaneous tissue, inflammation, acute		1 (1%)	–	–
Subcutaneous tissue, inflammation, chronic	1 (1%)	–	–	–
Subcutaneous tissue, inflammation, chronic active	–	2 (2%)	1 (1%)	2 (2%)
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Fibrous osteodystrophy	46 (51%)	18 (20%)	14 (16%)	6 (7%)
Increased bone	–	–	–	1 (1%)
Skeletal muscle	(90)	(90)	(90)	(90)
Degeneration	34 (38%)	49 (54%)	43 (48%)	37 (41%)
Mineral	2 (2%)	–	–	–
Nervous System				
Brain	(90)	(90)	(90)	(90)
Compression	7 (8%)	9 (10%)	4 (4%)	10 (11%)
Cyst	–	1 (1%)	1 (1%)	1 (1%)
Edema	–	2 (2%)	1 (1%)	–
Hemorrhage	2 (2%)	1 (1%)	2 (2%)	–
Infiltration cellular, mononuclear cell	1 (1%)	–	–	–
Mineral	5 (6%)	4 (4%)	6 (7%)	2 (2%)
Necrosis	7 (8%)	3 (3%)	4 (4%)	3 (3%)
Vacuolation, cytoplasmic	–	1 (1%)	–	–
Brain stem, hemorrhage	–	–	1 (1%)	–
Cerebellum, atrophy	–	–	–	2 (2%)
Choroid plexus, degeneration	1 (1%)	–	–	–
Choroid plexus, mineral	3 (3%)	1 (1%)	–	–
Glial cell, hyperplasia	–	2 (2%)	3 (3%)	1 (1%)
Meninges, hyperplasia	1 (1%)	–	–	–
Meninges, hyperplasia, granular cell	1 (1%)	–	1 (1%)	–
Meninges, metaplasia, osseous	–	–	1 (1%)	–
Meninges, mineral	–	1 (1%)	–	–
Perivascular, infiltration cellular, mononuclear cell	–	1 (1%)	–	–
Pineal gland, infiltration cellular, mononuclear cell	–	–	1 (1%)	1 (1%)
Pineal gland, mineral	3 (3%)	10 (11%)	8 (9%)	3 (3%)
Pineal gland, vacuolation, cytoplasmic	12 (13%)	19 (21%)	20 (22%)	13 (14%)
Nerve trigeminal	(84)	(88)	(87)	(88)
Degeneration	63 (75%)	69 (78%)	65 (75%)	63 (72%)
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Degeneration	86 (96%)	88 (98%)	90 (100%)	87 (97%)
Infiltration cellular, mononuclear cell	1 (1%)	–	–	–
Peripheral nerve, tibial	(88)	(89)	(90)	(88)
Degeneration	84 (95%)	84 (94%)	90 (100%)	85 (97%)
Spinal cord, cervical	(90)	(90)	(90)	(90)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Degeneration	30 (33%)	38 (42%)	41 (46%)	32 (36%)
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Degeneration	21 (23%)	10 (11%)	17 (19%)	12 (13%)
Nerve, degeneration	79 (88%)	82 (91%)	87 (97%)	81 (90%)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Degeneration	58 (64%)	68 (76%)	72 (80%)	69 (77%)
Hemorrhage, focal	1 (1%)	–	–	–
Trigeminal ganglion	(75)	(73)	(77)	(77)
Degeneration	23 (31%)	25 (34%)	22 (29%)	15 (19%)
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Congestion	13 (14%)	15 (17%)	11 (12%)	10 (11%)
Cyst	–	1 (1%)	–	–
Fibrosis	–	1 (1%)	–	–
Foreign body	4 (4%)	–	2 (2%)	1 (1%)
Hemorrhage	3 (3%)	4 (4%)	3 (3%)	
Inflammation, suppurative	3 (3%)	1 (1%)	2 (2%)	1 (1%)
Inflammation, granulomatous	–	–	3 (3%)	–
Inflammation, chronic active	2 (2%)	8 (9%)	5 (6%)	3 (3%)
Inflammation, subacute	2 (2%)	–	–	–
Mineral	–	–	1 (1%)	–
Alveolar epithelium, hyperplasia	–	–	1 (1%)	–
Alveolus, infiltration cellular, histiocyte	37 (41%)	40 (44%)	43 (48%)	48 (53%)
Artery, inflammation, chronic active	3 (3%)	5 (6%)	6 (7%)	3 (3%)
Artery, mineral	1 (1%)	–	–	–
Artery, mediastinum, inflammation, chronic active	2 (2%)	–	–	–
Bronchiole, hyperplasia, epithelial	–	–	1 (1%)	–
Epithelium, alveolus, hyperplasia	3 (3%)	3 (3%)	3 (3%)	1 (1%)
Interstitium, fibrosis	–	–	1 (1%)	–
Interstitium, mineral	1 (1%)	–	2 (2%)	–
Perivascular, inflammation, chronic active	1 (1%)	–	–	–
Nose	(89)	(90)	(90)	(89)
Foreign body	5 (6%)	3 (3%)	2 (2%)	4 (4%)
Fungus	–	1 (1%)	–	–
Hyperplasia, lymphocyte	–	–	2 (2%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Inflammation, suppurative	10 (11%)	12 (13%)	13 (14%)	10 (11%)
Inflammation, chronic active	–	1 (1%)	2 (2%)	–
Mineral	–	–	1 (1%)	–
Nasopharyngeal duct, respiratory epithelium, hyperplasia	1 (1%)	–	–	–
Olfactory epithelium, accumulation, hyaline droplet	79 (89%)	87 (97%)	82 (91%)	81 (91%)
Olfactory epithelium, atrophy	–	–	1 (1%)	–
Olfactory epithelium, hyperplasia	–	–	2 (2%)	–
Olfactory epithelium, metaplasia, respiratory	3 (3%)	6 (7%)	7 (8%)	2 (2%)
Respiratory epithelium, accumulation, hyaline droplet	3 (3%)	2 (2%)	1 (1%)	1 (1%)
Respiratory epithelium, atrophy	–	2 (2%)	–	–
Respiratory epithelium, hyperplasia	3 (3%)	11 (12%)	14 (16%)	11 (12%)
Respiratory epithelium, hyperplasia, goblet cell	1 (1%)	–	–	–
Respiratory epithelium, metaplasia, squamous	–	–	1 (1%)	–
Respiratory epithelium, mineral	1 (1%)	–	–	–
Septum, developmental malformation	–	–	–	1 (1%)
Trachea	(90)	(88)	(87)	(86)
Artery, inflammation, chronic active	–	1 (1%)	–	1 (1%)
Artery, mineral	1 (1%)	–	–	–
Epithelium, hyperplasia	1 (1%)	–	–	–
Epithelium, metaplasia, squamous	1 (1%)	–	–	–
Glands, inflammation, acute	–	–	–	1 (1%)
Special Senses System				
Eye	(85)	(86)	(87)	(83)
Retinal detachment	1 (1%)	–	–	–
Anterior chamber, inflammation, acute	4 (5%)	5 (6%)	3 (3%)	2 (2%)
Cornea, degeneration	–	1 (1%)	–	–
Cornea, fibrosis	1 (1%)	3 (3%)	4 (5%)	6 (7%)
Cornea, inflammation, acute	28 (33%)	33 (38%)	25 (29%)	25 (30%)
Cornea, neovascularization	10 (12%)	19 (22%)	20 (23%)	19 (23%)
Cornea, ulcer	6 (7%)	2 (2%)	1 (1%)	2 (2%)
Cornea, epithelium, degeneration	–	2 (2%)	2 (2%)	2 (2%)
Cornea, epithelium, hyperplasia	13 (15%)	17 (20%)	15 (17%)	20 (24%)
Cornea, epithelium, regeneration	–	2 (2%)	2 (2%)	–
Lens, cataract	–	2 (2%)	–	2 (2%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Retina, atrophy	6 (7%)	10 (12%)	12 (14%)	14 (17%)
Retina, degeneration	1 (1%)	–	–	–
Retina, dysplasia	–	1 (1%)	–	–
Retina, gliosis	–	1 (1%)	–	–
Harderian gland	(90)	(90)	(90)	(90)
Atrophy	1 (1%)	1 (1%)	1 (1%)	–
Cyst	–	–	1 (1%)	–
Degeneration, cystic	2 (2%)	–	–	3 (3%)
Hyperplasia	–	–	–	3 (3%)
Hypertrophy	–	2 (2%)		1 (1%)
Inflammation, granulomatous	–	–	2 (2%)	–
Inflammation, acute	2 (2%)	–	–	–
Inflammation, chronic	–	–	2 (2%)	–
Inflammation, chronic active	2 (2%)		1 (1%)	1 (1%)
Lacrimal gland	(2)	(1)	(2)	(2)
Inflammation, granulomatous	–	–	1 (50%)	–
Metaplasia, Harderian gland	2 (100%)	1 (100%)	2 (100%)	2 (100%)
Urinary System				
Kidney	(90)	(90)	(90)	(90)
Infarct	–	–	1 (1%)	–
Inflammation, suppurative	–	–	1 (1%)	1 (1%)
Mineral	1 (1%)	–	1 (1%)	1 (1%)
Nephropathy, chronic progressive	88 (98%)	89 (99%)	90 (100%)	89 (99%)
Thrombus	1 (1%)	–	–	–
Artery, inflammation, chronic active	–	–	–	1 (1%)
Artery, mineral	2 (2%)	1 (1%)	–	–
Pelvis, dilation	1 (1%)	2 (2%)	1 (1%)	–
Pelvis, inflammation, suppurative	–	1 (1%)	1 (1%)	1 (1%)
Pelvis, inflammation, chronic active	–	–	–	1 (1%)
Renal tubule, accumulation, hyaline droplet	–	–	1 (1%)	1 (1%)
Renal tubule, cyst	18 (20%)	17 (19%)	14 (16%)	6 (7%)
Renal tubule, hyperplasia	–	2 (2%)	1 (1%)	2 (2%)
Renal tubule, hyperplasia, atypical	2 (2%)	–	–	–
Renal tubule, hyperplasia, oncocytic	2 (2%)	–	–	–
Urothelium, hyperplasia	1 (1%)	2 (2%)	1 (1%)	2 (2%)
Ureter	(0)	(1)	(0)	(0)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Dilation	–	1 (100%)	–	–
Urethra	(0)	(0)	(1)	(0)
Urinary bladder	(89)	(89)	(86)	(85)
Dilation	–	–	1 (1%)	–
Hemorrhage	2 (2%)	–	–	–
Inflammation, suppurative	–	–	–	1 (1%)
Inflammation, acute	2 (2%)	–	–	–
Inflammation, chronic active	–	1 (1%)	–	–
Necrosis	1 (1%)	–	–	–
Artery, inflammation, chronic active		1 (1%)	–	–
Muscularis, degeneration	1 (1%)	–	–	–
Serosa, inflammation, chronic active	–	1 (1%)	–	–
Urothelium, hyperplasia	1 (1%)	1 (1%)	2 (2%)	1 (1%)

[a]Number of animals examined microscopically at the site and the number of animals with lesion.

Appendix B. Summary of Lesions in Female Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

Tables

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Table B-1. Summary of the Incidence of Neoplasms in Female Rats Exposed to GSM-modulated Cell Phone RFR for Two Years[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
Fourteen-week interim evaluation	15	15	15	15
Early deaths				
Accidental death	1	–	–	–
Moribund	30	25	31	22
Natural deaths	11	10	11	11
Survivors				
Died last week of study	1	3	1	–
Terminal euthanasia	47	52	47	57
Animals examined microscopically	100	100	100	100
Systems Examined at 14 Weeks with No Neoplasms Observed				
Alimentary System				
Cardiovascular System				
Endocrine System				
General Body System				
Genital System				
Hematopoietic System				
Integumentary System				
Musculoskeletal System				
Nervous System				
Respiratory System				
Special Senses System				
Urinary System				
Two-year Study				
Alimentary System				
Esophagus	(90)	(90)	(90)	(90)
Intestine large, cecum	(84)	(83)	(83)	(84)
Serosa, adenocarcinoma, metastatic, pancreas	–	–	1 (1%)	–
Intestine large, colon	(89)	(88)	(89)	(89)
Intestine large, rectum	(90)	(89)	(89)	(89)
Granular cell tumor benign	1 (1%)	–	–	–
Serosa, sarcoma, metastatic, uterus	–	1 (1%)	–	–
Intestine small, duodenum	(88)	(85)	(83)	(85)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adenocarcinoma	–	1 (1%)	–	–
Intestine small, ileum	(86)	(82)	(81)	(83)
Intestine small, jejunum	(83)	(82)	(81)	(84)
Leiomyosarcoma	1 (1%)	1 (1%)	–	–
Serosa, sarcoma stromal, metastatic, uterus	–	1 (1%)	–	–
Liver	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, intestine small, duodenum	–	1 (1%)	–	–
Adenocarcinoma, metastatic, pancreas	–	–	1 (1%)	–
Carcinoma, metastatic, kidney	–	1 (1%)	–	–
Carcinoma, metastatic, uncertain primary site	–	1 (1%)	–	–
Hepatocellular adenoma	7(8%)	1 (1%)	1 (1%)	3(3%)
Hepatocellular adenoma, multiple	–	1 (1%)	–	–
Hepatocellular carcinoma	–	1 (1%)	–	–
Sarcoma stromal, metastatic, uterus	–	1 (1%)	–	–
Serosa, adenocarcinoma, metastatic, uterus	–	1 (1%)	–	–
Mesentery	(4)	(5)	(5)	(5)
Adenocarcinoma, metastatic, intestine small, duodenum	–	1 (20%)	–	–
Adenocarcinoma, metastatic, pancreas	–	–	1 (20%)	–
Adenocarcinoma, metastatic, uterus	1 (25%)	–	–	–
Sarcoma stromal, metastatic, uterus	–	1 (20%)	–	–
Schwannoma malignant, metastatic, ovary	–	–	1 (20%)	
Oral mucosa	(1)	(0)	(0)	(0)
Squamous cell carcinoma	1 (100%)	–	–	–
Pancreas	(90)	(90)	(90)	(87)
Adenocarcinoma, metastatic, intestine small, duodenum	–	1 (1%)	–	–
Adenocarcinoma, metastatic, uterus	–	1 (1%)	–	–
Carcinoma, metastatic, uncertain primary site	–	1 (1%)	–	–
Sarcoma stromal metastatic, uterus	–	1 (1%)	–	–
Schwannoma malignant, metastatic, ovary	–	–	1 (1%)	–
Acinus, adenocarcinoma	–	–	2 (2%)	–
Salivary glands	(90)	(89)	(90)	(90)
Myoepithelioma	–	–	1 (1%)	–
Schwannoma malignant	–	–	–	1 (1%)
Parotid gland, adenoma	–	–	1 (1%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Stomach, forestomach	(90)	(90)	(90)	(90)
Sarcoma	1 (1%)	–	–	–
Squamous cell papilloma			1 (1%)	
Stomach, glandular	(90)	(89)	(90)	(89)
Sarcoma, metastatic, stomach, forestomach	1 (1%)	–	–	–
Serosa, adenocarcinoma, metastatic, pancreas	–	–	1 (1%)	–
Tongue	(1)	(0)	(0)	(0)
Squamous cell carcinoma	1 (100%)	–	–	–
Tooth	(0)	(0)	(1)	(0)
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Heart	(90)	(90)	(90)	(90)
Endocardium, schwannoma malignant	–	–	1 (1%)	–
Epicardium, paraganglioma	–	–	1 (1%)	–
Myocardium, schwannoma malignant	–	–	1 (1%)	–
Endocrine System				
Adrenal cortex	(90)	(90)	(89)	(90)
Adenocarcinoma, metastatic, intestine small, duodenum	–	1 (1%)	–	–
Adenoma, metastatic, pancreas	–	–	1 (1%)	–
Adenoma	1 (1%)	5 (6%)	1 (1%)	5 (6%)
Carcinoma	1 (1%)	–	–	–
Sarcoma stromal, metastatic, uterus	–	1 (1%)	–	–
Adrenal medulla	(86)	(90)	(90)	(86)
Adenocarcinoma, metastatic, pancreas	–	–	1 (1%)	–
Pheochromocytoma benign	1 (1%)	3 (3%)	3 (3%)	1 (1%)
Pheochromocytoma complex	–	1 (1%)	1 (1%)	–
Bilateral, pheochromocytoma benign	–	–	–	1 (1%)
Islets, pancreatic	(90)	(89)	(90)	(87)
Adenoma	5 (6%)	4 (4%)	3 (3%)	4 (5%)
Carcinoma	2 (2%)	1 (1%)	4 (4%)	4 (5%)
Parathyroid gland	(87)	(79)	(82)	(79)
Pituitary gland	(90)	(90)	(90)	(90)
Schwannoma malignant, metastatic, trigeminal ganglion	–	1 (1%)	–	–
Pars distalis, adenoma	42 (47%)	30 (33%)	35 (39%)	32 (36%)
Pars distalis, adenoma, multiple	1 (1%)	3 (3%)	3 (3%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pars distalis, carcinoma	1 (1%)	1 (1%)	–	1 (1%)
Thyroid gland	(90)	(88)	(90)	(88)
Carcinoma, metastatic, kidney	–	2 (2%)	–	–
Bilateral, C-cell, adenoma	–	1 (1%)	–	1 (1%)
Bilateral C-cell, adenoma, multiple	–	1 (1%)	–	–
C-cell, adenoma	6 (7%)	9 (10%)	8 (9%)	12 (14%)
C-cell, carcinoma	–	–	1 (1%)	1 (1%)
Follicular cell, carcinoma	1 (1%)	–	–	–
General Body System				
Tissue NOS	(8)	(10)	(8)	(10)
Chemodectoma benign	–	–	1 (13%)	–
Abdominal, schwannoma malignant	1 (13%)	–	–	–
Fat, adenocarcinoma, metastatic, uterus	–	–	1 (13%)	–
Mediastinum, paraganglioma	–	1 (10%)	–	–
Genital System				
Clitoral gland	(87)	(85)	(86)	(87)
Squamous cell carcinoma	–	–	–	1 (1%)
Ovary	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, intestine small, duodenum	–	1 (1%)	–	–
Adenocarcinoma, metastatic, pancreas	–	–	1 (1%)	–
Cystadenoma	1 (1%)	–	1 (1%)	–
Granulosa cell tumor benign	1 (1%)	–	–	–
Granulosa cell tumor malignant	2(2%)	–	1 (1%)	–
Lymphangiosarcoma	–	1 (1%)	–	–
Schwannoma malignant	–	–	1 (1%)	–
Sertoli cell tumor benign	1 (1%)	–	–	–
Sex cord stromal tumor, benign	–	1 (1%)	1 (1%)	–
Tubulostromal carcinoma	–	–	–	1 (1%)
Bilateral, rete ovarii, adenoma	–	–	–	1 (1%)
Periovarian tissue, sarcoma stromal, metastatic, uterus	–	1 (1%)	–	–
Oviduct	(1)	(0)	(0)	(0)
Uterus	(90)	(89)	(90)	(90)
Adenocarcinoma	3 (3%)	1 (1%)	2 (2%)	5 (6%)
Adenocarcinoma, metastatic, pancreas	–	–	1 (1%)	–
Adenoma	–	–	–	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Hemangioma	–	1 (1%)	–	–
Hemangiosarcoma	2 (2%)	–	–	–
Leiomyosarcoma	–	–	1 (1%)	–
Polyp stromal	15 (17%)	11 (12%)	10 (11%)	16 (18%)
Polyp stromal, multiple	1 (1%)	6 (7%)	1 (1%)	1 (1%)
Sarcoma stromal	–	1 (1%)	2 (2%)	–
Schwannoma malignant	1 (1%)	–	1 (1%)	–
Squamous cell carcinoma	–	–	2 (2%)	–
Cervix, leiomyosarcoma	1 (1%)	–	–	–
Cervix, malignant mixed mullerian tumor	–	–	–	1 (1%)
Cervix, polyp stromal	–	1 (1%)	–	–
Cervix, sarcoma	–	1 (1%)	–	–
Cervix, schwannoma malignant	1 (1%)	–	–	1 (1%)
Vagina	(2)	(3)	(1)	(1)
Granular cell tumor benign	–	1 (33%)	–	–
Sarcoma, metastatic, uterus	–	1 (33%)	–	–
Schwannoma malignant	1 (50%)	–	–	–
Schwannoma malignant, metastatic, uterus	1 (50%)	–	–	–
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Lymph node	(13)	(14)	(21)	(14)
Bronchial, adenocarcinoma, metastatic, intestine small, duodenum	–	1 (7%)	–	–
Lumbar, basal cell carcinoma, metastatic, skin	–	1 (7%)	–	–
Mediastinal, adenocarcinoma, metastatic, intestine small, duodenum	–	1 (7%)	–	–
Mediastinal, adenocarcinoma, metastatic, pancreas	–	–	1 (5%)	–
Lymph node, mandibular	(90)	(89)	(89)	(90)
Lymph node, mesenteric	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, intestine small, duodenum	–	1 (1%)	–	–
Hemangiosarcoma	–	–	1 (1%)	–
Spleen	(90)	(90)	(90)	(90)
Hemangiosarcoma	1 (1%)	–	–	1 (1%)
Sarcoma stromal, metastatic, uterus	–	1 (1%)		–
Capsule, adenocarcinoma, metastatic, intestine small, duodenum	–	1 (1%)	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Thymus	(87)	(86)	(88)	(86)
Schwannoma malignant	–	–	1 (1%)	–
Thymoma benign	1 (1%)	1 (1%)	–	–
Thymoma malignant	1 (1%)	1 (1%)	–	–
Integumentary System				
Mammary gland	(90)	(89)	(89)	(90)
Adenocarcinoma	9 (10%)	5 (6%)	8 (9%)	6 (7%)
Adenocarcinoma, multiple	1 (1%)	1 (1%)	–	–
Adenoma	4 (4%)	5 (6%)	2 (2%)	2 (2%)
Adenoma, multiple	4 (4%)	–	–	–
Fibroadenoma	34 (38%)	43 (48%)	38 (43%)	32 (36%)
Fibroadenoma, multiple	29 (32%)	25 (28%)	22 (25%)	30 (33%)
Myoepithelioma	–	–	–	1 (1%)
Skin	(90)	(90)	(90)	(90)
Basal cell carcinoma	–	1 (1%)	–	–
Keratoacanthoma	–	–	–	1 (1%)
Pilomatrixoma	–	–	1 (1%)	–
Squamous cell carcinoma	–	–	1 (1%)	1 (1%)
Squamous cell papilloma	–	–	1 (1%)	–
Subcutaneous tissue, fibroma	2 (2%)	3 (3%)	2 (2%)	4 (4%)
Subcutaneous tissue, fibrosarcoma	–	1 (1%)		1 (1%)
Subcutaneous tissue, lipoma	–	–	1 (1%)	–
Subcutaneous tissue, malignant fibrous histiocytoma	1 (1%)	–	–	–
Subcutaneous tissue, sarcoma	2 (2%)	–	–	–
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Skeletal muscle	(90)	(90)	(90)	(90)
Sarcoma stromal, metastatic, uterus	–	1 (1%)	–	–
Diaphragm, adenocarcinoma, metastatic, intestine small, duodenum	–	1 (1%)	–	–
Diaphragm, adenocarcinoma, metastatic, pancreas	–	–	1 (1%)	–
Diaphragm, carcinoma, metastatic, uncertain primary site	–	1 (1%)	–	–
Nervous System				
Brain	(90)	(90)	(90)	(90)
Carcinoma, metastatic, pituitary gland	1 (1%)	1 (1%)	–	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Glioma malignant	–	–	–	1 (1%)
Schwannoma malignant, metastatic, trigeminal ganglion	–	1 (1%)	–	–
Meninges, carcinoma, metastatic, kidney	–	2 (2%)	–	–
Meninges, granular cell tumor benign	1 (1%)	1 (1%)	2 (2%)	–
Meninges, granular cell tumor malignant	–	–	–	1 (1%)
Nerve trigeminal	(84)	(88)	(89)	(90)
Schwannoma malignant, metastatic, trigeminal ganglion	–	1 (1%)	–	–
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Peripheral nerve, tibial	(90)	(90)	(90)	(89)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Meninges, carcinoma, metastatic, kidney	–	1 (1%)	–	–
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Trigeminal ganglion	(81)	(79)	(80)	(79)
Schwannoma malignant	–	1 (1%)	–	–
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, uterus	–	–	1 (1%)	–
Alveolar/bronchiolar carcinoma	–	1 (1%)	–	–
Basal cell carcinoma, metastatic, skin	–	1 (1%)	–	–
Carcinoma, metastatic, thyroid gland	–	–	1 (1%)	1 (1%)
Carcinoma, metastatic, uncertain primary site	–	2 (2%)	–	–
Sarcoma stromal, metastatic, uterus	–	1 (1%)	–	–
Squamous cell carcinoma, metastatic, skin	–	–	1 (1%)	–
Nose	(90)	(90)	(90)	(90)
Trachea	(89)	(90)	(89)	(87)
Special Senses System				
Eye	(88)	(85)	(87)	(87)
Retrobulbar, schwannoma malignant, metastatic, trigeminal ganglion	–	1 (1%)	–	–
Harderian gland	(90)	(90)	(90)	(90)
Zymbal’s gland	(0)	(0)	(0)	(1)
Squamous cell papilloma	–	–	–	1 (100%)
Urinary System				
Kidney	(90)	(90)	(90)	(89)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adenocarcinoma, metastatic, intestine small, duodenum	–	1 (1%)	–	–
Carcinoma, metastatic, uncertain primary site	–	1 (1%)	–	–
Sarcoma stromal, metastatic, uterus	–	1 (1%)	–	–
Bilateral, renal tubule, carcinoma	1 (1%)	1 (1%)	–	–
Bilateral, renal tubule, carcinoma, multiple	–	1 (1%)	–	–
Renal tubule, adenoma	1 (1%)	1 (1%)	–	–
Renal tubule, adenoma, multiple	–	–	–	1 (1%)
Renal tubule, carcinoma, multiple	–	–	–	1 (1%)
Urinary bladder	(88)	(88)	(90)	(87)
Leiomyosarcoma	1 (1%)	–	–	–
Schwannoma malignant, metastatic, ovary	–	–	1 (1%)	–
Schwannoma malignant, metastatic, tissue NOS	1 (1%)	–	–	–
Serosa, adenocarcinoma, metastatic, pancreas	–	–	1 (1%)	–
Urothelium, carcinoma	–	1 (1%)	–	–
Systemic Lesions				
Multiple organs[b]	(90)	(90)	(90)	(90)
Histiocytic sarcoma	–	1 (1%)	–	1 (1%)
Leukemia mononuclear	–	1 (1%)	1 (1%)	–
Lymphoma malignant	5 (6%)	4 (4%)	2 (2%)	5 (6%)
Neoplasm Summary				
Total animals with primary neoplasms[c]				
Two-year study	89	87	82	86
Total primary neoplasms				
Two-year study	202	189	175	184
Total animals with benign neoplasms				
Two-year study	82	83	75	80
Total benign neoplasms				
Two-year study	159	159	140	150
Total animals with malignant neoplasms				
Two-year study	37	28	30	28
Total malignant neoplasms				
Two-year study	43	30	34	34
Total animals with metastatic neoplasms				
Two-year study	5	11	5	2
Total metastatic neoplasms				

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Two-year study	5	44	18	2
Total animals with malignant neoplasms-uncertain primary site				
Two-year study	–	2	–	–
Total animals with uncertain neoplasms-benign or malignant				
Two-year study	–	–	1	–
Total uncertain neoplasms				
Two-year study	–	–	1	–

[a]Number of animals examined microscopically at the site and the number of animals with neoplasm.
[b]Number of animals with any tissue examined microscopically.
[c]Primary neoplasms: all neoplasms except metastatic neoplasms.

Table B-2. Statistical Analysis of Primary Neoplasms in Female Rats Exposed to GSM-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adrenal Cortex: Adenoma				
Overall rate[a]	1/90 (1%)	5/90 (6%)	1/89 (1%)	5/90 (6%)
Rate per litters[b]	1/35 (3%)	5/35 (14%)	1/35 (3%)	4/35 (11%)
Adjusted rate[c]	1.4%	6.5%	1.4%	6.7%
Terminal rate[d]	1/48 (2%)	2/53 (4%)	1/48 (2%)	4/57 (7%)
First incidence (days)	737 (T)	464	737 (T)	651
Rao-Scott adjusted Poly-3 test[e]	P = 0.222	P = 0.182	P = 0.742N	P = 0.174
Litter C-A/Fisher's test[f]	P = 0.280	P = 0.099	P = 0.754	P = 0.178
Liver: Hepatocellular Adenoma				
Overall rate	7/90 (8%)	2/90 (2%)	1/90 (1%)	3/90 (3%)
Rate per litters	6/35 (17%)	2/35 (6%)	1/35 (3%)	3/35 (9%)
Adjusted rate	10.1%	2.6%	1.4%	4.0%
Terminal rate	6/48 (13%)	2/53 (4%)	1/48 (2%)	3/57 (5%)
First incidence (days)	707	737 (T)	737 (T)	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.168N	P = 0.106N	P = 0.061N	P = 0.183N
Litter C-A/Fisher's test	P = 0.207N	P = 0.130N	P = 0.053N	P = 0.239N
Liver: Hepatocellular Adenoma or Hepatocellular Carcinoma				
Overall rate	7/90 (8%)	3/90 (3%)	1/90 (1%)	3/90 (3%)
Rate per litters	6/35 (17%)	3/35 (9%)	1/35 (3%)	3/35 (9%)
Adjusted rate	10.1%	4.0%	1.4%	4.0%
Terminal rate	6/48 (13%)	2/53 (4%)	1/48 (2%)	3/57 (5%)
First incidence (days)	707	696	737 (T)	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.141N	P = 0.171N	P = 0.057N	P = 0.176N
Litter C-A/Fisher's test	P = 0.175N	P = 0.239N	P = 0.053N	P = 0.239N

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Mammary Gland: Fibroadenoma				
Overall rate	63/90 (70%)	68/90 (76%)	60/90 (67%)	62/90 (69%)
Rate per litters	31/35 (89%)	33/35 (94%)	33/35 (94%)	34/35 (97%)
Adjusted rate	77.0%	79.2%	71.7%	72.7%
Terminal rate	36/48 (75%)	39/53 (74%)	32/48 (67%)	38/57 (67%)
First incidence (days)	464	464	383	283
Rao-Scott adjusted Poly-3 test	P = 0.219N	P = 0.445	P = 0.287N	P = 0.334N
Litter C-A/Fisher's test	P = 0.134	P = 0.337	P = 0.337	P = 0.178
Mammary Gland: Adenoma				
Overall rate	8/90 (9%)	5/90 (6%)	2/90 (2%)	2/90 (2%)
Rate per litters	7/35 (20%)	5/35 (14%)	2/35 (6%)	2/35 (6%)
Adjusted rate	11.3%	6.6%	2.8%	2.7%
Terminal rate	5/48 (10%)	4/53 (8%)	2/48 (4%)	1/57 (2%)
First incidence (days)	524	718	737 (T)	669
Rao-Scott adjusted Poly-3 test	P = 0.038N	P = 0.271N	P = 0.075N	P = 0.065N
Litter C-A/Fisher's test	P = 0.036N	P = 0.376N	P = 0.075N	P = 0.075N
Mammary Gland: Fibroadenoma or Adenoma				
Overall rate	64/90 (71%)	69/90 (77%)	60/90 (67%)	62/90 (69%)
Rate per litters	31/35 (89%)	33/35 (94%)	33/35 (94%)	34/35 (97%)
Adjusted rate	77.7%	80.4%	71.7%	72.7%
Terminal rate	36/48 (75%)	40/53 (76%)	32/48 (67%)	38/57 (67%)
First incidence (days)	464	464	383	283
Rao-Scott adjusted Poly-3 test	P = 0.182N	P = 0.413	P = 0.260N	P = 0.304N
Litter C-A/Fisher's test	P = 0.134	P = 0.337	P = 0.337	P = 0.178
Mammary Gland: Adenocarcinoma				
Overall rate	10/90 (11%)	6/90 (7%)	8/90 (9%)	6/90 (7%)
Rate per litters	9/35 (26%)	6/35 (17%)	7/35 (20%)	6/35 (17%)
Adjusted rate	14.2%	7.8%	11.1%	7.9%
Terminal rate	6/48 (13%)	4/53 (8%)	5/48 (10%)	3/57 (5%)
First incidence (days)	622	332	489	489
Rao-Scott adjusted Poly-3 test	P = 0.218N	P = 0.171N	P = 0.379N	P = 0.177N
Litter C-A/Fisher's test	P = 0.284N	P = 0.281N	P = 0.388N	P = 0.281N
Mammary Gland: Adenoma or Adenocarcinoma				
Overall rate	16/90 (18%)	11/90 (12%)	10/90 (11%)	8/90 (9%)
Rate per litters	13/35 (37%)	11/35 (31%)	9/35 (26%)	7/35 (20%)
Adjusted rate	22.2%	14.2%	13.9%	10.5%
Terminal rate	9/48 (19%)	8/53 (15%)	7/48 (15%)	4/57 (7%)
First incidence (days)	524	332	489	489
Rao-Scott adjusted Poly-3 test	P = 0.052N	P = 0.153N	P = 0.146N	P = 0.049N

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Litter C-A/Fisher's test	P = 0.065N	P = 0.401N	P = 0.220N	P = 0.093N
Mammary Gland: Fibroadenoma, Adenoma, or Adenocarcinoma				
Overall rate	66/90 (73%)	72/90 (80%)	64/90 (71%)	64/90 (71%)
Rate per litters	31/35 (89%)	34/35 (97%)	35/35 (100%)	34/35 (97%)
Adjusted rate	79.6%	82.3%	75.3%	74.4%
Terminal rate	36/48 (75%)	41/53 (77%)	33/48 (69%)	39/57 (68%)
First incidence (days)	464	332	383	283
Rao-Scott adjusted Poly-3 test	P = 0.166N	P = 0.410	P = 0.326N	P = 0.282N
Litter C-A/Fisher's test	P = 0.102	P = 0.178	P = 0.057	P = 0.178
Pancreatic Islets: Adenoma				
Overall rate	5/90 (6%)	4/89 (4%)	3/90 (3%)	4/87 (5%)
Rate per litters	4/35 (11%)	4/35 (11%)	3/35 (9%)	4/35 (11%)
Adjusted rate	7.2%	5.3%	4.2%	5.5%
Terminal rate	5/48 (10%)	2/53 (4%)	2/48 (4%)	3/57 (5%)
First incidence (days)	737 (T)	699	621	669
Rao-Scott adjusted Poly-3 test	P = 0.422N	P = 0.451N	P = 0.346N	P = 0.466N
Litter C-A/Fisher's test	P = 0.555N	P = 0.645	P = 0.500N	P = 0.645
Pancreatic Islets: Carcinoma				
Overall rate	2/90 (2%)	1/89 (1%)	4/90 (4%)	4/87 (5%)
Rate per litters	2/35 (6%)	1/35 (3%)	4/35 (11%)	4/35 (11%)
Adjusted rate	2.9%	1.3%	5.6%	5.5%
Terminal rate	1/48 (2%)	1/53 (2%)	2/48 (4%)	4/57 (7%)
First incidence (days)	711	737 (T)	699	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.188	P = 0.467N	P = 0.349	P = 0.362
Litter C-A/Fisher's test	P = 0.157	P = 0.500N	P = 0.337	P = 0.337
Pancreatic Islets: Adenoma or Carcinoma				
Overall rate	7/90 (8%)	5/89 (6%)	7/90 (8%)	7/87 (8%)
Rate per litters	6/35 (17%)	5/35 (14%)	7/35 (20%)	7/35 (20%)
Adjusted rate	10.1%	6.7%	9.8%	9.6%
Terminal rate	6/48 (13%)	3/53 (6%)	4/48 (8%)	6/57 (11%)
First incidence (days)	711	699	621	669
Rao-Scott adjusted Poly-3 test	P = 0.487	P = 0.323N	P = 0.571N	P = 0.558N
Litter C-A/Fisher's test	P = 0.369	P = 0.500N	P = 0.500	P = 0.500
Pituitary Gland (Pars Distalis): Adenoma				
Overall rate	43/90 (48%)	33/90 (37%)	38/90 (42%)	32/90 (36%)
Rate per litters	28/35 (80%)	24/35 (69%)	26/35 (74%)	23/35 (66%)
Adjusted rate	57.1%	42.5%	51.2%	41.6%
Terminal rate	28/48 (58%)	23/53 (43%)	24/48 (50%)	24/57 (42%)
First incidence (days)	464	578	545	565

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Rao-Scott adjusted Poly-3 test	P = 0.077N	P = 0.049N	P = 0.283N	P = 0.038N
Litter C-A/Fisher's test	P = 0.162N	P = 0.206N	P = 0.388N	P = 0.141N
Pituitary Gland (Pars Distalis): Adenoma or Carcinoma				
Overall rate	44/90 (49%)	34/90 (38%)	38/90 (42%)	33/90 (37%)
Rate per litters	29/35 (83%)	24/35 (69%)	26/35 (74%)	23/35 (66%)
Adjusted rate	57.9%	43.8%	51.2%	42.8%
Terminal rate	28/48 (58%)	24/53 (45%)	24/48 (50%)	25/57 (44%)
First incidence (days)	464	578	545	565
Rao-Scott adjusted Poly-3 test	P = 0.082N	P = 0.056N	P = 0.250N	P = 0.044N
Litter C-A/Fisher's test	P = 0.111N	P = 0.132N	P = 0.281N	P = 0.085N
Skin (Subcutaneous Tissue): Fibroma, Fibrosarcoma, Sarcoma, or Malignant Fibrous Histiocytoma				
Overall rate	5/90 (6%)	4/90 (4%)	2/90 (2%)	5/90 (6%)
Rate per litters	5/35 (14%)	4/35 (11%)	2/35 (6%)	5/35 (14%)
Adjusted rate	7.0%	5.2%	2.8%	6.7%
Terminal rate	1/48 (2%)	3/53 (6%)	2/48 (4%)	4/57 (7%)
First incidence (days)	268	123	737 (T)	669
Rao-Scott adjusted Poly-3 test	P = 0.551N	P = 0.449N	P = 0.220N	P = 0.586N
Litter C-A/Fisher's test	P = 0.571	P = 0.500N	P = 0.214N	P = 0.633
Thyroid Gland (C-cell): Adenoma				
Overall rate	6/90 (7%)	11/88 (13%)	8/90 (9%)	13/88 (15%)
Rate per litters	6/35 (17%)	10/35 (29%)	8/35 (23%)	12/35 (34%)
Adjusted rate	8.5%	14.7%	11.3%	17.6%
Terminal rate	3/48 (6%)	8/53 (15%)	6/48 (13%)	12/57 (21%)
First incidence (days)	608	699	695	656
Rao-Scott adjusted Poly-3 test	P = 0.108	P = 0.188	P = 0.383	P = 0.091
Litter C-A/Fisher's test	P = 0.096	P = 0.197	P = 0.383	P = 0.085
Thyroid Gland (C-cell): Adenoma or Carcinoma				
Overall rate	6/90 (7%)	11/88 (13%)	9/90 (10%)	14/88 (16%)
Rate per litters	6/35 (17%)	10/35 (29%)	9/35 (26%)	13/35 (37%)
Adjusted rate	8.5%	14.7%	12.7%	18.9%
Terminal rate	3/48 (6%)	8/53 (15%)	7/48 (15%)	13/57 (23%)
First incidence (days)	608	699	695	656
Rao-Scott adjusted Poly-3 test	P = 0.068	P = 0.186	P = 0.290	P = 0.061
Litter C-A/Fisher's test	P = 0.054	P = 0.197	P = 0.281	P = 0.053
Uterus: Stromal Polyp				
Overall rate	16/90 (18%)	18/90 (20%)	11/90 (12%)	17/90 (19%)
Rate per litters	11/35 (31%)	14/35 (40%)	10/35 (29%)	13/35 (37%)
Adjusted rate	22.7%	23.5%	15.4%	22.3%
Terminal rate	14/48 (29%)	14/53 (26%)	6/48 (13%)	11/57 (19%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
First incidence (days)	531	602	656	594
Rao-Scott adjusted Poly-3 test	P = 0.461N	P = 0.530	P = 0.216N	P = 0.554N
Litter C-A/Fisher's test	P = 0.452	P = 0.309	P = 0.500N	P = 0.401
Uterus: Stromal Polyp or Stromal Sarcoma				
Overall rate	16/90 (18%)	19/90 (21%)	13/90 (14%)	17/90 (19%)
Rate per litters	11/35 (31%)	15/35 (43%)	11/35 (31%)	13/35 (37%)
Adjusted rate	22.7%	24.7%	18.2%	22.3%
Terminal rate	14/48 (29%)	14/53 (26%)	8/48 (17%)	11/57 (19%)
First incidence (days)	531	602	656	594
Rao-Scott adjusted Poly-3 test	P = 0.451N	P = 0.465	P = 0.341N	P = 0.553N
Litter C-A/Fisher's test	P = 0.476	P = 0.229	P = 0.601	P = 0.401
Uterus: Adenocarcinoma				
Overall rate	3/90 (3%)	1/90 (1%)	2/90 (2%)	5/90 (6%)
Rate per litters	2/35 (6%)	1/35 (3%)	2/35 (6%)	5/35 (14%)
Adjusted rate	4.3%	1.3%	2.8%	6.7%
Terminal rate	3/48 (6%)	1/53 (2%)	2/48 (4%)	5/57 (9%)
First incidence (days)	737 (T)	737 (T)	737 (T)	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.217	P = 0.316N	P = 0.500N	P = 0.420
Litter C-A/Fisher's test	P = 0.075	P = 0.500N	P = 0.693	P = 0.214
All Organs: Malignant Schwannoma				
Overall rate	4/90 (4%)	1/90 (1%)	5/90 (6%)	2/90 (2%)
Rate per litters	3/35 (9%)	1/35 (3%)	5/35 (14%)	2/35 (6%)
Adjusted rate	5.7%	1.3%	7.0%	2.7%
Terminal rate	2/48 (4%)	0/53 (0%)	2/48 (4%)	1/57 (2%)
First incidence (days)	489	480	578	622
Rao-Scott adjusted Poly-3 test	P = 0.428N	P = 0.212N	P = 0.519	P = 0.354N
Litter C-A/Fisher's test	P = 0.563N	P = 0.307N	P = 0.355	P = 0.500N
All Organs: Malignant Lymphoma				
Overall rate	5/90 (6%)	4/90 (4%)	2/90 (2%)	5/90 (6%)
Rate per litters	5/35 (14%)	3/35 (9%)	2/35 (6%)	5/35 (14%)
Adjusted rate	7.0%	5.2%	2.8%	6.5%
Terminal rate	0/48 (0%)	0/53 (0%)	0/48 (0%)	1/57 (2%)
First incidence (days)	268	545	450	299
Rao-Scott adjusted Poly-3 test	P = 0.537N	P = 0.454N	P = 0.229N	P = 0.572N
Litter C-A/Fisher's test	P = 0.518	P = 0.355N	P = 0.214N	P = 0.633
All Organs: Benign Neoplasms				
Overall rate	82/90 (91%)	83/90 (92%)	75/90 (83%)	80/90 (89%)
Rate per litters	35/35 (100%)	33/35 (94%)	35/35 (100%)	35/35 (100%)
Adjusted rate	97.1%	95.7%	88.3%	93.0%

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Terminal rate	48/48 (100%)	51/53 (96%)	41/48 (85%)	53/57 (93%)
First incidence (days)	464	464	383	283
Rao-Scott adjusted Poly-3 test	P = 0.129N	P = 0.469N	P = 0.031N	P = 0.196N
Litter C-A/Fisher's test	P = 0.405	P = 0.246N	–[g]	–
All Organs: Malignant Neoplasms				
Overall rate	37/90 (41%)	30/90 (33%)	30/90 (33%)	28/90 (31%)
Rate per litters	25/35 (71%)	21/35 (60%)	23/35 (66%)	22/35 (63%)
Adjusted rate	48.1%	36.6%	39.1%	35.3%
Terminal rate	21/48 (44%)	15/53 (28%)	14/48 (29%)	18/57 (32%)
First incidence (days)	268	123	439	299
Rao-Scott adjusted Poly-3 test	P = 0.100N	P = 0.093N	P = 0.165N	P = 0.070N
Litter C-A/Fisher's test	P = 0.349N	P = 0.225N	P = 0.399N	P = 0.306N
All Organs: Benign or Malignant Neoplasms				
Overall rate	89/90 (99%)	87/90 (97%)	82/90 (91%)	86/90 (96%)
Rate per litters	35/35 (100%)	34/35 (97%)	35/35 (100%)	35/35 (100%)
Adjusted rate	100%	97.2%	92.9%	97.2%
Terminal rate	48/48 (100%)	51/53 (96%)	42/48 (88%)	55/57 (97%)
First incidence (days)	268	123	383	283
Rao-Scott adjusted Poly-3 test	P = 0.220N	P = 0.226N	P = 0.028N	P = 0.226N
Litter C-A/Fisher's test	P = 0.567	P = 0.500N	–	–

(T) = terminal euthanasia.
[a]Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for adrenal gland, liver, pancreatic islets, pituitary gland, and thyroid gland; for other tissues, denominator is number of animals necropsied.
[b]Number of litters with tumor-bearing animals/number of litters examined at site.
[c]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[d]Observed incidence at terminal euthanasia.
[e]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
[f]The Litter Cochran-Armitage and Fishers exact tests directly compare the litter incidence rates.
[g]Value of statistic cannot be computed.

Table B-3. Historical Incidence of Malignant Schwannoma in Control Female Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Heart: Malignant Schwannoma	All Organs: Malignant Schwannoma
Historical Incidence: All Studies		
p-Chloro-α,α,α-trifluorotoluene (January 2011)	0/50	0/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	0/49	2/50
Indole-3-carbinol (March 2007)	0/50	2/50

Study (Study Start)	Heart: Malignant Schwannoma	All Organs: Malignant Schwannoma
Radiofrequency radiation (September 2012)	0/90	4/90
Overall Historical Incidence		
Total (%)	0/239	8/240 (3.3%)
Mean ± standard deviation	–	3.1% ± 2.1%
Range	–	0%–4%

[a]Data as of November 2017.

Table B-4. Historical Incidence of Brain Neoplasms in Control Female Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Malignant Glioma	Benign Granular Cell Tumor	Malignant Granular Cell Tumor	Benign or Malignant Granular Cell Tumor
Historical Incidence: All Studies				
p-Chloro-α,α,α-trifluorotoluene (January 2011)	1/50	0/50	0/50	0/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	0/50	1/50	0/50	1/50
Radiofrequency radiation (September 2012)	0/90	1/90	0/90	1/90
Overall Historical Incidence:				
Total (%)	1/190 (0.5%)	2/190 (1.1%)	0/190	2/190 (1.1%)
Mean ± standard deviation	0.7% ± 1.2%	1.0% ± 1.0%	–	1.0% ± 1.0%
Range	0%–2%	0%–2%	–	0%–2%

[a]Data as of November 2017; due to differences in sectioning method, indole-3-carbinol was excluded from evaluation of brain lesions.

Table B-5. Historical Incidence of Adrenal Medulla Neoplasms in Control Female Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Benign Pheochromocytoma	Malignant Pheochromocytoma	Complex Pheochromocytoma	Benign, Malignant, or Complex Pheochromocytoma
Historical Incidence: All Studies				
p-Chloro-α,α,α-trifluorotoluene (January 2011)	0/49	0/49	0/49	0/49
2-Hydroxy-4-methoxybenzophenone (November 2010)	3/50	2/50	0/50	5/50
Indole-3-carbinol (March 2007)	0/50	0/50	0/50	0/50
Radiofrequency radiation (September 2012)	1/86	0/86	0/86	1/86
Overall Historical Incidence				
Total (%)	4/235 (1.7%)	2/235 (0.9%)	0/235	6/235 (2.6%)
Mean ± standard deviation	1.8% ± 2.9%	1.0% ± 2.0%	–	2.8% ± 4.8%
Range	0%–6%	0%–4%	–	0%–10%

[a]Data as of November 2017.

Table B-6. Historical Incidence of Pituitary Gland (Pars Distalis) Adenoma in Control Female Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Incidence in Controls
Historical Incidence: All Studies	
p-Chloro-α,α,α-trifluorotoluene (January 2011)	18/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	19/50
Indole-3-carbinol (March 2007)	18/50
Radiofrequency radiation (September 2012)	43/90
Overall Historical Incidence	
Total (%)	98/240 (40.8%)
Mean ± standard deviation	39.4% ± 5.6%
Range	36%–48%

[a]Data as of November 2017.

Table B-7. Summary of the Incidence of Non-neoplastic Lesions in Female Rats Exposed to GSM-modulated Cell Phone RFR for Two Years[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
Fourteen-week interim evaluation	15	15	15	15
Early deaths				
Accidental death	1			
Moribund	30	25	31	22
Natural deaths	11	10	11	11
Survivors				
Died last week of study	1	3	1	–
Terminal euthanasia	47	52	47	57
Animals examined microscopically	100	100	100	100
Fourteen-week Interim Evaluation				
Alimentary System				
Esophagus	(10)	(10)	(10)	(10)
Intestine large, cecum	(10)	(10)	(10)	(10)
Intestine large colon	(10)	(10)	(10)	(10)
Intestine large, rectum	(10)	(10)	(10)	(10)
Lymphoid tissue, hyperplasia	1 (10%)	1 (10%)	–	–
Intestine small, duodenum	(10)	(10)	(10)	(10)
Intestine small, ileum	(10)	(10)	(10)	(10)
Intestine small, jejunum	(10)	(10)	(10)	(10)
Liver	(10)	(10)	(10)	(10)
Infiltration cellular, mixed cell	1 (10%)	1 (10%)	2 (20%)	1 (10%)
Pancreas	(10)	(10)	(10)	(10)
Salivary glands	(10)	(10)	(10)	(10)
Degeneration, cystic	–	–	–	1 (10%)
Stomach, forestomach	(10)	(10)	(10)	(10)
Stomach, glandular	(10)	(10)	(10)	(10)
Cardiovascular System				
Aorta	(10)	(10)	(10)	(10)
Heart	(10)	(10)	(10)	(10)
Cardiomyopathy	–	2 (20%)	1 (10%)	2 (20%)
Ventricle right, cardiomyopathy	–	–	1 (10%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Endocrine System				
Adrenal cortex	(10)	(10)	(10)	(10)
Adrenal medulla	(10)	(10)	(10)	(10)
Islets, pancreatic	(10)	(10)	(10)	(10)
Parathyroid gland	(10)	(8)	(9)	(9)
Pituitary gland	(10)	(10)	(10)	(10)
Pars distalis, cyst	2 (20%)	–	–	–
Pars intermedia, cyst	1 (10%)	1 (10%)	–	–
Thyroid gland	(10)	(10)	(10)	(10)
Genital System				
Clitoral gland	(10)	(10)	(10)	(10)
Inflammation, chronic active	4 (40%)	5 (50%)	6 (60%)	4 (40%)
Ovary	(10)	(10)	(10)	(10)
Cyst	–	–	–	1 (10%)
Follicle, cyst	1 (10%)	–	–	–
Uterus	(10)	(10)	(10)	(10)
Hematopoietic System				
Bone marrow	(10)	(10)	(10)	(10)
Lymph node, mandibular	(10)	(10)	(10)	(10)
Hemorrhage	–	2 (20%)		–
Hyperplasia, lymphocyte	–	–	–	2 (20%)
Proliferation, plasma cell	–	–	–	3 (30%)
Lymph node, mesenteric	(10)	(10)	(10)	(10)
Spleen	(10)	(10)	(10)	(10)
Thymus	(10)	(10)	(10)	(10)
Hemorrhage	1 (10%)		1 (10%)	–
Musculoskeletal System				
Bone	(10)	(10)	(10)	(10)
Skeletal muscle	(10)	(10)	(10)	(10)
Infiltration cellular, histiocyte	–	–	1 (10%)	–
Respiratory System				
Lung	(10)	(10)	(10)	(10)
Congestion	–	–	1 (10%)	–
Inflammation, chronic active	–	–	1 (10%)	–
Alveolus, infiltration cellular, histiocyte	2 (20%)	–	–	–
Epithelium alveolus, hyperplasia	–	–	1 (10%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Nose	(10)	(10)	(10)	(10)
Trachea	(10)	(10)	(10)	(10)
Special Senses System				
Eye	(10)	(10)	(10)	(10)
Harderian gland	(10)	(10)	(10)	(10)
Inflammation cellular, lymphocyte	1 (10%)	–	–	–
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Nephropathy, chronic progressive	3 (30%)	4 (40%)	3 (30%)	2 (20%)
Urinary bladder	(10)	(10)	(10)	(10)
Systems Examined with No Lesions Observed				
General Body System				
Integumentary System				
Nervous System				
Two-year Study				
Alimentary System				
Esophagus	(90)	(90)	(90)	(90)
Dilation	–	–	1 (1%)	–
Intestine large, cecum	(84)	(83)	(83)	(84)
Serosa, inflammation, acute	–	1 (1%)	–	–
Intestine large, colon	(89)	(88)	(89)	(89)
Intestine large, rectum	(90)	(89)	(89)	(89)
Hyperplasia, lymphocyte	–	1 (1%)	–	1 (1%)
Inflammation, acute	–	–	–	1 (1%)
Necrosis	–	–	–	1 (1%)
Epithelium, hyperplasia	–	–	–	1 (1%)
Epithelium, metaplasia, squamous	–	–	1 (1%)	–
Intestine small, duodenum	(88)	(85)	(83)	(85)
Ectopic tissue	–	–	–	1 (1%)
Ulcer	–	1 (1%)	–	–
Intestine small, ileum	(86)	(82)	(81)	(83)
Hyperplasia, lymphocyte	1 (1%)	–	–	–
Necrosis, lymphoid	–	–	–	1 (1%)
Serosa, inflammation, acute	–	1 (1%)	–	–
Intestine small, jejunum	(83)	(82)	(81)	(84)
Liver	(90)	(90)	(90)	(90)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Angiectasis	6 (7%)	4 (4%)	6 (7%)	6 (7%)
Basophilic focus	11 (12%)	17 (19%)	11 (12%)	8 (9%)
Clear cell focus	2 (2%)	3 (3%)	6 (7%)	6 (7%)
Congestion	–	1 (1%)	2 (2%)	–
Eosinophilic focus	9 (10%)	26 (29%)	23 (26%)	23 (26%)
Extramedullary hematopoiesis	15 (17%)	19 (21%)	17 (19%)	12 (13%)
Fibrosis	–	1 (1%)	–	–
Hepatodiaphragmatic nodule	1 (1%)	–	1 (1%)	–
Infiltration cellular, histiocyte	–	–	2 (2%)	–
Infiltration cellular, mixed cell	1 (1%)	4 (4%)	1 (1%)	2 (2%)
Infiltration cellular, mononuclear cell	–	1 (1%)	–	–
Inflammation, acute	–	1 (1%)	–	1 (1%)
Inflammation, chronic	–	1 (1%)	–	–
Inflammation, chronic active	–	–	2 (2%)	–
Mixed cell focus	29 (32%)	23 (26%)	33 (37%)	28 (31%)
Pigment	–	1 (1%)	–	–
Bile duct, cyst	11 (12%)	6 (7%)	5 (6%)	8 (9%)
Bile duct, fibrosis	1 (1%)	–	–	–
Bile duct, hyperplasia	9 (10%)	5 (6%)	10 (11%)	9 (10%)
Bile duct, inflammation, chronic active	–	–	1 (1%)	–
Centrilobular, hepatocyte, necrosis	–	3 (3%)	–	2 (2%)
Centrilobular, hepatocyte, vacuolation, cytoplasmic	–	1 (1%)	–	–
Hepatocyte, degeneration	–	–	1 (1%)	–
Hepatocyte, hypertrophy	2 (2%)	5 (6%)	2 (2%)	6 (7%)
Hepatocyte, increased mitoses	2 (2%)	–	–	–
Hepatocyte, necrosis	4 (4%)	2 (2%)	5 (6%)	8 (9%)
Hepatocyte, vacuolation, cytoplasmic	1 (1%)	1 (1%)	1 (1%)	3 (3%)
Kupffer cell, hyperplasia	3 (3%)	–	–	–
Kupffer cell, hypertrophy	2 (2%)	–	–	–
Periductal, cholangiofibrosis	1 (1%)	–	–	1 (1%)
Serosa, inflammation, suppurative	–	1 (1%)	–	–
Serosa, inflammation, chronic active	1 (1%)	–	–	–
Sinusoid, dilation	–	1 (1%)	1 (1%)	–
Mesentery	(4)	(5)	(5)	(5)
Inflammation, chronic active	1 (25%)	–	1 (20%)	–
Necrosis	1 (25%)	3 (60%)	2 (40%)	3 (60%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Artery, inflammation, chronic active	–	–	–	1 (20%)
Oral mucosa	(1)	(0)	(0)	(0)
Pancreas	(90)	(90)	(90)	(87)
Ectopic liver	1 (1%)	–	–	–
Inflammation, acute	–	1 (1%)	–	–
Inflammation, chronic active	1 (1%)	–	2 (2%)	–
Acinus, atrophy	5 (6%)	4 (4%)	4 (4%)	5 (6%)
Acinus, hyperplasia	1 (1%)	2 (2%)	5 (6%)	2 (2%)
Artery, inflammation, chronic active	–	2 (2%)	1 (1%)	1 (1%)
Periductal, cholangiofibrosis	–	–	7 (8%)	4 (5%)
Salivary glands	(90)	(89)	(90)	(90)
Duct, parotid gland, dilation	1 (1%)	–	–	1 (1%)
Parotid gland, atrophy	4 (4%)	11 (12%)	8 (9%)	4 (4%)
Parotid gland, inflammation, acute	–	–	–	1 (1%)
Parotid gland, vacuolation, cytoplasmic	–	–	–	1 (1%)
Sublingual gland, atrophy	–	–	–	2 (2%)
Sublingual gland, metaplasia	–	–	1 (1%)	–
Submandibular gland, atrophy	–	1 (1%)	1 (1%)	–
Stomach, forestomach	(90)	(90)	(90)	(90)
Edema	2 (2%)	2 (2%)	1 (1%)	–
Erosion	2 (2%)	–	–	–
Fibrosis	1 (1%)	–	–	–
Inflammation, acute	–	1 (1%)	–	–
Inflammation, chronic active	4 (4%)	5 (6%)	–	–
Ulcer	1 (1%)	7 (8%)	2 (2%)	2 (2%)
Epithelium, hyperplasia	10 (11%)	14 (16%)	8 (9%)	8 (9%)
Epithelium, hyperplasia, basal cell	1 (1%)	–	–	1 (1%)
Stomach, glandular	(90)	(89)	(90)	(89)
Erosion	1 (1%)	1 (1%)	–	–
Tongue	(1)	(0)	(0)	(0)
Tooth	(0)	(0)	(1)	(0)
Dysplasia	–	–	1 (100%)	–
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Dilation	–	–	1 (1%)	–
Mineral	–	2 (2%)	1 (1%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Heart	(90)	(90)	(90)	(90)
Cardiomyopathy	40 (44%)	30 (33%)	39 (43%)	27 (30%)
Atrium, myocardium, hypertrophy	–	1 (1%)	–	–
Myocardium, hypertrophy	–	1 (1%)	–	–
Myocardium, mineral	–	1 (1%)	–	–
Myocardium, necrosis	–	–	–	1 (1%)
Myocardium, Schwann cell, hyperplasia	–	–	–	1 (1%)
Myocardium, ventricle right, degeneration	–	–	1 (1%)	–
Vein, mineral	–	1 (1%)	–	–
Ventricle right, cardiomyopathy	4 (4%)	9 (10%)	14 (16%)	15 (17%)
Endocrine System				
Adrenal cortex	(90)	(90)	(89)	(90)
Accessory adrenal cortical nodule	5(6%)	7 (8%)	6 (7%)	6 (7%)
Angiectasis	–	1 (1%)	–	–
Atrophy	1 (1%)	1 (1%)	–	–
Cyst	–	–	1 (1%)	–
Degeneration, cystic	22 (24%)	26 (29%)	36 (40%)	29 (32%)
Extramedullary hematopoiesis	–	1 (1%)	1 (1%)	–
Hemorrhage	–	1 (1%)	1 (1%)	–
Hyperplasia	14 (16%)	26 (29%)	40 (45%)	26 (29%)
Hypertrophy	52 (58%)	54 (60%)	51 (57%)	56 (62%)
Mineral	–	–	1 (1%)	–
Necrosis	2 (2%)	4 (4%)	1 (1%)	2 (2%)
Pigment	1 (1%)	–	–	–
Thrombus	–	3 (3%)	1 (1%)	–
Vacuolation, cytoplasmic	18 (20%)	21 (23%)	11 (12%)	8 (9%)
Adrenal medulla	(86)	(90)	(90)	(86)
Hyperplasia	13 (15%)	19 (21%)	14 (16%)	25 (29%)
Necrosis	1 (1%)	–	–	–
Islets, pancreatic	(90)	(89)	(90)	(87)
Ectopic tissue	–	–	1 (1%)	–
Hyperplasia	15 (17%)	6 (7%)	11 (12%)	12 (14%)
Parathyroid gland	(87)	(79)	(82)	(79)
Cyst	–	1 (1%)	–	–
Fibrosis	13 (15%)	4 (5%)	9 (11%)	6 (8%)
Hyperplasia	–	1 (1%)	2 (2%)	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Hyperplasia, focal	3 (3%)	–	2 (2%)	–
Hypertrophy	–	1 (1%)	–	–
Pituitary gland	(90)	(90)	(90)	(90)
Cyst	1 (1%)	–	–	–
Pars distalis, angiectasis	2 (2%)	–	–	–
Pars distalis, atrophy	–	1 (1%)	–	–
Pars distalis, cyst	7 (8%)	3 (3%)	4 (4%)	4 (4%)
Pars distalis, hyperplasia	20 (22%)	26 (29%)	22 (24%)	22 (24%)
Pars distalis, vacuolation, cytoplasmic	–	–	1 (1%)	–
Pars intermedia, cyst	3 (3%)	3 (3%)	2 (2%)	2 (2%)
Pars intermedia, hyperplasia	1 (1%)	–	1 (1%)	–
Pars intermedia, vacuolation, cytoplasmic	–	–	1 (1%)	–
Pars nervosa, cyst	–	1 (1%)	–	–
Thyroid gland	(90)	(88)	(90)	(88)
C-cell, hyperplasia	28 (31%)	49 (56%)	45 (50%)	43 (49%)
Follicle, cyst	1 (1%)	2 (2%)	–	1 (1%)
General Body System				
Tissue NOS	(8)	(10)	(8)	(10)
Inflammation, chronic active	1 (13%)	–	–	–
Abdominal, necrosis	–	1 (10%)	–	–
Fat, necrosis	6 (75%)	8 (80%)	7 (88%)	9 (90%)
Genital System				
Clitoral gland	(87)	(85)	(86)	(87)
Hyperplasia, focal	–	3 (4%)	–	–
Inflammation, suppurative	1 (1%)	1 (1%)	–	–
Inflammation, granulomatous	–	1 (1%)	–	1 (1%)
Inflammation, acute	–	–	1 (1%)	1 (1%)
Inflammation, chronic active	28 (32%)	24 (28%)	32 (37%)	40 (46%)
Metaplasia, squamous	–	1 (1%)	–	–
Duct, dilation	47 (54%)	47 (55%)	44 (51%)	40 (46%)
Ovary	(90)	(90)	(90)	(90)
Atrophy	72 (80%)	63 (70%)	66 (73%)	71 (79%)
Congestion	1 (1%)	–	–	–
Cyst	22 (24%)	24 (27%)	23 (26%)	27 (30%)
Fibrosis	–	1 (1%)	–	–
Inflammation, suppurative	–	1 (1%)	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Inflammation, chronic	–	–	–	1 (1%)
Inflammation, chronic active	–	–	–	2 (2%)
Necrosis	–	–	–	1 (1%)
Bursa, dilation	4 (4%)	5 (6%)	6 (7%)	6 (7%)
Interstitial cell, hyperplasia	–	2 (2%)	–	–
Periovarian tissue, cyst	–	–	1 (1%)	–
Periovarian tissue, hemorrhage	–	–	1 (1%)	–
Periovarian tissue, inflammation, chronic active	–	–	–	1 (1%)
Rete ovarii, hyperplasia	15 (17%)	25 (28%)	13 (14%)	12 (13%)
Oviduct	(1)	(0)	(0)	(0)
Cyst	1 (100%)	–	–	–
Uterus	(90)	(89)	(90)	(90)
Adenomyosis	–	–	–	1 (1%)
Angiectasis	1 (1%)	1 (1%)	–	–
Cyst	5 (6%)	3 (3%)	11 (12%)	7 (8%)
Dilation	8 (9%)	7 (8%)	12 (13%)	4 (4%)
Fibrosis	1 (1%)	1 (1%)	1 (1%)	–
Hemorrhage	–	3 (3%)	4 (4%)	1 (1%)
Hyperplasia, stromal	–	3 (3%)	1 (1%)	4 (4%)
Inflammation cellular, mononuclear cell	–	–	1 (1%)	–
Inflammation, suppurative	4 (4%)	11 (12%)	6 (7%)	10 (11%)
Inflammation, acute	1 (1%)	–	–	2 (2%)
Inflammation, chronic active	–	2 (2%)	6 (7%)	1 (1%)
Pigment	–	1 (1%)	–	1 (1%)
Thrombus	1 (1%)	2 (2%)	–	1 (1%)
Artery, inflammation, chronic active	–	–	–	1 (1%)
Cervix, cyst	–	–	1 (1%)	–
Cervix, hyperplasia, stromal	2 (2%)	–	–	1 (1%)
Cervix, serosa, fibrosis	1 (1%)	–	–	–
Endometrium, hyperplasia, cystic	37 (41%)	33 (37%)	28 (31%)	39 (43%)
Epithelium, metaplasia, squamous	48 (53%)	38 (43%)	39 (43%)	45 (50%)
Serosa, fibrosis	–	–	1 (1%)	–
Serosa, inflammation, suppurative	–	1 (1%)	–	–
Vein, thrombus	–	1 (1%)	–	
Vagina	(2)	(3)	(1)	(1)
Exudate	–	1 (33%)	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Inflammation, chronic active	–	–	–	1 (100%)
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Hypercellularity	56 (62%)	57 (63%)	55 (61%)	56 (62%)
Myelofibrosis	–	–	–	1 (1%)
Lymph node	(13)	(14)	(21)	(14)
Axillary, hyperplasia, lymphocyte	–	–	–	1 (7%)
Axillary, proliferation, plasma cell	1 (8%)	–	–	1 (7%)
Deep cervical, fibrosis	–	–	–	1 (7%)
Deep cervical, inflammation, chronic active	–	–	–	1 (7%)
Iliac, congestion	–	–	–	1 (7%)
Iliac, erythrophagocytosis	3 (23%)	1 (7%)	3 (14%)	2 (14%)
Iliac, hyperplasia, lymphocyte	1 (8%)	–	2 (10%)	2 (14%)
Iliac, infiltration cellular, histiocyte	–	–	1 (5%)	–
Iliac, inflammation, acute	1 (8%)	–	–	–
Iliac, pigment	1 (8%)	–	1 (5%)	1 (7%)
Iliac, proliferation, plasma cell	6 (46%)	1 (7%)	2 (10%)	–
Iliac, lymphatic sinus, ectasia	–	1 (7%)	3 (14%)	–
Inguinal, erythrophagocytosis	1 (8%)	–	–	–
Inguinal, hyperplasia, lymphocyte	–	1 (7%)	–	–
Inguinal, infiltration cellular, plasma cell	–	1 (7%)	–	–
Inguinal, pigment	–	–	1 (5%)	–
Inguinal, proliferation, plasma cell	1 (8%)	–	–	–
Inguinal, lymphatic sinus, ectasia	1 (8%)	–	–	–
Lumbar, erythrophagocytosis	1 (8%)	1 (7%)	1 (5%)	1 (7%)
Lumbar, hyperplasia, lymphocyte	–	2 (14%)	–	–
Lumbar, infiltration cellular, histiocyte	–	–	1 (5%)	–
Lumbar, inflammation, chronic active	–	1 (7%)	–	–
Lumbar, proliferation, plasma cell	–	2 (14%)	1 (5%)	–
Lumbar, lymphatic sinus, ectasia	–	1 (7%)	–	–
Lymphatic sinus, mediastinal, ectasia	–	–	1 (5%)	–
Lymphatic sinus, renal, ectasia	–	1 (7%)	–	–
Mediastinal, congestion	1 (8%)	–	1 (5%)	–
Mediastinal, erythrophagocytosis	–	3 (21%)	5 (24%)	3 (21%)
Mediastinal, hyperplasia, lymphocyte	–	–	2 (10%)	1 (7%)
Mediastinal, proliferation, plasma cell	1 (8%)	–	3 (14%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pancreatic, erythrophagocytosis	1 (8%)	1 (7%)	1 (5%)	
Renal, erythrophagocytosis	–	2 (14%)	1 (5%)	2 (14%)
Renal, inflammation, chronic active	–	1 (7%)	–	–
Lymph node, mandibular	(90)	(89)	(89)	(90)
Congestion	–	–	–	1 (1%)
Erythrophagocytosis	–	2 (2%)	4 (4%)	3 (3%)
Hemorrhage	1 (1%)	–	–	–
Hyperplasia, lymphocyte	46 (51%)	40 (45%)	44 (49%)	51 (57%)
Hyperplasia, reticulum cell	–	–	–	1 (1%)
Infiltration cellular, histiocyte	–	1 (1%)	–	–
Inflammation, chronic active	–	–	1 (1%)	–
Proliferation, plasma cell	68 (76%)	57 (64%)	65 (73%)	56 (62%)
Lymphatic sinus, ectasia	1 (1%)	3 (3%)	7 (8%)	1 (1%)
Lymph node, mesenteric	(90)	(90)	(90)	(90)
Atrophy	1 (1%)	1 (1%)	–	–
Erythrophagocytosis	1 (1%)	3 (3%)	3 (3%)	5 (6%)
Hemorrhage	–	1 (1%)	–	–
Hyperplasia, lymphocyte	–	1 (1%)	–	1 (1%)
Infiltration cellular, histiocyte	2 (2%)	3 (3%)	2 (2%)	1 (1%)
Necrosis, lymphocyte	–	–	–	1 (1%)
Pigment	–	–	–	1 (1%)
Proliferation, plasma cell	–	1 (1%)	1 (1%)	–
Lymphatic sinus, ectasia	–	1 (1%)	–	–
Spleen	(90)	(90)	(90)	(90)
Accessory spleen	–	–	1 (1%)	–
Congestion	–	2 (2%)	1 (1%)	–
Developmental malformation	–	–	1 (1%)	–
Extramedullary hematopoiesis	80 (89%)	77 (86%)	78 (87%)	78 (87%)
Hemorrhage	–	–	1 (1%)	–
Hyperplasia, stromal	1 (1%)	–	–	–
Pigment	74 (82%)	40 (44%)	47 (52%)	46 (51%)
Red pulp, atrophy	7 (8%)	5 (6%)	2 (2%)	1 (1%)
Red pulp, hyperplasia	–	2 (2%)	–	–
White pulp, atrophy	3 (3%)	6 (7%)	6 (7%)	2 (2%)
Thymus	(87)	(86)	(88)	(86)
Atrophy	75 (86%)	70 (81%)	62 (70%)	61 (71%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Cyst	39 (45%)	30 (35%)	33 (38%)	28 (33%)
Ectopic parathyroid gland	1 (1%)	1 (1%)	2 (2%)	–
Ectopic thyroid	–	1 (1%)	–	1 (1%)
Hemorrhage	2 (2%)	2 (2%)	2 (2%)	3 (3%)
Hyperplasia, epithelial	55 (63%)	19 (22%)	19 (22%)	20 (23%)
Necrosis, lymphocyte	–	–	–	1 (1%)
Integumentary System				
Mammary gland	(90)	(89)	(89)	(90)
Galactocele	24 (27%)	18 (20%)	14 (16%)	10 (11%)
Hyperplasia	49 (54%)	41 (46%)	51 (57%)	28 (31%)
Hyperplasia, atypical	–	–	–	3 (3%)
Inflammation, granulomatous	–	–	1 (1%)	–
Duct, dilation	56 (62%)	52 (58%)	55 (62%)	58 (64%)
Lymphatic, dilation	–	–	1 (1%)	–
Skin	(90)	(90)	(90)	(90)
Cyst epithelial inclusion	1 (1%)	2 (2%)	–	–
Inflammation, acute	–	1 (1%)	–	–
Inflammation, chronic active	1 (1%)	–	–	–
Ulcer	–	–	1 (1%)	–
Dermis, fibrosis	–	1 (1%)	–	–
Epidermis, hyperplasia	2 (2%)	–	–	–
Subcutaneous tissue, edema	–	–	1 (1%)	1 (1%)
Subcutaneous tissue, inflammation, chronic active	–	–	1 (1%)	1 (1%)
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Fibrosis	–	1 (1%)	1 (1%)	–
Fibrous osteodystrophy	–	1 (1%)	2 (2%)	–
Increased bone	–	1 (1%)	1 (1%)	–
Cranium, fracture	1 (1%)	–	–	–
Mandible, fracture	1 (1%)	–	–	–
Maxilla, fracture	1 (1%)	–	–	–
Vertebra, increased bone	–	1 (1%)	–	–
Vertebra, inflammation, chronic active	–	1 (1%)	–	–
Skeletal muscle	(90)	(90)	(90)	(90)
Degeneration	3 (3%)	7 (8%)	4 (4%)	3 (3%)
Mineral	–	–	–	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Nervous System				
Brain	(90)	(90)	(90)	(90)
Compression	26 (29%)	16 (18%)	18 (20%)	11 (12%)
Congestion	1 (1%)	1 (1%)	–	–
Cyst	–	–	–	1 (1%)
Edema	2 (2%)	–	–	2 (2%)
Hemorrhage	–	1 (1%)	–	1 (1%)
Mineral	–	–	1 (1%)	–
Necrosis	–	–	–	1 (1%)
Pigment	–	1 (1%)	–	–
Cerebellum, hemorrhage	–	1 (1%)	–	–
Glial cell, hyperplasia	–	–	1 (1%)	–
Hypothalamus, cyst	–	1 (1%)	–	–
Meninges, hyperplasia	1 (1%)	–	–	–
Meninges, hyperplasia, granular cell	1 (1%)	–	1 (1%)	–
Pineal gland, mineral	1 (1%)	1 (1%)	3 (3%)	1 (1%)
Pineal gland, vacuolation, cytoplasmic	1 (1%)	3 (3%)	2 (2%)	1 (1%)
Nerve trigeminal	(84)	(88)	(89)	(90)
Degeneration	64 (76%)	71 (81%)	65 (73%)	74 (82%)
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Degeneration	80 (89%)	84 (93%)	81 (90%)	84 (93%)
Infiltration cellular, mixed cell	1 (1%)	–	–	–
Peripheral nerve, tibial	(90)	(90)	(90)	(89)
Degeneration	77 (86%)	83 (92%)	80 (89%)	80 (90%)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Degeneration	24 (27%)	29 (32%)	43 (48%)	23 (26%)
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Cyst	–	1 (1%)	–	1 (1%)
Degeneration	10 (11%)	7 (8%)	13 (14%)	12 (13%)
Nerve, degeneration	74 (82%)	81 (90%)	70 (78%)	78 (87%)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Degeneration	59 (66%)	64 (71%)	61 (68%)	65 (72%)
Trigeminal ganglion	(81)	(79)	(80)	(79)
Degeneration	33 (41%)	31 (39%)	28 (35%)	17 (22%)
Respiratory System				
Lung	(90)	(90)	(90)	(90)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Congestion	3 (3%)	5 (6%)	5 (6%)	3 (3%)
Foreign body	–	2 (2%)	–	–
Hemorrhage	1 (1%)	1 (1%)	4 (4%)	2 (2%)
Inflammation, suppurative	2 (2%)	1 (1%)	–	–
Inflammation, granulomatous	1 (1%)	2 (2%)	3 (3%)	–
Inflammation, chronic active	6 (7%)	8 (9%)	9 (10%)	8 (9%)
Pigment	–	–	–	1 (1%)
Alveolar epithelium, metaplasia, squamous	–	–	1 (1%)	1 (1%)
Alveolus, infiltration cellular, histiocyte	71 (79%)	75 (83%)	83 (92%)	82 (91%)
Alveolus, pigment	–	–	2 (2%)	1 (1%)
Artery, inflammation, chronic active	1 (1%)	–	–	–
Bronchiole, hyperplasia	–	–	1 (1%)	–
Epithelium alveolus, hyperplasia	2 (2%)	2 (2%)	6 (7%)	2 (2%)
Interstitium, fibrosis	–	1 (1%)	–	–
Nose	(90)	(90)	(90)	(90)
Foreign body	–	–	1 (1%)	–
Inflammation, suppurative	1 (1%)	2 (2%)	3 (3%)	1 (1%)
Inflammation, acute	–	–	1 (1%)	1 (1%)
Inflammation, chronic active	–	–	1 (1%)	–
Nasopharyngeal duct, inflammation, chronic active	–	1 (1%)	–	–
Nerve, olfactory epithelium, degeneration	–	1 (1%)	–	–
Olfactory epithelium, accumulation, hyaline droplet	89 (99%)	86 (96%)	88 (98%)	87 (97%)
Olfactory epithelium, atrophy	–	1 (1%)	1 (1%)	–
Olfactory epithelium, degeneration	–	1 (1%)	–	–
Olfactory epithelium, metaplasia, respiratory	1 (1%)	–	1 (1%)	–
Respiratory epithelium, accumulation, hyaline droplet	12 (13%)	8 (9%)	10 (11%)	10 (11%)
Respiratory epithelium, hyperplasia	–	–	1 (1%)	–
Respiratory epithelium, metaplasia, squamous	–	–	2 (2%)	–
Trachea	(89)	(90)	(89)	(87)
Inflammation, chronic active	1 (1%)	–	–	–
Artery, inflammation, chronic active	–	–	–	1 (1%)
Epithelium, hyperplasia	–	1 (1%)	–	–
Glands, cyst	1 (1%)	–	–	–
Special Senses System				
Eye	(88)	(85)	(87)	(87)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Cornea, inflammation, acute	1 (1%)	–	–	–
Cornea, epithelium, hyperplasia	1 (1%)	–	–	–
Lens, cataract	1 (1%)	–	–	–
Retina, atrophy	18 (20%)	15 (18%)	16 (18%)	13 (15%)
Retina, dysplasia	1 (1%)	–	–	–
Sclera, inflammation, acute	–	–	1 (1%)	–
Harderian gland	(90)	(90)	(90)	(90)
Atrophy	13 (14%)	12 (13%)	15 (17%)	24 (27%)
Hyperplasia	–	–	1 (1%)	–
Infiltration cellular, lymphocyte	2 (2%)	–	–	–
Inflammation, granulomatous	7 (8%)	9 (10%)	9 (10%)	10 (11%)
Inflammation, acute	–	1 (1%)	–	–
Inflammation, chronic	7 (8%)	4 (4%)	1 (1%)	2 (2%)
Inflammation, chronic active	1 (1%)	1 (1%)	2 (2%)	2 (2%)
Zymbal's gland	(0)	(0)	(0)	(1)
Urinary System				
Kidney	(90)	(90)	(90)	(89)
Ectopic tissue	–	1 (1%)	–	–
Infarct	–	1 (1%)	–	–
Inflammation, granulomatous	–	–	–	1 (1%)
Inflammation, acute	1 (1%)	–	–	–
Inflammation, chronic active	–	1 (1%)	1 (1%)	–
Mineral	–	–	–	1 (1%)
Necrosis	–	1 (1%)	–	–
Nephropathy, chronic progressive	74 (82%)	61 (68%)	68 (76%)	59 (66%)
Artery, inflammation, chronic active	1 (1%)	–	–	1 (1%)
Pelvis, dilation	3 (3%)	2 (2%)	1 (1%)	–
Renal tubule, accumulation, hyaline droplet	–	2 (2%)	1 (1%)	–
Renal tubule, cyst	3 (3%)	2 (2%)	1 (1%)	1 (1%)
Renal tubule, hyperplasia, atypical	–	1 (1%)	–	1 (1%)
Renal tubule, hypertrophy	–	1 (1%)	–	–
Renal tubule, necrosis	–	2 (2%)	–	–
Renal tubule, pigment	–	1 (1%)	–	–
Urothelium, hyperplasia	–	–	–	1 (1%)
Urinary bladder	(88)	(88)	(90)	(87)
Dilation	1 (1%)	–	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Edema	–	1 (1%)	–	–
Hemorrhage	–	1 (1%)	1 (1%)	–
Infiltration cellular, histiocyte	–	–	–	1 (1%)
Infiltration cellular, mononuclear cell	–	–	–	1 (1%)
Inflammation, acute	3 (3%)	–	1 (1%)	–
Necrosis	1 (1%)	–	–	–
Urothelium, hyperplasia	1 (1%)	1 (1%)	–	–

[a]Number of animals examined microscopically at the site and the number of animals with lesion.

Appendix C. Summary of Lesions in Male Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

Tables

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Table C-1. Summary of the Incidence of Neoplasms in Male Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
Fourteen-week interim evaluation	15	15	15	15
Early deaths				
Accidental death	1			
Moribund	44	24	13	6
Natural deaths	20	23	21	41
Survivors				
Died last week of study	–	–	1	–
Terminal euthanasia	25	43	55	43
Animals examined microscopically	100	100	100	100
Systems Examined at 14 Weeks with No Neoplasms Observed				
Alimentary System				
Cardiovascular System				
Endocrine System				
General Body System				
Genital System				
Hematopoietic System				
Integumentary System				
Musculoskeletal System				
Nervous System				
Respiratory System				
Special Senses System				
Urinary System				
Two-year Study				
Alimentary System				
Esophagus	(90)	(90)	(90)	(90)
Intestine large, cecum	(75)	(76)	(74)	(68)
Intestine large, colon	(81)	(83)	(82)	(76)
Intestine large, rectum	(83)	(81)	(80)	(76)
Serosa, sarcoma, metastatic, skeletal muscle	–	–	–	1(1%)
Intestine small, duodenum	(81)	(84)	(83)	(66)
Adenocarcinoma	–	–	1 (1%)	–
Osteosarcoma	–	–	1 (1%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Serosa, sarcoma, metastatic, skeletal muscle	–	–	–	1 (2%)
Intestine small, ileum	(78)	(76)	(77)	(63)
Intestine small, jejunum	(73)	(73)	(75)	(62)
Adenocarcinoma	2 (3%)	1 (1%)	–	–
Liver	(90)	(90)	(89)	(88)
Hepatocellular adenoma	–	2 (2%)	4 (4%)	–
Hepatocellular carcinoma	–	–	1 (1%)	1 (1%)
Sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Mesentery	(39)	(19)	(17)	(6)
Oral mucosa	(0)	(1)	(1)	(0)
Squamous cell carcinoma	–	–	1 (100%)	–
Pancreas	(90)	(88)	(87)	(78)
Adenoma	13 (14%)	16 (18%)	19 (22%)	5 (6%)
Adenoma, multiple	5 (6%)	6 (7%)	7 (8%)	2 (3%)
Carcinoma	1 (1%)	1 (1%)	2 (2%)	–
Pheochromocytoma malignant, metastatic, adrenal medulla	–	1 (1%)	–	–
Salivary glands	(90)	(90)	(90)	(86)
Parotid gland, adenoma	–	–	1 (1%)	–
Sublingual gland, schwannoma malignant, metastatic, uncertain primary site	1 (1%)	–	–	–
Stomach, forestomach	(90)	(90)	(89)	(90)
Squamous cell carcinoma	–	–	1 (1%)	–
Squamous cell papilloma	1 (1%)	–	–	–
Stomach, glandular	(86)	(86)	(85)	(78)
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Blood vessel	(1)	(2)	(1)	(0)
Heart	(90)	(90)	(90)	(90)
Atrium, schwannoma malignant	–	–	–	1 (1%)
Endocardium, schwannoma malignant	–	1 (1%)	–	4 (4%)
Myocardium, schwannoma malignant	–	1 (1%)	3 (3%)	1 (1%)
Pericardium, schwannoma malignant, metastatic, thymus	–	–	1 (1%)	–
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(89)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adenoma	1 (1%)	3 (3%)	–	–
Carcinoma	–	3 (3%)	–	2 (2%)
Adrenal medulla	(88)	(90)	(90)	(90)
Pheochromocytoma benign	8 (9%)	17 (19%)	19 (21%)	12 (13%)
Pheochromocytoma benign, multiple	1 (1%)	1 (1%)	2 (2%)	–
Pheochromocytoma complex	1 (1%)	–	–	–
Pheochromocytoma malignant	1 (1%)	3 (3%)	3 (3%)	1 (1%)
Bilateral, pheochromocytoma benign	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Islets, pancreatic	(90)	(88)	(87)	(79)
Adenoma	5 (6%)	11 (13%)	7 (8%)	7 (9%)
Adenoma, multiple	–	–	1 (1%)	–
Carcinoma	8 (9%)	6 (7%)	13 (15%)	5 (6%)
Carcinoma, multiple	–	–	–	1 (1%)
Parathyroid gland	(83)	(83)	(83)	(82)
Adenoma	1 (1%)	1 (1%)	2 (2%)	1 (1%)
Pituitary gland	(89)	(90)	(90)	(90)
Schwannoma malignant, metastatic, tissue NOS	–	1 (1%)	–	1 (1%)
Schwannoma malignant, metastatic, uncertain primary site	–	1 (1%)	–	–
Pars distalis, adenoma	17 (19%)	25 (28%)	33 (37%)	11 (12%)
Pars distalis, adenoma, multiple	–	–	1 (1%)	2 (2%)
Thyroid gland	(89)	(87)	(86)	(85)
Bilateral, C-cell, adenoma	–	2 (2%)	–	–
Bilateral, C-cell, carcinoma	–	–	1 (1%)	–
C-cell, adenoma	8 (9%)	12 (14%)	14 (16%)	11 (13%)
C-cell, carcinoma	2 (2%)	–	1 (1%)	4 (5%)
Follicular cell, adenoma	–	–	–	1 (1%)
General Body System				
Tissue NOS	(3)	(1)	(3)	(3)
Schwannoma malignant	–	1 (100%)	–	1 (33%)
Fat, schwannoma malignant	–	–	–	1 (33%)
Mediastinum, chemodectoma benign	–	–	1 (33%)	–
Mediastinum, schwannoma malignant	1 (33%)	–	–	–
Genital System				
Bulbourethral gland	(1)	(1)	(0)	(0)
Coagulating gland	(0)	(2)	(3)	(0)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Ductus deferens	(1)	(0)	(1)	(0)
Leiomyoma	–	–	1 (100%)	–
Epididymis	(90)	(90)	(90)	(90)
Penis	(0)	(4)	(2)	(1)
Preputial gland	(88)	(88)	(89)	(89)
Sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Prostate	(90)	(90)	(90)	(85)
Adenoma	2 (2%)	–	2 (2%)	1 (1%)
Sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Seminal vesicle	(90)	(90)	(90)	(90)
Sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Testis	(90)	(89)	(90)	(90)
Hemangioma	–	–	1 (1%)	–
Interstitial cell, adenoma	2 (2%)	2 (2%)	1 (1%)	1 (1%)
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Pheochromocytoma malignant, metastatic, adrenal medulla	–	–	1 (1%)	–
Sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Lymph node	(25)	(23)	(24)	(16)
Iliac, sarcoma, metastatic, skeletal muscle	–	–	–	1 (6%)
Lumbar, sarcoma, metastatic, skeletal muscle	–	–	–	1 (6%)
Lymph node, mandibular	(89)	(90)	(90)	(88)
Lymph node, mesenteric	(90)	(89)	(88)	(88)
Sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Spleen	(90)	(90)	(90)	(85)
Hemangiosarcoma	3 (3%)	–	–	–
Thymus	(88)	(85)	(87)	(82)
Sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Schwannoma malignant	–	–	1 (1%)	–
Integumentary System				
Mammary gland	(82)	(77)	(80)	(80)
Fibroadenoma	2 (2%)	5 (6%)	2 (3%)	1 (1%)
Skin	(90)	(90)	(90)	(90)
Basal cell adenoma	1 (1%)	1 (1%)	–	–
Keratoacanthoma	5 (6%)	4 (4%)	5 (6%)	4 (4%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Squamous cell carcinoma	–	–	3 (3%)	–
Squamous cell papilloma	2 (2%)	1 (1%)	3 (3%)	1 (1%)
Trichoepithelioma	–	–	–	1 (1%)
Conjunctiva, sarcoma	–	–	1 (1%)	–
Sebaceous gland, adenoma	1 (1%)	–	–	1 (1%)
Subcutaneous tissue, fibroma	2 (2%)	11 (12%)	6 (7%)	4 (4%)
Subcutaneous tissue, fibroma, multiple	–	–	1 (1%)	–
Subcutaneous tissue, fibrosarcoma	1 (1%)	–	1 (1%)	–
Subcutaneous tissue, hemangiosarcoma	–	1 (1%)	–	–
Subcutaneous tissue, hibernoma	–	–	1 (1%)	–
Subcutaneous tissue, lipoma	2 (2%)	5 (6%)	4 (4%)	1 (1%)
Subcutaneous tissue, malignant fibrous histiocytoma	–	–	–	1 (1%)
Subcutaneous tissue, myxosarcoma	–	2 (2%)	–	–
Subcutaneous tissue, myxosarcoma, multiple	–	–	1 (1%)	–
Subcutaneous tissue, neural crest tumor	–	1 (1%)	–	–
Subcutaneous tissue, sarcoma	2 (2%)	–	–	–
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Bone, vertebra	(0)	(0)	(1)	(0)
Skeletal muscle	(90)	(90)	(90)	(90)
Hemangioma	–	1 (1%)	–	–
Hemangiosarcoma	–	–	1 (1%)	–
Sarcoma	–	–	–	1 (1%)
Nervous System				
Brain	(90)	(90)	(90)	(90)
Glioma malignant	–	–	–	3 (3%)
Meningioma benign	–	–	1 (1%)	–
Schwannoma malignant, metastatic, tissue NOS	–	1 (1%)	–	1 (1%)
Schwannoma malignant, metastatic, uncertain primary site	–	1 (1%)	–	–
Choroid plexus, granular cell tumor benign	–	–	–	1 (1%)
Meninges, granular cell tumor benign	1 (1%)	–	1 (1%)	1 (1%)
Meninges, granular cell tumor malignant		1 (1%)	1 (1%)	–
Nerve trigeminal	(84)	(90)	(88)	(90)
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Peripheral nerve, tibial	(88)	(90)	(90)	(89)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Trigeminal ganglion	(75)	(77)	(79)	(83)
Schwannoma malignant, metastatic, tissue NOS	–	1 (1%)	–	–
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Alveolar/bronchiolar adenoma	1 (1%)	2 (2%)	1 (1%)	–
Alveolar/bronchiolar carcinoma	–	–	1 (1%)	–
Carcinoma, metastatic, kidney	–	–	–	1 (1%)
Carcinoma, metastatic, thyroid gland	–	–	1 (1%)	–
Hepatocellular carcinoma, metastatic, liver	–	–	–	1 (1%)
Pheochromocytoma malignant, metastatic, adrenal medulla	–	–	2 (2%)	–
Sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Schwannoma malignant, metastatic, thymus	–	–	1 (1%)	–
Nose	(89)	(90)	(90)	(87)
Schwannoma malignant, metastatic, tissue NOS	–	–	–	1 (1%)
Schwannoma malignant, metastatic, uncertain primary site	1 (1%)	1 (1%)	–	–
Trachea	(90)	(88)	(88)	(72)
Special Senses System				
Eye	(85)	(83)	(81)	(72)
Schwannoma malignant, metastatic, uncertain primary site	–	1 (1%)	–	–
Retrobulbar, schwannoma malignant, metastatic, uncertain primary site	2 (2%)	–	–	–
Harderian gland	(90)	(90)	(90)	(89)
Schwannoma malignant, metastatic, tissue NOS	–	1 (1%)	–	1 (1%)
Schwannoma malignant, metastatic, uncertain primary site	2 (2%)	1 (1%)	–	–
Lacrimal gland	(2)	(1)	(1)	(1)
Zymbal's gland	(0)	(0)	(1)	(1)
Adenoma	–	–	1 (100%)	1 (100%)
Urinary System				
Kidney	(90)	(90)	(90)	(87)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Lipoma	1 (1%)	2 (2%)	1 (1%)	–
Liposarcoma	–	–	1 (1%)	–
Nephroblastoma	–	–	–	1 (1%)
Oncocytoma benign	1 (1%)	–	–	–
Bilateral, renal tubule, adenoma	–	–	1 (1%)	–
Bilateral, renal tubule, adenoma, multiple	1 (1%)	–	–	–
Bilateral, renal tubule, carcinoma	1 (1%)	–	1 (1%)	–
Pelvis, urothelium, carcinoma	–	–	–	1 (1%)
Perirenal tissue, sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Renal tubule, adenoma	1 (1%)	1 (1%)	–	–
Renal tubule, adenoma, multiple	1 (1%)	–	–	–
Renal tubule, carcinoma	–	–	–	1 (1%)
Urinary bladder	(89)	(83)	(83)	(78)
Serosa, sarcoma, metastatic, skeletal muscle	–	–	–	1 (1%)
Systemic Lesions				
Multiple organs[b]	(90)	(90)	(90)	(90)
Histiocytic sarcoma	–	–	–	1 (1%)
Leukemia mononuclear	–	2 (2%)	4 (4%)	1 (1%)
Lymphoma malignant	2 (2%)	2 (2%)	1 (1%)	4 (4%)
Mesothelioma malignant	2 (2%)	2 (2%)	3 (3%)	1 (1%)
Neoplasm Summary				
Total animals with primary neoplasms[c]				
Two-year study	56	74	76	63
Total primary neoplasms				
Two-year study	114	160	193	108
Total animals with benign neoplasms				
Two-year study	49	66	69	49
Total benign neoplasms				
Two-year study	87	132	145	71
Total animals with malignant neoplasms				
Two-year study	24[d]	26[d]	41	34
Total malignant neoplasms				
Two-year study	27	27	48	37
Total animals with metastatic neoplasms				
Two-year study	2	3	5	4
Total metastatic neoplasms				

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Two-year study	6	10	6	22
Total animals with malignant neoplasms-uncertain primary site				
Two-year study	2	1	–	–
Total animals with uncertain neoplasms-benign or malignant				
Two-year study	–	1	–	–
Total uncertain neoplasms				
Two-year study	–	1	–	–

[a]Number of animals examined microscopically at the site and the number of animals with neoplasm.
[b]Number of animals with any tissue examined microscopically.
[c]Primary neoplasms: all neoplasms except metastatic neoplasms.
[d]Includes one animal that had a neoplasm of unknown primary origin.

Table C-2. Statistical Analysis of Primary Neoplasms in Male Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adrenal Medulla: Benign Pheochromocytoma				
Overall rate[a]	10/88 (11%)	19/90 (21%)	22/90 (24%)	13/90 (14%)
Rate per litters[b]	8/35 (23%)	13/35 (37%)	17/35 (49%)	12/35 (34%)
Adjusted rate[c]	15.2%	25.4%	27.8%	19.1%
Terminal rate[d]	3/23 (13%)	14/43 (33%)	16/56 (29%)	9/43 (21%)
First incidence (days)	510	647	642	497
Rao-Scott adjusted Poly-3 test[e]	P = 0.440	P = 0.121	P = 0.070	P = 0.363
Litter C-A/Fisher's test[f]	P = 0.219	P = 0.148	P = 0.022	P = 0.214
Adrenal Medulla: Benign, Complex, or Malignant Pheochromocytoma				
Overall rate	11/88 (13%)	21/90 (23%)	23/90 (26%)	14/90 (16%)
Rate per litters	9/35 (26%)	15/35 (43%)	18/35 (51%)	13/35 (37%)
Adjusted rate	16.7%	28.1%	29.1%	20.4%
Terminal rate	3/23 (13%)	16/43 (37%)	17/56 (30%)	9/43 (21%)
First incidence (days)	510	647	642	497
Rao-Scott adjusted Poly-3 test	P = 0.474	P = 0.091	P = 0.070	P = 0.368
Litter C-A/Fisher's test	P = 0.251	P = 0.104	P = 0.024	P = 0.220
Heart: Schwannoma Malignant				
Overall rate	0/90 (0%)	2/90 (2%)	3/90 (3%)	6/90 (7%)
Rate per litters	0/35 (0%)	2/35 (6%)	3/35 (9%)	6/35 (17%)
Adjusted rate	0%	2.7%	3.8%	8.8%
Terminal rate	0/25 (0%)	2/43 (5%)	2/56 (4%)	3/43 (7%)
First incidence (days)	–[g]	730 (T)	642	488

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Rao-Scott adjusted Poly-3 test	P = 0.011	P = 0.273	P = 0.175	P = 0.030
Litter C-A/Fisher's test	P = 0.006	P = 0.246	P = 0.120	P = 0.012
Mammary Gland: Fibroadenoma				
Overall rate	2/90 (2%)	5/90 (6%)	2/90 (2%)	1/90 (1%)
Rate per litters	2/35 (6%)	5/35 (14%)	2/35 (6%)	1/35 (3%)
Adjusted rate	3.0%	6.7%	2.6%	1.5%
Terminal rate	1/25 (4%)	4/43 (9%)	2/56 (4%)	1/43 (2%)
First incidence (days)	440	550	730 (T)	730 (T)
Rao-Scott adjusted Poly-3 test	P = 0.229N	P = 0.271	P = 0.608N	P = 0.468N
Litter C-A/Fisher's test	P = 0.219N	P = 0.214	P = 0.693	P = 0.500N
Pancreas: Adenoma				
Overall rate	18/90 (20%)	22/88 (25%)	26/87 (30%)	7/78 (9%)
Rate per litters	16/35 (46%)	16/35 (46%)	18/35 (51%)	7/35 (20%)
Adjusted rate	26.8%	29.6%	33.5%	11.2%
Terminal rate	9/25 (36%)	13/43 (30%)	20/56 (36%)	5/43 (12%)
First incidence (days)	580	568	577	621
Rao-Scott adjusted Poly-3 test	P = 0.034N	P = 0.436	P = 0.268	P = 0.035N
Litter C-A/Fisher's test	P = 0.015N	P = 0.595	P = 0.406	P = 0.020N
Pancreas: Adenoma or Carcinoma				
Overall rate	18/90 (20%)	22/88 (25%)	27/87 (31%)	7/78 (9%)
Rate per litters	16/35 (46%)	16/35 (46%)	18/35 (51%)	7/35 (20%)
Adjusted rate	26.8%	29.6%	34.8%	11.2%
Terminal rate	9/25 (36%)	13/43 (30%)	21/56 (38%)	5/43 (12%)
First incidence (days)	580	568	577	621
Rao-Scott adjusted Poly-3 test	P = 0.037N	P = 0.436	P = 0.223	P = 0.035N
Litter C-A/Fisher's test	P = 0.015N	P = 0.595	P = 0.406	P = 0.020N
Pancreatic Islets: Adenoma				
Overall rate	5/90 (6%)	11/88 (13%)	8/87 (9%)	7/79 (9%)
Rate per litters	5/35 (14%)	8/35 (23%)	6/35 (17%)	7/35 (20%)
Adjusted rate	7.6%	15.1%	10.5%	11.2%
Terminal rate	2/25 (8%)	8/43 (19%)	7/56 (13%)	6/43 (14%)
First incidence (days)	624	605	726	665
Rao-Scott adjusted Poly-3 test	P = 0.458	P = 0.167	P = 0.396	P = 0.364
Litter C-A/Fisher's test	P = 0.413	P = 0.270	P = 0.500	P = 0.376
Pancreatic Islets: Carcinoma				
Overall rate	8/90 (9%)	6/88 (7%)	13/87 (15%)	6/79 (8%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Rate per litters	8/35 (23%)	6/35 (17%)	12/35 (34%)	6/35 (17%)
Adjusted rate	12.0%	8.3%	17.0%	9.5%
Terminal rate	3/25 (12%)	6/43 (14%)	10/56 (18%)	3/43 (7%)
First incidence (days)	663	730 (T)	642	582
Rao-Scott adjusted Poly-3 test	P = 0.512N	P = 0.313N	P = 0.268	P = 0.411N
Litter C-A/Fisher's test	P = 0.446N	P = 0.383N	P = 0.214	P = 0.383N
Pancreatic Islets: Adenoma or Carcinoma				
Overall rate	13/90 (14%)	17/88 (19%)	18/87 (21%)	13/79 (16%)
Rate per litters	12/35 (34%)	14/35 (40%)	14/35 (40%)	11/35 (31%)
Adjusted rate	19.4%	23.3%	23.5%	20.6%
Terminal rate	5/25 (20%)	14/43 (33%)	14/56 (25%)	9/43 (21%)
First incidence (days)	624	605	642	582
Rao-Scott adjusted Poly-3 test	P = 0.510	P = 0.357	P = 0.343	P = 0.506
Litter C-A/Fisher's test	P = 0.395N	P = 0.402	P = 0.402	P = 0.500N
Pituitary Gland (Pars Distalis): Adenoma				
Overall rate	17/89 (19%)	25/90 (28%)	34/90 (38%)	13/90 (14%)
Rate per litters	13/35 (37%)	18/35 (51%)	24/35 (69%)	12/35 (34%)
Adjusted rate	24.9%	32.7%	41.8%	19.0%
Terminal rate	5/25 (20%)	16/43 (37%)	22/56 (39%)	6/43 (14%)
First incidence (days)	527	605	471	567
Rao-Scott adjusted Poly-3 test	P = 0.226N	P = 0.208	P = 0.030	P = 0.273N
Litter C-A/Fisher's test	P = 0.398N	P = 0.168	P = 0.008	P = 0.500N
Skin: Keratoacanthoma				
Overall rate	5/90 (6%)	4/90 (4%)	5/90 (6%)	4/90 (4%)
Rate per litters	5/35 (14%)	3/35 (9%)	5/35 (14%)	3/35 (9%)
Adjusted rate	7.4%	5.4%	6.4%	6.0%
Terminal rate	0/25 (0%)	2/43 (5%)	4/56 (7%)	3/43 (7%)
First incidence (days)	552	610	726	665
Rao-Scott adjusted Poly-3 test	P = 0.477N	P = 0.430N	P = 0.521N	P = 0.490N
Litter C-A/Fisher's test	P = 0.360N	P = 0.355N	P = 0.633	P = 0.355N
Skin: Squamous Cell Papilloma or Keratoacanthoma				
Overall rate	7/90 (8%)	5/90 (6%)	8/90 (9%)	5/90 (6%)
Rate per litters	7/35 (20%)	4/35 (11%)	8/35 (23%)	4/35 (11%)
Adjusted rate	10.4%	6.7%	10.2%	7.5%
Terminal rate	1/25 (4%)	3/43 (7%)	6/56 (11%)	4/43 (9%)
First incidence (days)	552	610	717	665

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Rao-Scott adjusted Poly-3 test	P = 0.413N	P = 0.302N	P = 0.577N	P = 0.372N
Litter C-A/Fisher's test	P = 0.308N	P = 0.256N	P = 0.500	P = 0.256N
Skin: Squamous Cell Papilloma, Keratoacanthoma, or Squamous Cell Carcinoma				
Overall rate	7/90 (8%)	5/90 (6%)	10/90 (11%)	5/90 (6%)
Rate per litters	7/35 (20%)	4/35 (11%)	10/35 (29%)	4/35 (11%)
Adjusted rate	10.4%	6.7%	12.8%	7.5%
Terminal rate	1/25 (4%)	3/43 (7%)	7/56 (13%)	4/43 (9%)
First incidence (days)	552	610	717	665
Rao-Scott adjusted Poly-3 test	P = 0.443N	P = 0.297N	P = 0.410	P = 0.368N
Litter C-A/Fisher's test	P = 0.341N	P = 0.256N	P = 0.289	P = 0.256N
Skin: Squamous Cell Papilloma, Keratoacanthoma, Trichoepithelioma, Basal Cell Adenoma or Squamous Cell Carcinoma				
Overall rate	8/90 (9%)	6/90 (7%)	10/90 (11%)	6/90 (7%)
Rate per litters	8/35 (23%)	5/35 (14%)	10/35 (29%)	5/35 (14%)
Adjusted rate	11.9%	8.0%	12.8%	9.0%
Terminal rate	2/25 (8%)	4/43 (9%)	7/56 (13%)	4/43 (9%)
First incidence (days)	552	610	717	630
Rao-Scott adjusted Poly-3 test	P = 0.430N	P = 0.296N	P = 0.516	P = 0.371N
Litter C-A/Fisher's test	P = 0.334N	P = 0.270N	P = 0.393	P = 0.270N
Skin (Subcutaneous Tissue): Lipoma				
Overall rate	2/90 (2%)	5/90 (6%)	4/90 (4%)	1/90 (1%)
Rate per litters	2/35 (6%)	3/35 (9%)	4/35 (11%)	1/35 (3%)
Adjusted rate	3.1%	6.7%	5.1%	1.5%
Terminal rate	2/25 (8%)	3/43 (7%)	3/56 (5%)	0/43 (0%)
First incidence (days)	730 (T)	693	726	713
Rao-Scott adjusted Poly-3 test	P = 0.331N	P = 0.350	P = 0.480	P = 0.536N
Litter C-A/Fisher's test	P = 0.370N	P = 0.500	P = 0.337	P = 0.500N
Skin (Subcutaneous Tissue): Fibroma				
Overall rate	2/90 (2%)	11/90 (12%)	7/90 (8%)	4/90 (4%)
Rate per litters	2/35 (6%)	11/35 (31%)	7/35 (20%)	4/35 (11%)
Adjusted rate	3.1%	14.4%	8.8%	6.0%
Terminal rate	2/25 (8%)	5/43 (12%)	6/56 (11%)	2/43 (5%)
First incidence (days)	730 (T)	447	383	665
Rao-Scott adjusted Poly-3 test	P = 0.500N	P = 0.025	P = 0.143	P = 0.328
Litter C-A/Fisher's test	P = 0.500N	P = 0.006	P = 0.075	P = 0.337

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Skin (Subcutaneous Tissue): Fibroma, Fibrosarcoma, Sarcoma, Myxosarcoma, or Malignant Fibrous Histiocytoma				
Overall rate	5/90 (6%)	13/90 (14%)	10/90 (11%)	5/90 (6%)
Rate per litters	5/35 (14%)	12/35 (34%)	10/35 (29%)	5/35 (14%)
Adjusted rate	7.5%	16.9%	12.6%	7.5%
Terminal rate	2/25 (8%)	6/43 (14%)	8/56 (14%)	3/43 (7%)
First incidence (days)	567	447	383	665
Rao-Scott adjusted Poly-3 test	P = 0.335N	P = 0.081	P = 0.236	P = 0.600N
Litter C-A/Fisher's test	P = 0.342N	P = 0.046	P = 0.122	P = 0.633
Thyroid Gland (C-cell): Adenoma				
Overall rate	8/89 (9%)	14/87 (16%)	14/86 (16%)	11/85 (13%)
Rate per litters	7/35 (20%)	13/35 (37%)	13/34 (38%)	10/35 (29%)
Adjusted rate	12.1%	18.7%	18.5%	16.8%
Terminal rate	6/25 (24%)	5/43 (12%)	13/56 (23%)	8/43 (19%)
First incidence (days)	498	517	677	582
Rao-Scott adjusted Poly-3 test	P = 0.327	P = 0.186	P = 0.194	P = 0.278
Litter C-A/Fisher's test	P = 0.363	P = 0.093	P = 0.080	P = 0.289
Thyroid Gland (C-cell): Carcinoma				
Overall rate	2/89 (2%)	0/87 (0%)	2/86 (2%)	4/85 (5%)
Rate per litters	1/35 (3%)	0/35 (0%)	1/34 (3%)	4/35 (11%)
Adjusted rate	3.0%	0%	2.6%	6.2%
Terminal rate	0/25 (0%)	0/43 (0%)	2/56 (4%)	3/43 (7%)
First incidence (days)	541	–	730 (T)	717
Rao-Scott adjusted Poly-3 test	P = 0.173	P = 0.339N	P = 0.666N	P = 0.398
Litter C-A/Fisher's test	P = 0.034	P = 0.500N	P = 0.746	P = 0.178
Thyroid Gland (C-cell): Adenoma or Carcinoma				
Overall rate	10/89 (11%)	14/87 (16%)	16/86 (19%)	15/85 (18%)
Rate per litters	8/35 (23%)	13/35 (37%)	13/34 (38%)	12/35 (34%)
Adjusted rate	14.9%	18.7%	21.1%	22.9%
Terminal rate	6/25 (24%)	5/43 (12%)	15/56 (27%)	11/43 (26%)
First incidence (days)	498	517	677	582
Rao-Scott adjusted Poly-3 test	P = 0.151	P = 0.351	P = 0.233	P = 0.172
Litter C-A/Fisher's test	P = 0.249	P = 0.148	P = 0.130	P = 0.214
All Organs: Benign Neoplasms				
Overall rate	49/90 (54%)	66/90 (73%)	69/90 (77%)	49/90 (54%)
Rate per litters	26/35 (74%)	34/35 (97%)	35/35 (100%)	31/35 (89%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adjusted rate	65.3%	80.2%	82.3%	66.4%
Terminal rate	18/25 (72%)	37/43 (86%)	50/56 (89%)	28/43 (65%)
First incidence (days)	440	447	383	497
Rao-Scott adjusted Poly-3 test	P = 0.422N	P = 0.036	P = 0.017	P = 0.508
Litter C-A/Fisher's test	P = 0.108	P = 0.007	P < 0.001	P = 0.109
All Organs: Malignant Neoplasms				
Overall rate	24/90 (27%)	26/90 (29%)	41/90 (46%)	34/90 (38%)
Rate per litters	18/35 (51%)	20/35 (57%)	28/35 (80%)	25/35 (71%)
Adjusted rate	33.2%	34.2%	49.2%	46.1%
Terminal rate	5/25 (20%)	18/43 (42%)	26/56 (46%)	17/43 (40%)
First incidence (days)	212	550	393	153
Rao-Scott adjusted Poly-3 test	P = 0.034	P = 0.509	P = 0.031	P = 0.076
Litter C-A/Fisher's test	P = 0.030	P = 0.405	P = 0.011	P = 0.070
All Organs: Benign or Malignant Neoplasms				
Overall rate	57/90 (63%)	75/90 (83%)	76/90 (84%)	63/90 (70%)
Rate per litters	29/35 (83%)	35/35 (100%)	35/35 (100%)	33/35 (94%)
Adjusted rate	72.8%	89.2%	88.0%	79.7%
Terminal rate	20/25 (80%)	41/43 (95%)	51/56 (91%)	34/43 (79%)
First incidence (days)	212	447	383	153
Rao-Scott adjusted Poly-3 test	P = 0.337	P = 0.009	P = 0.014	P = 0.210
Litter C-A/Fisher's test	P = 0.109	P = 0.012	P = 0.012	P = 0.130

(T) = terminal euthanasia.
[a]Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for adrenal gland, heart, pancreas, pancreatic islets, pituitary gland, and thyroid gland; for other tissues, denominator is number of animals necropsied.
[b]Number of litters with tumor-bearing animals/number of litters examined at site.
[c]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[d]Observed incidence at terminal euthanasia.
[e]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
[f]The Litter Cochran-Armitage and Fishers exact tests directly compare the litter incidence rates.
[g]Not applicable; no neoplasms in animal group.

Table C-3. Historical Incidence of Malignant Schwannoma of the Heart in Control Male Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Incidence in Controls
Historical Incidence: All Studies	
p-Chloro-α,α,α-trifluorotoluene (January 2011)	0/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	1/50

Study (Study Start)	Incidence in Controls
Indole-3-carbinol (March 2007)	1/50
Radiofrequency radiation (September 2012)	0/90
Overall Historical Incidence	
Total (%)	2/240 (0.8%)
Mean ± standard deviation	1.0% ± 1.2%
Range	0%–2%

[a]Data as of November 2017.

Table C-4. Historical Incidence of Malignant Glioma of the Brain in Control Male Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Incidence in Controls
Historical Incidence: All Studies	
p-Chloro-α,α,α-trifluorotoluene (January 2011)	2/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	0/50
Radiofrequency radiation (September 2012)	0/90
Overall Historical Incidence	
Total (%)	2/190 (1.1%)
Mean ± standard deviation	1.3% ± 2.3%
Range	0%–4%

[a]Data as of November 2017; due to differences in sectioning method, indole-3-carbinol was excluded from evaluation of brain lesions.

Table C-5. Historical Incidence of Pituitary Gland (Pars Distalis) Adenoma in Control Male Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Incidence in Controls
Historical Incidence: All Studies	
p-Chloro-α,α,α-trifluorotoluene (January 2011)	11/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	14/50
Indole-3-carbinol (March 2007)	5/50
Radiofrequency radiation (September 2012)	17/89
Overall Historical Incidence	
Total (%)	47/239 (19.7%)
Mean ± standard deviation	19.8% ± 7.5%
Range	10%-28%

[a]Data as of November 2017.

Table C-6. Historical Incidence of Hepatocellular Neoplasms in Control Male Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Adenoma	Carcinoma	Adenoma or Carcinoma
Historical Incidence: All Studies			
p-Chloro-α,α,α-trifluorotoluene (January 2011)	0/50	0/50	0/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	0/50	0/50	0/50
Indole-3-carbinol (March 2007)	1/50	0/50	1/50
Radiofrequency radiation (September 2012)	0/90	0/90	0/90
Overall Historical Incidence			
Total (%)	1/240 (0.4%)	0/240	1/240 (0.4%)
Mean ± standard deviation	0.5% ± 1.0%	–	0.5% ± 1.0%
Range	0%-2%	–	0%-2%

[a]Data as of November 2017.

Table C-7. Summary of the Incidence of Non-neoplastic Lesions in Male Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
Fourteen-week interim evaluation	15	15	15	15
Early deaths				
Accidental death	1	–	–	–
Moribund	44	24	13	6
Natural deaths	20	23	21	41
Survivors				
Died last week of study	–	–	1	–
Terminal euthanasia	25	43	55	43
Animals examined microscopically	100	100	100	100
Fourteen-week Interim Evaluation				
Alimentary System				
Esophagus	(10)	(10)	(9)	(10)
Intestine large, cecum	(10)	(10)	(9)	(10)
Intestine large, colon	(10)	(10)	(9)	(10)
Intestine large, rectum	(10)	(10)	(10)	(10)
Lymphoid tissue, hyperplasia	1 (10%)	1 (10%)	–	1 (10%)
Intestine small, duodenum	(10)	(10)	(10)	(10)
Intestine small, ileum	(10)	(10)	(9)	(10)
Intestine small, jejunum	(10)	(10)	(10)	(10)
Liver	(10)	(10)	(10)	(10)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Hepatodiaphragmatic nodule	–	–	1 (10%)	–
Infiltration cellular, mixed cell	1 (10%)	1 (10%)	1 (10%)	3 (30%)
Hepatocyte, necrosis	–	–	1 (10%)	–
Pancreas	(10)	(10)	(10)	(10)
Salivary glands	(10)	(10)	(9)	(10)
Stomach, forestomach	(10)	(10)	(10)	(10)
Stomach, glandular	(10)	(10)	(10)	(10)
Cardiovascular System				
Aorta	(10)	(10)	(10)	(10)
Heart	(10)	(10)	(10)	(10)
Cardiomyopathy	2 (20%)	–	3(30%)	6 (60%)
Artery, inflammation, chronic active	–	1 (10%)	1 (10%)	–
Ventricle right, cardiomyopathy	1 (10%)	5 (50%)	4 (40%)	4 (40%)
Endocrine System				
Adrenal cortex	(10)	(10)	(10)	(10)
Adrenal medulla	(10)	(10)	(10)	(10)
Islets, pancreatic	(10)	(10)	(10)	(10)
Parathyroid gland	(9)	(9)	(9)	(10)
Pituitary gland	(10)	(10)	(10)	(10)
Pars intermedia, cyst	–	1 (10%)	–	2 (20%)
Rathke’s cleft, cyst	–	1 (10%)	1 (10%)	–
Thyroid gland	(10)	(10)	(9)	(10)
Genital System				
Epididymis	(10)	(10)	(10)	(10)
Granuloma sperm	–	–	1 (10%)	–
Hypospermia	–	–	–	1 (10%)
Preputial gland	(10)	(10)	(10)	(10)
Inflammation, chronic active	7 (70%)	3 (30%)	6 (60%)	3 (30%)
Prostate	(10)	(10)	(10)	(10)
Inflammation, chronic active	–	–	–	1 (10%)
Seminal vesicle	(10)	(10)	(10)	(10)
Testis	(10)	(10)	(10)	(10)
Germ cell, degeneration	–	–	–	1 (10%)
Hematopoietic System				
Bone marrow	(10)	(10)	(9)	(10)
Lymph node	(0)	(0)	(1)	(0)
Inguinal, pigment	–	–	1 (100%)	–
Lymph node, mandibular	(10)	(10)	(9)	(10)
Hemorrhage	2 (20%)	1 (10%)	–	1 (10%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Lymph node, mesenteric	(10)	(10)	(9)	(10)
Hemorrhage	–	–	–	1 (10%)
Spleen	(10)	(10)	(9)	(10)
Thymus	(10)	(10)	(10)	(10)
Hemorrhage	5 (50%)	3 (30%)	4 (40%)	2 (20%)
Respiratory System				
Lung	(10)	(10)	(10)	(10)
Congestion	–	1 (10%)	–	1 (10%)
Inflammation, chronic active	1 (10%)	–	1 (10%)	1 (10%)
Nose	(10)	(10)	(10)	(10)
Trachea	(10)	(10)	(9)	(10)
Special Senses System				
Eye	(10)	(10)	(10)	(10)
Retina, developmental malformation	–	1 (10%)	–	–
Harderian gland	(10)	(10)	(10)	(10)
Infiltration cellular, lymphocyte	–	1 (10%)	–	–
Inflammation, chronic active	–	1 (10%)	3 (30%)	1 (10%)
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Congestion	–	–	2 (20%)	–
Nephropathy, chronic progressive	9 (90%)	7 (70%)	8 (80%)	9 (90%)
Pelvis, dilation	–	–	–	1 (10%)
Urinary bladder	(10)	(10)	(10)	(10)
Systems Examined with No Lesions Observed				
General Body System				
Integumentary System				
Musculoskeletal System				
Nervous System				
Two-year Study				
Alimentary System				
Esophagus	(90)	(90)	(90)	(90)
Dilation	2 (2%)	–	–	–
Hyperplasia	1 (1%)	–	–	–
Intestine large, cecum	(75)	(76)	(74)	(68)
Edema	11 (15%)	–	–	–
Erosion	10 (13%)	1 (1%)	1 (1%)	1 (1%)
Hemorrhage	–	1 (1%)	–	–
Inflammation, acute	10 (13%)	1 (1%)	–	1 (1%)
Inflammation, chronic active	1 (1%)	1 (1%)	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Necrosis	–	–	1 (1%)	–
Ulcer	6 (8%)	–	–	–
Artery, inflammation, chronic active	20 (27%)	8 (11%)	7 (9%)	2 (3%)
Artery, mineral	1 (1%)	–	–	–
Artery, thrombus	–	–	1 (1%)	–
Epithelium, regeneration	14 (19%)	1 (1%)	–	1 (1%)
Intestine large, colon	(81)	(83)	(82)	(76)
Cyst	–	1 (1%)	–	–
Erosion	1 (1%)	1 (1%)	–	–
Inflammation, acute	1 (1%)	–	–	–
Ulcer	1 (1%)	–	–	–
Artery, inflammation, chronic active	12 (15%)	4 (5%)	5 (6%)	1 (1%)
Artery, mineral	2 (2%)	–	–	–
Epithelium, regeneration	5 (6%)	–	–	–
Intestine large, rectum	(83)	(81)	(80)	(76)
Edema	1 (1%)	–	–	–
Erosion	1 (1%)	–	–	–
Hyperplasia, lymphocyte	1 (1%)	–	–	–
Inflammation, acute	2 (2%)	–	–	–
Inflammation, chronic active	–	1 (1%)	–	–
Artery, inflammation, chronic active	4 (5%)	1 (1%)	1 (1%)	1 (1%)
Epithelium, regeneration	3 (4%)	–	–	–
Intestine small, duodenum	(81)	(84)	(83)	(66)
Dilation	–	1 (1%)	–	–
Ectopic tissue	–	1 (1%)	–	–
Erosion	1 (1%)	–	–	–
Ulcer	1 (1%)	1 (1%)	–	–
Artery, inflammation, chronic active	–	–	3 (4%)	–
Intestine small, ileum	(78)	(76)	(77)	(63)
Congestion	–	1 (1%)	–	–
Hemorrhage	–	–	1 (1%)	–
Inflammation, acute	–	1 (1%)	–	–
Artery, inflammation, chronic active	2 (3%)	–	1 (1%)	–
Epithelium, regeneration	1 (1%)	–	–	–
Intestine small, jejunum	(73)	(73)	(75)	(62)
Artery, inflammation, chronic active	–	–	1 (1%)	–
Liver	(90)	(90)	(89)	(88)
Angiectasis	1 (1%)	1 (1%)	–	1 (1%)
Basophilic focus	1 (1%)	–	2 (2%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Clear cell focus	8 (9%)	4 (4%)	5 (6%)	5 (6%)
Eosinophilic focus	12 (13%)	5 (6%)	11 (12%)	4 (5%)
Extramedullary hematopoiesis	5 (6%)	4 (4%)	3 (3%)	1 (1%)
Hepatodiaphragmatic nodule	1 (1%)	1 (1%)	–	1 (1%)
Infiltration cellular, mixed cell	3 (3%)	1 (1%)	3 (3%)	2 (2%)
Mixed cell focus	32 (36%)	51 (57%)	47 (53%)	37 (42%)
Artery, inflammation, chronic active	2 (2%)	1 (1%)	–	–
Artery, mineral	1 (1%)	1 (1%)	–	–
Bile duct, cyst	3 (3%)	5 (6%)	2 (2%)	1 (1%)
Bile duct, fibrosis	–	–	–	1 (1%)
Bile duct, hyperplasia	41 (46%)	33 (37%)	26 (29%)	14 (16%)
Hepatocyte, degeneration	1 (1%)	–	1 (1%)	1 (1%)
Hepatocyte, necrosis	5 (6%)	6 (7%)	6 (7%)	6 (7%)
Hepatocyte, vacuolation, cytoplasmic	6 (7%)	6 (7%)	7 (8%)	7 (8%)
Kupffer cell, pigment	1 (1%)	–	–	–
Periductal, cholangiofibrosis	2 (2%)	2 (2%)	2 (2%)	–
Mesentery	(39)	(19)	(17)	(6)
Fibrosis	–	1 (5%)	–	
Hemorrhage	1 (3%)	–	–	1 (17%)
Inflammation, chronic	2 (5%)	–	–	–
Necrosis	2 (5%)	1 (5%)	1 (6%)	1 (17%)
Neovascularization	1 (3%)	2 (11%)	3 (18%)	–
Artery, inflammation, chronic active	32 (82%)	16 (84%)	13 (76%)	3 (50%)
Artery, mineral	21 (54%)	5 (26%)	2 (12%)	–
Vein, degeneration	1 (3%)	–	–	–
Vein, inflammation, chronic active	1 (3%)	2 (11%)	1 (6%)	–
Oral mucosa	(0)	(1)	(1)	(0)
Ulcer	–	1 (100%)	–	–
Pancreas	(90)	(88)	(87)	(78)
Cyst	1 (1%)	–	–	1 (1%)
Inflammation, chronic active	–	1 (1%)	–	–
Thrombus	1 (1%)	1 (1%)	–	–
Acinus, atrophy	13 (14%)	9 (10%)	10 (11%)	8 (10%)
Acinus, hyperplasia	63 (70%)	55 (63%)	49 (56%)	28 (36%)
Artery, inflammation, chronic active	48 (53%)	28 (32%)	23 (26%)	5 (6%)
Artery, mineral	11 (12%)	2 (2%)	–	–
Duct, crystals	–	–	1 (1%)	–
Duct, inflammation, acute	–	–	1 (1%)	–
Salivary glands	(90)	(90)	(90)	(86)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Artery, inflammation, chronic active	11 (12%)	6 (7%)	2 (2%)	1 (1%)
Artery, mineral	2 (2%)	1 (1%)	1 (1%)	–
Duct, parotid gland, dilation	5 (6%)	1 (1%)	1 (1%)	–
Duct, parotid gland, inflammation, acute	1 (1%)	1 (1%)	–	–
Parotid gland, atrophy	18 (20%)	15 (17%)	8 (9%)	3 (3%)
Parotid gland, inflammation, acute	2 (2%)	4 (4%)	2 (2%)	
Parotid gland, vacuolation, cytoplasmic	1 (1%)	2 (2%)	–	–
Sublingual gland, atrophy	–	–	1 (1%)	1 (1%)
Sublingual gland, mineral	–	–	–	1 (1%)
Submandibular gland, atrophy	–	2 (2%)	–	–
Stomach, forestomach	(90)	(90)	(89)	(90)
Cyst	–	1 (1%)	–	–
Edema	5 (6%)	5 (6%)	1 (1%)	1 (1%)
Erosion	–	1 (1%)	–	–
Inflammation, acute	1 (1%)	1 (1%)	1 (1%)	–
Inflammation, chronic	–	–	–	1 (1%)
Inflammation, chronic active	7 (8%)	4 (4%)	10 (11%)	1 (1%)
Mineral	1 (1%)	1 (1%)	–	–
Ulcer	6 (7%)	8 (9%)	4 (4%)	1 (1%)
Artery, inflammation, chronic active	–	1 (1%)	–	–
Epithelium, hyperplasia	11 (12%)	17 (19%)	11 (12%)	6 (7%)
Epithelium, hyperplasia, atypical	1 (1%)	–	–	–
Epithelium, hyperplasia, basal cell	–	–	1 (1%)	1 (1%)
Erosion	3(3%)	2 (2%)	3 (4%)	–
Inflammation, acute	1 (1%)	–	–	–
Inflammation, chronic active	1 (1%)	–	–	–
Mineral	31 (36%)	9 (10%)	6 (7%)	1 (1%)
Necrosis	–	–	3 (4%)	–
Artery, inflammation, chronic active	3 (3%)	–	–	–
Artery, mineral	–	–	1 (1%)	–
Epithelium, hyperplasia, focal	–	–	–	1 (1%)
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Dilation	–	5 (6%)	1 (1%)	–
Mineral	30 (33%)	8 (9%)	6 (7%)	2 (2%)
Blood vessel	(1)	(2)	(1)	(0)
Inflammation, chronic active	–	–	1 (100%)	–
Mineral	1 (100%)	–	–	–
Pulmonary artery, mineral	–	1 (50%)	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pulmonary artery, necrosis	–	1 (50%)	–	–
Heart	(90)	(90)	(90)	(90)
Cardiomyopathy	79 (88%)	84 (93%)	83 (92%)	85 (94%)
Congestion	1 (1%)	–	–	–
Hemorrhage	–	–	–	1 (1%)
Inflammation, suppurative	–	–	1 (1%)	–
Thrombus	1 (1%)	–	3 (3%)	–
Artery, degeneration	–	1 (1%)	–	–
Artery, inflammation, chronic active	–	–	2 (2%)	–
Artery, mineral	20 (22%)	7 (8%)	2 (2%)	1 (1%)
Artery, pericardium, inflammation, chronic active	–	–	–	1 (1%)
Artery, pericardium, pigment	–	1 (1%)	–	–
Atrium, dilation	3 (3%)	1 (1%)	–	4 (4%)
Atrium, thrombus	1 (1%)	5 (6%)	–	1 (1%)
Atrium, myocardium, hypertrophy	1 (1%)	1 (1%)	–	1 (1%)
Atrium, myocardium, necrosis	–	1 (1%)	–	–
Atrium left, mineral	–	–	1 (1%)	–
Endocardium, hyperplasia, Schwann cell	–	–	–	3 (3%)
Myocardium, mineral	9 (10%)	2 (2%)	1 (1%)	–
Myocardium, necrosis	1 (1%)	1 (1%)	–	1 (1%)
Pericardium, hemorrhage	–	–	1 (1%)	–
Valve, inflammation, chronic active	1 (1%)	–	–	–
Ventricle right, cardiomyopathy	54 (60%)	45 (50%)	62 (69%)	74 (82%)
Ventricle right, dilation	–	–	1 (1%)	–
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(89)
Accessory adrenal cortical nodule	6 (7%)	4 (4%)	7 (8%)	7 (8%)
Angiectasis	–	1 (1%)	–	–
Atrophy	–	1 (1%)	–	1 (1%)
Degeneration	3 (3%)	1 (1%)	1 (1%)	2 (2%)
Degeneration, cystic	–	3 (3%)	–	1 (1%)
Extramedullary hematopoiesis	–	–	1 (1%)	–
Hyperplasia	47 (52%)	42 (47%)	45 (50%)	44 (49%)
Hypertrophy	35 (39%)	42 (47%)	55 (61%)	44 (49%)
Necrosis	5 (6%)	5 (6%)	1 (1%)	1 (1%)
Pigment	–	–	–	1 (1%)
Thrombus	2 (2%)	2 (2%)	1 (1%)	–
Vacuolation, cytoplasmic	20 (22%)	18 (20%)	21 (23%)	12 (13%)
Adrenal medulla	(88)	(90)	(90)	(90)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Hyperplasia	42 (48%)	34 (38%)	32 (36%)	21 (23%)
Thrombus	1 (1%)	–	–	–
Islets, pancreatic	(90)	(88)	(87)	(79)
Hyperplasia	12 (13%)	15 (17%)	13 (15%)	12 (15%)
Parathyroid gland	(83)	(83)	(83)	(82)
Fibrosis	–	–	3 (4%)	–
Hyperplasia	51 (61%)	35 (42%)	32 (39%)	17 (21%)
Hyperplasia, focal	–	1 (1%)	–	–
Pituitary gland	(89)	(90)	(90)	(90)
Craniopharyngeal duct, cyst	1 (1%)	–	–	1 (1%)
Pars distalis, angiectasis	–	–	–	1 (1%)
Pars distalis, atrophy	–	–	–	1 (1%)
Pars distalis, cyst	5 (6%)	15 (17%)	7 (8%)	6 (7%)
Pars distalis, hyperplasia	32 (36%)	32 (36%)	34 (38%)	27 (30%)
Pars distalis, necrosis	–	1 (1%)	–	–
Pars intermedia, angiectasis	1 (1%)	1 (1%)	–	–
Pars intermedia, cyst	6 (7%)	1 (1%)	5 (6%)	7 (8%)
Pars intermedia, hyperplasia	1 (1%)	3 (3%)	–	2 (2%)
Pars nervosa, cyst	–	1 (1%)	–	–
Thyroid gland	(89)	(87)	(86)	(85)
C-cell, hyperplasia	16 (18%)	17 (20%)	17 (20%)	22 (26%)
Follicle, cyst	–	2 (2%)	–	1 (1%)
Follicle, hyperplasia, cystic	1 (1%)	–	–	–
General Body System				
Tissue NOS	(3)	(1)	(3)	(3)
Abdominal, fat, hemorrhage	1 (33%)	–	–	–
Fat, hemorrhage	–	–	1 (33%)	–
Fat, necrosis	2 (67%)	–	1 (33%)	1 (33%)
Genital System				
Bulbourethral gland	(1)	(1)	(0)	(0)
Coagulating gland	(0)	(2)	(3)	(0)
Inflammation, suppurative	–	–	1 (33%)	–
Inflammation, chronic active	–	2 (100%)	2 (67%)	–
Ductus deferens	(1)	(0)	(1)	(0)
Granuloma	1 (100%)	–	–	–
Epididymis	(90)	(90)	(90)	(90)
Exfoliated germ cell	51 (57%)	33 (37%)	33 (37%)	17 (19%)
Granuloma sperm	1 (1%)	1 (1%)	–	–
Hypospermia	28 (31%)	24 (27%)	13 (14%)	13 (14%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Inflammation, chronic	–	–	–	1 (1%)
Inflammation, chronic active	–	–	1 (1%)	–
Artery, inflammation, chronic active	2 (2%)	3 (3%)	3 (3%)	3 (3%)
Artery, thrombus	–	–	–	1 (1%)
Tail, developmental malformation	–	1 (1%)	–	–
Penis	(0)	(4)	(2)	(1)
Concretion	–	3 (75%)	2 (100%)	1 (100%)
Prolapse	–	1 (25%)	–	–
Preputial gland	(88)	(88)	(89)	(89)
Atrophy	1 (1%)	1 (1%)	–	–
Fibrosis	–	–	2 (2%)	–
Hyperplasia	1 (1%)	–	–	–
Inflammation, suppurative		1 (1%)	–	–
Inflammation, granulomatous	1 (1%)	–	–	–
Inflammation, acute	1 (1%)	–	–	1 (1%)
Inflammation, chronic active	46 (52%)	53 (60%)	46 (52%)	49 (55%)
Metaplasia, squamous	–	–	1 (1%)	–
Artery, inflammation, chronic active	1 (1%)	–	–	–
Duct, dilation	51 (58%)	54 (61%)	50 (56%)	48 (54%)
Duct, hyperplasia	–	1 (1%)	–	1 (1%)
Prostate	(90)	(90)	(90)	(85)
Decreased secretory fluid	4 (4%)	5 (6%)	7 (8%)	3 (4%)
Hemorrhage	1 (1%)	–	1 (1%)	–
Infiltration cellular, mononuclear cell	1 (1%)	–	–	1 (1%)
Inflammation, acute	7 (8%)	9 (10%)	4 (4%)	2 (2%)
Inflammation, chronic active	6 (7%)	10 (11%)	10 (11%)	5 (6%)
Artery, inflammation, chronic active	1 (1%)	–	3 (3%)	–
Artery, thrombus	–	1 (1%)	–	–
Epithelium, hyperplasia	5 (6%)	11 (12%)	9 (10%)	15 (18%)
Seminal vesicle	(90)	(90)	(90)	(90)
Decreased secretory fluid	35 (39%)	34 (38%)	18 (20%)	7 (8%)
Developmental malformation	–	–	1 (1%)	–
Dilation	–	–	1 (1%)	–
Hemorrhage	1 (1%)	–	1 (1%)	–
Hyperplasia, atypical	–	–	–	1 (1%)
Inflammation, acute	4 (4%)	1 (1%)	3 (3%)	1 (1%)
Inflammation, chronic active	1 (1%)	4 (4%)	–	–
Artery, inflammation, chronic active	1 (1%)	–	–	–
Epithelium, hyperplasia	1 (1%)	–	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Lumen, hemorrhage	–	–	1 (1%)	
Testis	(90)	(89)	(90)	(90)
Cyst	1 (1%)	–	–	–
Edema	–	2 (2%)	–	–
Inflammation, chronic active	2 (2%)	–	–	–
Pigment	1 (1%)	–	–	–
Artery, inflammation, chronic active	52 (58%)	37 (42%)	30 (33%)	12 (13%)
Germ cell, degeneration	51 (57%)	37 (42%)	31 (34%)	24 (27%)
Germinal epithelium, mineral	–	1 (1%)	–	–
Interstitial cell, hyperplasia	1 (1%)	2 (2%)	–	1 (1%)
Rete testis, dilation	1 (1%)	–	–	–
Seminiferous tubule, dilation	1 (1%)	1 (1%)	1 (1%)	–
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Hemorrhage	–	5 (6%)	3 (3%)	–
Hypercellularity	15 (17%)	25 (28%)	18 (20%)	13 (14%)
Hypocellularity	–	–	1 (1%)	1 (1%)
Lymph node	(25)	(23)	(24)	(16)
Bronchial, erythrophagocytosis	–	2 (9%)	–	–
Bronchial, hyperplasia, lymphocyte	–	1 (4%)	–	–
Iliac, erythrophagocytosis	2 (8%)	2 (9%)	1 (4%)	–
Iliac, hyperplasia, lymphocyte	2 (8%)	–	2 (8%)	–
Iliac, infiltration cellular, histiocyte	2 (8%)	1 (4%)	–	–
Iliac, pigment	–	–	1 (4%)	–
Iliac, proliferation, plasma cell	3 (12%)	–	1 (4%)	–
Iliac, lymphatic sinus, ectasia	5 (20%)	3 (13%)	1 (4%)	–
Inguinal, hyperplasia, lymphocyte	–	–	1 (4%)	–
Inguinal, lymphatic sinus, ectasia	–	–	1 (4%)	–
Lumbar, erythrophagocytosis	2 (8%)	2 (9%)	1 (4%)	1 (6%)
Lumbar, proliferation, plasma cell	–	1 (4%)	–	–
Lumbar, lymphatic sinus, ectasia	–	2 (9%)	1 (4%)	2 (13%)
Lymphatic sinus, mediastinal, ectasia	1 (4%)	1 (4%)	1 (4%)	1 (6%)
Lymphatic sinus, popliteal, ectasia	–	1 (4%)	–	–
Lymphatic sinus, renal, ectasia	–	4 (17%)	3 (13%)	–
Mediastinal, erythrophagocytosis	6 (24%)	7 (30%)	7 (29%)	3 (19%)
Mediastinal, extramedullary hematopoiesis	–	–	1 (4%)	–
Mediastinal, hemorrhage	1 (4%)	1 (4%)	1 (4%)	1 (6%)
Mediastinal, hyperplasia, lymphocyte	–	–	1 (4%)	–
Mediastinal, infiltration cellular, histiocyte	–	1 (4%)	1 (4%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Mediastinal, inflammation, acute	–	1 (4%)	–	–
Mediastinal, pigment	–	1 (4%)	–	–
Mediastinal, proliferation, plasma cell	–	–	1 (4%)	–
Pancreatic, erythrophagocytosis	3 (12%)	1 (4%)	4 (17%)	3 (19%)
Pancreatic, hemorrhage	1 (4%)	–	–	–
Pancreatic, hyperplasia, lymphocyte	1 (4%)	–	–	–
Pancreatic, infiltration cellular, mixed cell	–	–	–	1 (6%)
Renal, erythrophagocytosis	8 (32%)	6 (26%)	4 (17%)	
Renal, hyperplasia, lymphocyte	–	1 (4%)	–	–
Renal, infiltration cellular, mixed cell	–	–	–	1 (6%)
Renal, proliferation, plasma cell	2 (8%)	–	–	–
Lymph node, mandibular	(89)	(90)	(90)	(88)
Congestion	–	1 (1%)	2 (2%)	–
Erythrophagocytosis	–	3 (3%)	2 (2%)	1 (1%)
Hemorrhage	–	–	1 (1%)	–
Hyperplasia, lymphocyte	41 (46%)	50 (56%)	52 (58%)	40 (45%)
Infiltration cellular, histiocyte	–	2 (2%)	–	1 (1%)
Infiltration cellular, polymorphonuclear	2 (2%)	–	–	–
Necrosis, lymphocyte	–	1 (1%)	–	–
Proliferation, plasma cell	49 (55%)	61 (68%)	62 (69%)	57 (65%)
Lymphatic sinus, ectasia	16 (18%)	24 (27%)	29 (32%)	14 (16%)
Lymph node, mesenteric	(90)	(89)	(88)	(88)
Erythrophagocytosis	17 (19%)	5 (6%)	5 (6%)	9 (10%)
Hyperplasia, lymphocyte	2 (2%)	3 (3%)	3 (3%)	3 (3%)
Infiltration cellular, histiocyte	1 (1%)	–	–	–
Infiltration cellular, polymorphonuclear	2 (2%)	–	–	1 (1%)
Proliferation, plasma cell	–	1 (1%)	–	–
Lymphatic sinus, ectasia	–	2 (2%)	3 (3%)	1 (1%)
Lymphocyte, depletion	2 (2%)	–	–	–
Spleen	(90)	(90)	(90)	(85)
Congestion	–	–	1 (1%)	–
Developmental malformation	1 (1%)	–	–	–
Extramedullary hematopoiesis	45 (50%)	60 (67%)	56 (62%)	48 (56%)
Hemorrhage	–	1 (1%)	1 (1%)	–
Hyperplasia, lymphocyte	5 (6%)	–	–	–
Necrosis	–	–	2 (2%)	–
Pigment	57 (63%)	54 (60%)	64 (71%)	63 (74%)
Thrombus	–	1 (1%)	–	–
Arteriole, mineral	1 (1%)	–	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Red pulp, atrophy	26 (29%)	14 (16%)	12 (13%)	13 (15%)
White pulp, atrophy	30 (33%)	11 (12%)	10 (11%)	24 (28%)
Thymus	(88)	(85)	(87)	(82)
Atrophy	79 (90%)	76 (89%)	80 (92%)	65 (79%)
Cyst	10 (11%)	10 (12%)	10 (11%)	17 (21%)
Ectopic parathyroid gland	6 (7%)	6 (7%)	7 (8%)	5 (6%)
Ectopic thyroid	1 (1%)	–	–	–
Hemorrhage	2 (2%)	2 (2%)	2 (2%)	20 (24%)
Hyperplasia, epithelial	2 (2%)	2 (2%)	4 (5%)	4 (5%)
Artery, inflammation, chronic active	6 (7%)	3 (4%)	2 (2%)	1 (1%)
Integumentary System				
Mammary gland	(82)	(77)	(80)	(80)
Atrophy	1 (1%)	2 (3%)	3 (4%)	–
Galactocele	1 (1%)	1 (1%)	2 (3%)	–
Duct, dilation	3 (4%)	8 (10%)	9 (11%)	3 (4%)
Skin	(90)	(90)	(90)	(90)
Cyst epithelial inclusion	3 (3%)	12 (13%)	3 (3%)	2 (2%)
Inflammation, suppurative	–	2 (2%)	–	–
Inflammation, chronic active	1 (1%)	2 (2%)	2 (2%)	1 (1%)
Ulcer	2 (2%)	2 (2%)	4 (4%)	–
Adnexa, atrophy	–	–	–	1 (1%)
Artery, subcutaneous tissue, inflammation, chronic active	1 (1%)	–	–	–
Dermis, fibrosis	–	–	–	1 (1%)
Epidermis, hyperplasia	1 (1%)	1 (1%)	–	1 (1%)
Hair follicle, congestion	–	–	–	1 (1%)
Hair follicle, degeneration	–	–	1 (1%)	–
Prepuce, hyperplasia	–	2 (2%)	–	1 (1%)
Prepuce, inflammation, acute	–	–	–	1 (1%)
Prepuce, inflammation, chronic active	–	1 (1%)	–	–
Prepuce, ulcer	–	2 (2%)	–	1 (1%)
Subcutaneous tissue, hemorrhage	–	1 (1%)	–	–
Subcutaneous tissue, inflammation, suppurative	1 (1%)	–	1 (1%)	2 (2%)
Subcutaneous tissue, inflammation, chronic	1 (1%)	–	1 (1%)	–
Subcutaneous tissue, inflammation, chronic active	–	–	2 (2%)	–
Subcutaneous tissue, necrosis	–	–	1 (1%)	–
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Fibrous osteodystrophy	46 (51%)	20 (22%)	15 (17%)	5 (6%)
Cranium, inflammation, chronic active	–	–	1 (1%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Bone, vertebra	(0)	(0)	(1)	(0)
Developmental malformation	–	–	1 (100%)	–
Skeletal muscle	(90)	(90)	(90)	(90)
Degeneration	34 (38%)	35 (39%)	30 (33%)	26 (29%)
Inflammation, chronic active	–	–	1 (1%)	–
Mineral	2 (2%)	–	1 (1%)	–
Diaphragm, hernia	–	1 (1%)	–	–
Nervous System				
Brain	(90)	(90)	(90)	(90)
Compression	7 (8%)	12 (13%)	6 (7%)	3 (3%)
Edema	–	1 (1%)	–	–
Hemorrhage	2 (2%)	3 (3%)	–	–
Infiltration cellular, mononuclear cell	1 (1%)	–	–	–
Inflammation, suppurative	–	–	1 (1%)	–
Mineral	5 (6%)	3 (3%)	4 (4%)	4 (4%)
Necrosis	7 (8%)	7 (8%)	3 (3%)	–
Choroid plexus, degeneration	1 (1%)	–	–	–
Choroid plexus, mineral	3 (3%)	1 (1%)	–	–
Glial cell, hyperplasia	–	2 (2%)	–	2 (2%)
Hypothalamus, cyst	–	3 (3%)	–	–
Meninges, fibrosis	–	1 (1%)	–	–
Meninges, hyperplasia	1 (1%)	–	1 (1%)	–
Meninges, hyperplasia, granular cell	1 (1%)	1 (1%)	–	–
Meninges, mineral	–	1 (1%)	–	–
Pineal gland, mineral	3 (3%)	3 (3%)	2(2%)	–
Pineal gland, vacuolation, cytoplasmic	12 (13%)	6 (7%)	9 (10%)	4 (4%)
Nerve trigeminal	(84)	(90)	(88)	(90)
Degeneration	63 (75%)	66 (73%)	67 (76%)	49 (54%)
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Degeneration	86 (96%)	90 (100%)	88 (98%)	84 (93%)
Infiltration cellular, histiocyte	–	–	1 (1%)	–
Infiltration cellular, mononuclear cell	1 (1%)	–	–	–
Peripheral nerve, tibial	(88)	(90)	(90)	(89)
Degeneration	84 (95%)	90 (100%)	89 (99%)	81 (91%)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Degeneration	30 (33%)	36 (40%)	42 (47%)	35 (39%)
Meninges, inflammation, suppurative	–	–	1 (1%)	–
Spinal cord, lumbar	(90)	(90)	(90)	(90)
Degeneration	21 (23%)	15 (17%)	21 (23%)	24 (27%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Nerve, degeneration	79 (88%)	85 (94%)	83 (92%)	76 (84%)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Degeneration	58 (64%)	69 (77%)	74 (82%)	62 (69%)
Hemorrhage, focal	1 (1%)	–	–	–
Meninges, inflammation, suppurative	–	–	1 (1%)	–
Trigeminal ganglion	(75)	(77)	(79)	(83)
Degeneration	23 (31%)	22 (29%)	21 (27%)	16 (19%)
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Congestion	13 (14%)	13 (14%)	11 (12%)	33 (37%)
Foreign body	4 (4%)	2 (2%)	1 (1%)	1 (1%)
Hemorrhage	3 (3%)	5 (6%)	2 (2%)	4 (4%)
Inflammation, suppurative	3 (3%)	–	1 (1%)	2 (2%)
Inflammation, granulomatous	–	6 (7%)	1 (1%)	–
Inflammation, chronic	–	1 (1%)	1 (1%)	–
Inflammation, chronic active	2 (2%)	1 (1%)	1 (1%)	–
Inflammation, subacute	2 (2%)	–	–	–
Metaplasia, osseous	–	–	1 (1%)	–
Alveolus, infiltration cellular, histiocyte	37 (41%)	38 (42%)	42 (47%)	47 (52%)
Artery, inflammation, chronic active	3 (3%)	3 (3%)	1 (1%)	–
Artery, mineral	1 (1%)	–	–	–
Artery, mediastinum, inflammation, chronic active	2 (2%)	–	–	–
Epithelium alveolus, hyperplasia	3 (3%)	2 (2%)	1 (1%)	1 (1%)
Interstitium, inflammation, chronic	–	5 (6%)	–	–
Interstitium, inflammation, chronic active	–	1 (1%)	–	–
Interstitium, mineral	1 (1%)	1 (1%)	1 (1%)	–
Mediastinum, inflammation, suppurative	–	–	1 (1%)	–
Perivascular, infiltration cellular, lymphocyte	–	–	1 (1%)	–
Perivascular, inflammation, chronic active	1 (1%)	–	–	–
Nose	(89)	(90)	(90)	(87)
Foreign body	5 (6%)	2 (2%)	3 (3%)	8 (9%)
Hyperplasia, lymphocyte	–	1 (1%)	–	–
Inflammation, suppurative	10 (11%)	6 (7%)	10 (11%)	17 (20%)
Inflammation, chronic active	–	–	–	2 (2%)
Mineral	–	–	–	1 (1%)
Nasopharyngeal duct, respiratory epithelium, hyperplasia	1 (1%)	–	–	–
Olfactory epithelium, accumulation, hyaline droplet	79 (89%)	88 (98%)	90 (100%)	76 (87%)
Olfactory epithelium, hyperplasia	–	1 (1%)	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Olfactory epithelium, metaplasia, respiratory	3 (3%)	2 (2%)	1 (1%)	4 (5%)
Respiratory epithelium, accumulation, hyaline droplet	3 (3%)	1 (1%)	2 (2%)	3 (3%)
Respiratory epithelium, hyperplasia	3 (3%)	4 (4%)	8 (9%)	7 (8%)
Respiratory epithelium, hyperplasia, goblet cell	1 (1%)	–	–	–
Respiratory epithelium, mineral	1 (1%)	–	–	–
Trachea	(90)	(88)	(88)	(72)
Artery, inflammation, chronic active		1 (1%)	–	–
Artery, mineral	1 (1%)	–	–	–
Epithelium, hyperplasia	1 (1%)	–	–	–
Epithelium, metaplasia, squamous	1 (1%)	–	–	–
Special Senses System				
Eye	(85)	(83)	(81)	(72)
Phthisis bulbi	–	–	1 (1%)	–
Retinal detachment	1 (1%)	–	1 (1%)	–
Anterior chamber, inflammation, acute	4 (5%)	8 (10%)	5 (6%)	1 (1%)
Cornea, fibrosis	1 (1%)	2 (2%)	2 (2%)	4 (6%)
Cornea, inflammation, acute	28 (33%)	18 (22%)	19 (23%)	17 (24%)
Cornea, neovascularization	10 (12%)	14 (17%)	14 (17%)	21 (29%)
Cornea, ulcer	6 (7%)	1 (1%)	–	–
Cornea, epithelium, degeneration	–	–	1 (1%)	2 (3%)
Cornea, epithelium, hyperplasia	13 (15%)	15 (18%)	17 (21%)	20 (28%)
Lens, cataract	–	1 (1%)	–	–
Retina, atrophy	6 (7%)	17 (20%)	17 (21%)	8 (11%)
Retina, degeneration	1 (1%)	–	–	–
Retina, dysplasia	–	–	–	1 (1%)
Harderian gland	(90)	(90)	(90)	(89)
Atrophy	1 (1%)	4 (4%)	2 (2%)	3 (3%)
Degeneration	–	–	1 (1%)	1 (1%)
Degeneration, cystic	2 (2%)	4 (4%)	1 (1%)	–
Hyperplasia	–	–	–	2 (2%)
Infiltration cellular, lymphocyte	–	3 (3%)	–	3 (3%)
Inflammation, suppurative	–	1 (1%)	–	–
Inflammation, granulomatous	–	5 (6%)	2 (2%)	–
Inflammation, acute	2 (2%)	1 (1%)	–	–
Inflammation, chronic	–	1 (1%)	–	1 (1%)
Inflammation, chronic active	2 (2%)	2 (2%)	1 (1%)	1 (1%)
Lacrimal gland	(2)	(1)	(1)	(1)
Metaplasia, harderian gland	2 (100%)	1 (100%)	1 (100%)	1 (100%)
Zymbal's gland	(0)	(0)	(1)	(1)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Urinary System				
Kidney	(90)	(90)	(90)	(87)
Mineral	1 (1%)	–	2 (2%)	–
Necrosis	–	–	1 (1%)	–
Nephropathy, chronic progressive	88 (98%)	90 (100%)	90 (100%)	86 (99%)
Thrombus	1 (1%)	1 (1%)	1 (1%)	–
Artery, inflammation, chronic active	–	–	1 (1%)	–
Artery, mineral	2 (2%)	–	–	–
Pelvis, dilation	1 (1%)	–	1 (1%)	1 (1%)
Pelvis, inflammation, suppurative	–	1 (1%)	1 (1%)	–
Pelvis, urothelium, hyperplasia	–	3 (3%)	1 (1%)	–
Perirenal tissue, hemorrhage	–	–	–	1 (1%)
Perirenal tissue, thrombus	–	–	1 (1%)	
Renal tubule, accumulation, hyaline droplet	–	–	–	1 (1%)
Renal tubule, cyst	18 (20%)	17 (19%)	9 (10%)	6 (7%)
Renal tubule, hyperplasia, atypical	2 (2%)	1 (1%)	3 (3%)	–
Renal tubule, hyperplasia, oncocytic	2 (2%)	–	–	–
Renal tubule, inflammation, suppurative	–	–	1 (1%)	–
Renal tubule, necrosis	–	–	–	1 (1%)
Urothelium, hyperplasia	1 (1%)	–	1 (1%)	–
Urinary bladder	(89)	(83)	(83)	(78)
Dilation	–	1 (1%)	–	–
Hemorrhage	2 (2%)	1 (1%)	2 (2%)	1 (1%)
Inflammation, acute	2 (2%)	1 (1%)	1 (1%)	–
Inflammation, chronic active	–	2 (2%)	–	–
Necrosis	1 (1%)	–	1 (1%)	–
Artery, inflammation, chronic active	–	1 (1%)	–	1 (1%)
Muscularis, degeneration	1 (1%)	–	–	–
Serosa, inflammation, chronic active	–	–	1 (1%)	–
Urothelium, hyperplasia	1 (1%)	4 (5%)	2 (2%)	1 (1%)

[a]Number of animals examined microscopically at the site and the number of animals with lesion.

Appendix D. Summary of Lesions in Female Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

Tables

Table D-1. Summary of the Incidence of Neoplasms in Female Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years D-2
Table D-2. Statistical Analysis of Primary Neoplasms in Female Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years D-8
Table D-3. Historical Incidence of Malignant Schwannoma in Control Female Hsd:Sprague Dawley® SD® Rats D-13
Table D-4. Historical Incidence of Malignant Glioma of the Brain in Control Female Hsd:Sprague Dawley® SD® Rats D-14
Table D-5. Historical Incidence of Pituitary Gland (Pars Distalis) Neoplasms in Control Female Hsd:Sprague Dawley® SD® Rats D-14
Table D-6. Historical Incidence of Hepatocellular Neoplasms in Control Female Hsd:Sprague Dawley® SD® Rats D-15
Table D-7. Historical Incidence of Adrenal Medulla Neoplasms in Control Female Hsd:Sprague Dawley® SD® Rats D-15
Table D-8. Summary of the Incidence of Non-neoplastic Lesions in Female Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years D-16

Table D-1. Summary of the Incidence of Neoplasms in Female Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
Fourteen-week interim evaluation	15	15	15	15
Early deaths				
Accidental death	1	–	–	–
Moribund	30	29	28	16
Natural deaths	11	15	12	13
Survivors				
Died last week of study	1	2	–	–
Terminal euthanasia	47	44	50	61
Animals examined microscopically	100	100	100	100
Fourteen-week Interim Evaluation				
Integumentary System				
Mammary gland	(10)	(10)	(10)	(10)
Adenocarcinoma	–	–	–	1 (10%)
Skin	(10)	(10)	(10)	(10)
Systems Examined with No Neoplasms Observed				
Alimentary System				
Cardiovascular System				
Endocrine System				
General Body System				
Genital System				
Hematopoietic System				
Musculoskeletal System				
Nervous System				
Respiratory System				
Special Senses System				
Urinary System				
Two-year Study				
Alimentary System				
Esophagus	(90)	(90)	(90)	(90)
Intestine large, cecum	(84)	(82)	(86)	(80)
Intestine large, colon	(89)	(89)	(88)	(88)
Intestine large, rectum	(90)	(88)	(87)	(88)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Granular cell tumor benign	1 (1%)	–	–	–
Intestine small, duodenum	(88)	(86)	(87)	(85)
Intestine small, ileum	(86)	(83)	(84)	(83)
Intestine small, jejunum	(83)	(81)	(84)	(79)
Leiomyosarcoma	1 (1%)	–	–	–
Liver	(90)	(90)	(90)	(90)
Carcinoma, metastatic, adrenal cortex	–	–	1 (1%)	
Hepatocellular adenoma	7 (8%)	2 (2%)	2 (2%)	1 (1%)
Hepatocellular carcinoma	–	–	–	1 (1%)
Mesentery	(4)	(3)	(11)	(4)
Adenocarcinoma, metastatic, uterus	1 (25%)	–	–	–
Oral mucosa	(1)	(1)	(0)	(0)
Squamous cell carcinoma	1 (100%)	–	–	–
Pancreas	(90)	(90)	(90)	(89)
Salivary glands	(90)	(90)	(90)	(90)
Parotid gland, squamous cell carcinoma	–	–	–	1 (1%)
Stomach, forestomach	(90)	(90)	(90)	(90)
Sarcoma	1 (1%)	–	–	1 (1%)
Squamous cell papilloma	–	–	–	1 (1%)
Stomach, glandular	(90)	(90)	(89)	(88)
Sarcoma, metastatic, stomach, forestomach	1 (1%)	–	–	–
Tongue	(1)	(0)	(0)	(0)
Squamous cell carcinoma	1 (100%)	–	–	–
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Blood vessel	(0)	(0)	(0)	(1)
Heart	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, mammary gland	–	–	1 (1%)	–
Endocardium, schwannoma malignant	–	1 (1%)	–	2 (2%)
Myocardium, schwannoma malignant	–	1 (1%)	–	–
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(90)
Adenoma	1 (1%)	2 (2%)	2 (2%)	1 (1%)
Carcinoma	1 (1%)	–	1 (1%)	–
Adrenal medulla	(86)	(89)	(87)	(88)
Pheochromocytoma benign	1 (1%)	7 (8%)	3 (3%)	4 (5%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pheochromocytoma complex	–	–	1 (1%)	–
Pheochromocytoma malignant	–	2 (2%)	1 (1%)	–
Islets, pancreatic	(90)	(89)	(90)	(88)
Adenoma	5 (6%)	4 (4%)	5 (6%)	4 (5%)
Carcinoma	2 (2%)	2 (2%)	–	2 (2%)
Parathyroid gland	(87)	(80)	(85)	(85)
Pituitary gland	(90)	(89)	(89)	(90)
Pars distalis, adenoma	42 (47%)	40 (45%)	30 (34%)	40 (44%)
Pars distalis, adenoma, multiple	1 (1%)	1 (1%)	–	–
Pars distalis, carcinoma	1 (1%)	1 (1%)	1 (1%)	–
Thyroid gland	(90)	(90)	(90)	(89)
Bilateral, C-cell, adenoma	–	–	–	1 (1%)
Bilateral, C-cell, carcinoma	–	–	1 (1%)	–
C-cell, adenoma	6 (7%)	9 (10%)	3 (3%)	7 (8%)
C-cell, carcinoma	–	3 (3%)	2 (2%)	2 (2%)
Follicular cell, adenoma	–	–	1 (1%)	–
Follicular cell, carcinoma	1 (1%)	1 (1%)	–	–
General Body System				
Tissue NOS	(8)	(11)	(8)	(6)
Abdominal, schwannoma malignant	1 (13%)	–	–	–
Abdominal, fat, lipoma	–	–	1 (13%)	–
Genital System				
Clitoral gland	(87)	(88)	(89)	(86)
Carcinoma	–	–	1 (1%)	–
Schwannoma malignant	–	–	–	1 (1%)
Ovary	(90)	(90)	(89)	(90)
Cystadenocarcinoma	–	1 (1%)	–	–
Cystadenoma	1 (1%)	–	–	–
Granulosa cell tumor benign	1 (1%)	–	–	–
Granulosa cell tumor malignant	2 (2%)	–	–	–
Sertoli cell tumor benign	1 (1%)	–	–	–
Periovarian tissue, schwannoma malignant	–	–	–	1 (1%)
Rete ovarii, adenoma	–	–	1 (1%)	1 (1%)
Oviduct	(1)	(0)	(0)	(0)
Uterus	(90)	(90)	(90)	(90)
Adenocarcinoma	3 (3%)	–	–	3 (3%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adenoma	–	–	1 (1%)	1 (1%)
Carcinoma	–	1 (1%)	–	–
Hemangiosarcoma	2 (2%)	–	–	–
Leiomyosarcoma	–	–	2 (2%)	–
Polyp, glandular	–	2 (2%)	2 (2%)	–
Polyp stromal	15 (17%)	11 (12%)	9 (10%)	12 (13%)
Polyp stromal, multiple	1 (1%)	4 (4%)	4 (4%)	5 (6%)
Schwannoma malignant	1 (1%)	–	–	–
Squamous cell carcinoma	–	–	1 (1%)	–
Cervix, leiomyosarcoma	1 (1%)	–	–	–
Cervix, polyp stromal	–	–	1 (1%)	–
Cervix, schwannoma malignant	1 (1%)	–	2 (2%)	–
Vagina	(2)	(1)	(0)	(1)
Polyp, stromal	–	1 (100%)	–	–
Schwannoma malignant	1 (50%)	–	–	–
Schwannoma malignant, metastatic, uterus	1 (50%)	–	–	–
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)
Lymph node	(13)	(8)	(11)	(20)
Iliac, adenocarcinoma, metastatic, mammary gland	–	–	–	1 (5%)
Lymph node, mandibular	(90)	(90)	(89)	(90)
Lymph node, mesenteric	(90)	(90)	(90)	(89)
Spleen	(90)	(90)	(90)	(90)
Hemangiosarcoma	1 (1%)	–	–	–
Thymus	(87)	(83)	(87)	(87)
Thymoma benign	1 (1%)	–	1 (1%)	–
Thymoma malignant	1 (1%)	–	–	–
Integumentary System				
Mammary gland	(90)	(90)	(90)	(90)
Adenocarcinoma	9 (10%)	7 (8%)	6 (7%)	3 (3%)
Adenocarcinoma, multiple	1 (1%)	1 (1%)	–	1 (1%)
Adenoma	4 (4%)	4 (4%)	1 (1%)	2 (2%)
Adenoma, multiple	4 (4%)	–	–	–
Fibroadenoma	34 (38%)	40 (44%)	34 (38%)	32 (36%)
Fibroadenoma, multiple	29 (32%)	21 (23%)	29 (32%)	30 (33%)
Skin	(90)	(90)	(90)	(90)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Keratoacanthoma	–	1 (1%)	2 (2%)	1 (1%)
Squamous cell carcinoma, metastatic, salivary glands	–	–	–	1 (1%)
Subcutaneous tissue, fibroma	2 (2%)	–	1 (1%)	3 (3%)
Subcutaneous tissue, lipoma	–	–	1 (1%)	2 (2%)
Subcutaneous tissue, malignant fibrous histiocytoma	1 (1%)	–	–	–
Subcutaneous tissue, sarcoma	2 (2%)	–	–	–
Subcutaneous tissue, squamous cell carcinoma	–	2 (2%)	–	–
Vulva, squamous cell carcinoma	–	–	–	1 (1%)
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Vertebra, chondroma	–	–	1 (1%)	–
Skeletal muscle	(90)	(90)	(90)	(90)
Nervous System				
Brain	(90)	(90)	(90)	(90)
Carcinoma, metastatic, pituitary gland	1 (1%)	–	–	–
Glioma malignant	–	3 (3%)	–	–
Meningioma malignant	–	–	–	1 (1%)
Neuroblastoma	–	1 (1%)	–	–
Sarcoma	–	1 (1%)	–	–
Meninges, granular cell tumor benign	1 (1%)	1 (1%)	–	2 (2%)
Pineal gland, pinealoma	–	1 (1%)	–	–
Nerve trigeminal	(84)	(84)	(85)	(84)
Squamous cell carcinoma, metastatic, salivary glands	–	–	–	1 (1%)
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Peripheral nerve, tibial	(90)	(90)	(89)	(89)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Spinal cord, lumbar	(90)	(90)	(89)	(90)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Trigeminal ganglion	(81)	(77)	(81)	(75)
Carcinoma, metastatic, pituitary gland	–	–	1 (1%)	–
Squamous cell carcinoma, metastatic, salivary glands	–	–	–	1 (1%)
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Adenocarcinoma, metastatic, mammary gland	–	2 (2%)	1 (1%)	1 (1%)
Carcinoma, metastatic, adrenal cortex	–	–	1 (1%)	–
Carcinoma, metastatic, thyroid gland	–	1 (1%)	1 (1%)	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Carcinoma, metastatic, uncertain primary site	–	1 (1%)	–	–
Nose	(90)	(89)	(90)	(89)
Trachea	(89)	(88)	(89)	(89)
Carcinoma, metastatic, thyroid gland	–	–	1 (1%)	–
Special Senses System				
Ear	(0)	(0)	(1)	(1)
Neural crest tumor	–	–	1 (100%)	1 (100%)
Eye	(88)	(86)	(88)	(86)
Sarcoma	–	1 (1%)	–	–
Harderian gland	(90)	(90)	(90)	(90)
Urinary System				
Kidney	(90)	(90)	(90)	(89)
Bilateral, renal tubule, carcinoma	1 (1%)	–	2 (2%)	–
Renal tubule, adenoma	1 (1%)	–	1 (1%)	–
Urinary bladder	(88)	(88)	(90)	(90)
Leiomyosarcoma	1 (1%)	–	–	–
Schwannoma malignant, metastatic, tissue NOS	1 (1%)	–	–	–
Systemic Lesions				
Multiple organsb	(90)	(90)	(90)	(90)
Histiocytic sarcoma	–	–	2 (2%)	–
Leukemia mononuclear	–	3 (3%)	–	–
Lymphoma malignant	5 (6%)	2 (2%)	4 (4%)	3 (3%)
Neoplasm Summary				
Total animals with primary neoplasmsc				
Fourteen-week interim evaluation	–	–	–	1
Two-year study	89	88	82	85
Total primary neoplasms				
Fourteen-week interim evaluation	–	–	–	1
Two-year study	202	185	164	174
Total animals with benign neoplasms				
Two-year study	82	84	78	79
Total benign neoplasms				
Two-year study	159	151	136	150
Total animals with malignant neoplasms				
Fourteen-week interim evaluation	–	–	–	1
Two-year study	37	27	24	22

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Total malignant neoplasms				
Fourteen-week interim evaluation	–	–	–	1
Two-year study	43	34	27	23
Total animals with metastatic neoplasms				
Two-year study	5	4	6	2
Total metastatic neoplasms				
Two-year study	5	4	11	5
Total animals with malignant neoplasms-uncertain primary site				
Two-year study	–	1	–	–
Total animals with uncertain neoplasms-benign or malignant				
Two-year study	–	–	1	1
Total uncertain neoplasms benign or malignant				
Two-year study	–	–	1	1

[a]Number of animals examined microscopically at the site and the number of animals with neoplasm.
[b]Number of animals with any tissue examined microscopically.
[c]Primary neoplasms: all neoplasms except metastatic neoplasms.

Table D-2. Statistical Analysis of Primary Neoplasms in Female Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Adrenal Medulla: Benign Pheochromocytoma				
Overall rate[a]	1/86 (1%)	7/89 (8%)	3/87 (3%)	4/88 (5%)
Rate per litters[b]	1/35 (3%)	7/34 (21%)	3/35 (9%)	4/35 (11%)
Adjusted rate[c]	1.5%	9.6%	4.4%	5.2%
Terminal rate[d]	1/45 (2%)	5/44 (11%)	3/48 (6%)	4/60 (7%)
First incidence (days)	737 (T)	464	737 (T)	737 (T)
Rao-Scott adjusted Poly-3 test[e]	P = 0.466	P = 0.059	P = 0.322	P = 0.248
Litter C-A/Fisher's test[f]	P = 0.379	P = 0.025	P = 0.307	P = 0.178
Adrenal Medulla: Benign, Complex, or Malignant Pheochromocytoma				
Overall rate	1/86 (1%)	9/89 (10%)	5/87 (6%)	4/88 (5%)
Rate per litters	1/35 (3%)	9/34 (26%)	5/35 (14%)	4/35 (11%)
Adjusted rate	1.5%	12.3%	7.2%	5.2%
Terminal rate	1/45 (2%)	7/44 (16%)	4/48 (8%)	4/60 (7%)
First incidence (days)	737 (T)	464	652	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.546	P = 0.022	P = 0.126	P = 0.242
Litter C-A/Fisher's test	P = 0.457	P = 0.006	P = 0.099	P = 0.178

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Liver: Hepatocellular Adenoma				
Overall rate	7/90 (8%)	2/90 (2%)	2/90 (2%)	1/90 (1%)
Rate per litters	6/35 (17%)	2/34 (6%)	2/35 (6%)	1/35 (3%)
Adjusted rate	10.1%	2.7%	2.8%	1.3%
Terminal rate	6/48 (13%)	1/45 (2%)	2/50 (4%)	1/61 (2%)
First incidence (days)	707	493	737 (T)	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.042N	P = 0.118N	P = 0.125N	P = 0.052N
Litter C-A/Fisher's test	P = 0.039N	P = 0.139N	P = 0.130N	P = 0.053N
Liver: Hepatocellular Adenoma or Hepatocellular Carcinoma				
Overall rate	7/90 (8%)	2/90 (2%)	2/90 (2%)	2/90 (2%)
Rate per litters	6/35 (17%)	2/34 (6%)	2/35 (6%)	2/35 (6%)
Adjusted rate	10.1%	2.7%	2.8%	2.5%
Terminal rate	6/48 (13%)	1/45 (2%)	2/50 (4%)	2/61 (3%)
First incidence (days)	707	493	737 (T)	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.089N	P = 0.112N	P = 0.118N	P = 0.096N
Litter C-A/Fisher's test	P = 0.108N	P = 0.139N	P = 0.130N	P = 0.130N
Mammary Gland: Fibroadenoma				
Overall rate	63/90 (70%)	61/90 (68%)	63/90 (70%)	62/90 (69%)
Rate per litters	31/35 (89%)	32/34 (94%)	33/35 (94%)	31/35 (89%)
Adjusted rate	77.0%	73.3%	75.2%	73.1%
Terminal rate	36/48 (75%)	33/45 (73%)	36/50 (72%)	45/61 (74%)
First incidence (days)	464	300	268	492
Rao-Scott adjusted Poly-3 test	P = 0.356N	P = 0.358N	P = 0.462N	P = 0.344N
Litter C-A/Fisher's test	P = 0.505N	P = 0.351	P = 0.337	P = 0.645
Mammary Gland: Adenoma				
Overall rate	8/90 (9%)	4/90 (4%)	1/90 (1%)	2/90 (2%)
Rate per litters	7/35 (20%)	4/34 (12%)	1/35 (3%)	2/35 (6%)
Adjusted rate	11.3%	5.4%	1.4%	2.5%
Terminal rate	5/48 (10%)	1/45 (2%)	1/50 (2%)	2/61 (3%)
First incidence (days)	524	647	737 (T)	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.035N	P = 0.207N	P = 0.039N	P = 0.063N
Litter C-A/Fisher's test	P = 0.034N	P = 0.274N	P = 0.027N	P = 0.075N
Mammary Gland: Fibroadenoma or Adenoma				
Overall rate	64/90 (71%)	62/90 (69%)	63/90 (70%)	62/90 (69%)
Rate per litters	31/35 (89%)	32/34 (94%)	33/35 (94%)	31/35 (89%)
Adjusted rate	77.7%	74.5%	75.2%	73.1%

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Terminal rate	36/48 (75%)	33/45 (73%)	36/50 (72%)	45/61 (74%)
First incidence (days)	464	300	268	492
Rao-Scott adjusted Poly-3 test	P = 0.307N	P = 0.386N	P = 0.427N	P = 0.312N
Litter C-A/Fisher's test	P = 0.505N	P = 0.351	P = 0.337	P = 0.645
Mammary Gland: Adenocarcinoma				
Overall rate	10/90 (11%)	8/90 (9%)	6/90 (7%)	4/90 (4%)
Rate per litters	9/35 (26%)	8/34 (24%)	5/35 (14%)	4/35 (11%)
Adjusted rate	14.2%	10.4%	8.2%	4.9%
Terminal rate	6/48 (13%)	1/45 (2%)	3/50 (6%)	2/61 (3%)
First incidence (days)	622	300	493	305
Rao-Scott adjusted Poly-3 test	P = 0.042N	P = 0.330N	P = 0.196N	P = 0.055N
Litter C-A/Fisher's test	P = 0.059N	P = 0.528N	P = 0.185N	P = 0.109N
Mammary Gland: Adenoma or Adenocarcinoma				
Overall rate	16/90 (18%)	12/90 (13%)	7/90 (8%)	6/90 (7%)
Rate per litters	13/35 (37%)	12/34 (35%)	5/35 (14%)	6/35 (17%)
Adjusted rate	22.2%	15.5%	9.5%	7.4%
Terminal rate	9/48 (19%)	2/45 (4%)	4/50 (8%)	4/61 (7%)
First incidence (days)	524	300	493	305
Rao-Scott adjusted Poly-3 test	P = 0.009N	P = 0.214N	P = 0.041N	P = 0.014N
Litter C-A/Fisher's test	P = 0.017N	P = 0.536N	P = 0.027N	P = 0.053N
Mammary Gland: Fibroadenoma, Adenoma, or Adenocarcinoma				
Overall rate	66/90 (73%)	68/90 (76%)	66/90 (73%)	66/90 (73%)
Rate per litters	31/35 (89%)	33/34 (97%)	34/35 (97%)	32/35 (91%)
Adjusted rate	79.6%	78.7%	78.1%	76.6%
Terminal rate	36/48 (75%)	33/45 (73%)	37/50 (74%)	47/61 (77%)
First incidence (days)	464	300	268	305
Rao-Scott adjusted Poly-3 test	P = 0.352N	P = 0.518N	P = 0.478N	P = 0.393N
Litter C-A/Fisher's test	P = 0.519	P = 0.187	P = 0.178	P = 0.500
Pancreatic Islets: Adenoma				
Overall rate	5/90 (6%)	4/89 (4%)	5/90 (6%)	4/88 (5%)
Rate per litters	4/35 (11%)	4/34 (12%)	5/35 (14%)	4/35 (11%)
Adjusted rate	7.2%	5.5%	6.9%	5.1%
Terminal rate	5/48 (10%)	2/45 (4%)	3/50 (6%)	4/61 (7%)
First incidence (days)	737 (T)	647	627	737 (T)
Rao-Scott adjusted Poly-3 test	P = 0.405N	P = 0.460N	P = 0.585N	P = 0.424N
Litter C-A/Fisher's test	P = 0.558	P = 0.629	P = 0.500	P = 0.645

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pancreatic Islets: Adenoma or Carcinoma				
Overall rate	7/90 (8%)	6/89 (7%)	5/90 (6%)	6/88 (7%)
Rate per litters	6/35 (17%)	6/34 (18%)	5/35 (14%)	6/35 (17%)
Adjusted rate	10.1%	8.2%	6.9%	7.6%
Terminal rate	6/48 (13%)	3/45 (7%)	3/50 (6%)	5/61 (8%)
First incidence (days)	711	647	627	702
Rao-Scott adjusted Poly-3 test	P = 0.367N	P = 0.451N	P = 0.347N	P = 0.404N
Litter C-A/Fisher's test	P = 0.538N	P = 0.603	P = 0.500N	P = 0.624
Pituitary Gland (Pars Distalis): Adenoma				
Overall rate	43/90 (48%)	41/89 (46%)	30/89 (34%)	40/90 (44%)
Rate per litters	28/35 (80%)	26/34 (76%)	21/35 (60%)	25/35 (71%)
Adjusted rate	57.1%	52.9%	39.5%	49.0%
Terminal rate	28/48 (58%)	20/45 (44%)	16/50 (32%)	31/61 (51%)
First incidence (days)	464	578	493	626
Rao-Scott adjusted Poly-3 test	P = 0.156N	P = 0.360N	P = 0.026N	P = 0.204N
Litter C-A/Fisher's test	P = 0.203N	P = 0.474N	P = 0.058N	P = 0.289N
Pituitary Gland (Pars Distalis): Adenoma or Carcinoma				
Overall rate	44/90 (49%)	42/89 (47%)	31/89 (35%)	40/90 (44%)
Rate per litters	29/35 (83%)	26/34 (76%)	21/35 (60%)	25/35 (71%)
Adjusted rate	57.9%	54.1%	40.7%	49.0%
Terminal rate	28/48 (58%)	20/45 (44%)	16/50 (32%)	31/61 (51%)
First incidence (days)	464	578	493	626
Rao-Scott adjusted Poly-3 test	P = 0.131N	P = 0.381N	P = 0.030N	P = 0.180N
Litter C-A/Fisher's test	P = 0.144N	P = 0.360N	P = 0.031N	P = 0.197N
Skin (Subcutaneous Tissue): Fibroma, Sarcoma, or Malignant Fibrous Histiocytoma				
Overall rate	5/90 (6%)	0/90 (0%)	1/90 (1%)	3/90 (3%)
Rate per litters	5/35 (14%)	0/34 (0%)	1/35 (3%)	3/35 (9%)
Adjusted rate	7.0%	0%	1.4%	3.7%
Terminal rate	1/48 (2%)	0/45 (0%)	1/50 (2%)	1/61 (2%)
First incidence (days)	268	–[g]	737 (T)	550
Rao-Scott adjusted Poly-3 test	P = 0.413N	P = 0.057N	P = 0.142N	P = 0.335N
Litter C-A/Fisher's test	P = 0.426N	P = 0.029N	P = 0.099N	P = 0.355N
Thyroid Gland (C-cell): Adenoma				
Overall rate	6/90 (7%)	9/90 (10%)	3/90 (3%)	8/89 (9%)
Rate per litters	6/35 (17%)	9/34 (26%)	3/35 (9%)	7/35 (20%)
Adjusted rate	8.5%	12.1%	4.2%	10.1%

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Terminal rate	3/48 (6%)	6/45 (13%)	2/50 (4%)	6/61 (10%)
First incidence (days)	608	578	674	669
Rao-Scott adjusted Poly-3 test	P = 0.548N	P = 0.326	P = 0.232N	P = 0.473
Litter C-A/Fisher's test	P = 0.507N	P = 0.259	P = 0.239N	P = 0.500
Thyroid Gland: (C-cell): Adenoma or Carcinoma				
Overall rate	6/90 (7%)	11/90 (12%)	6/90 (7%)	10/89 (11%)
Rate per litters	6/35 (17%)	11/34 (32%)	6/35 (17%)	9/35 (26%)
Adjusted rate	8.5%	14.8%	8.3%	12.6%
Terminal rate	3/48 (6%)	8/45 (18%)	4/50 (8%)	8/61 (13%)
First incidence (days)	608	578	643	669
Rao-Scott adjusted Poly-3 test	P = 0.390	P = 0.175	P = 0.579N	P = 0.288
Litter C-A/Fisher's test	P = 0.402	P = 0.118	P = 0.624	P = 0.281
Uterus: Polyp Stromal				
Overall rate	16/90 (18%)	17/90 (19%)	16/90 (18%)	17/90 (19%)
Rate per litters	11/35 (31%)	13/34 (38%)	15/35 (43%)	13/35 (37%)
Adjusted rate	22.7%	23.0%	22.0%	21.2%
Terminal rate	14/48 (29%)	13/45 (29%)	12/50 (24%)	14/61 (23%)
First incidence (days)	531	605	631	587
Rao-Scott adjusted Poly-3 test	P = 0.437N	P = 0.552	P = 0.532N	P = 0.490N
Litter C-A/Fisher's test	P = 0.374	P = 0.367	P = 0.229	P = 0.401
All Organs: Malignant Lymphoma				
Overall rate	5/90 (6%)	2/90 (2%)	4/90 (4%)	3/90 (3%)
Rate per litters	5/35 (14%)	2/34 (6%)	4/35 (11%)	3/35 (9%)
Adjusted rate	7.0%	2.7%	5.5%	3.7%
Terminal rate	0/48 (0%)	1/45 (2%)	1/50 (2%)	0/61 (0%)
First incidence (days)	268	706	483	587
Rao-Scott adjusted Poly-3 test	P = 0.339N	P = 0.209N	P = 0.472N	P = 0.297N
Litter C-A/Fisher's test	P = 0.382N	P = 0.226N	P = 0.500N	P = 0.355N
All Organs: Benign Neoplasms				
Overall rate	82/90 (91%)	84/90 (93%)	78/90 (87%)	79/90 (88%)
Rate per litters	35/35 (100%)	34/34 (100%)	35/35 (100%)	35/35 (100%)
Adjusted rate	97.1%	97.7%	90.7%	91.1%
Terminal rate	48/48 (100%)	44/45 (98%)	44/50 (88%)	55/61 (90%)
First incidence (days)	464	300	268	492
Rao-Scott adjusted Poly-3 test	P = 0.039N	P = 0.602	P = 0.098N	P = 0.113N
Litter C-A/Fisher's test	–[h]	–	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
All Organs: Malignant Neoplasms				
Overall rate	37/90 (41%)	28/90 (31%)	24/90 (27%)	22/90 (24%)
Rate per litters	25/35 (71%)	19/34 (56%)	19/35 (54%)	20/35 (57%)
Adjusted rate	48.1%	35.1%	31.0%	26.6%
Terminal rate	21/48 (44%)	10/45 (22%)	10/50 (20%)	14/61 (23%)
First incidence (days)	268	300	483	305
Rao-Scott adjusted Poly-3 test	P = 0.006N	P = 0.073N	P = 0.026N	P = 0.005N
Litter C-A/Fisher's test	P = 0.185N	P = 0.137N	P = 0.108N	P = 0.159N
All Organs: Benign or Malignant Neoplasms				
Overall rate	89/90 (99%)	88/90 (98%)	82/90 (91%)	85/90 (94%)
Rate per litters	35/35 (100%)	34/34 (100%)	35/35 (100%)	35/35 (100%)
Adjusted rate	100%	98.7%	93.0%	96.4%
Terminal rate	48/48 (100%)	44/45 (98%)	44/50 (88%)	59/61 (97%)
First incidence (days)	268	300	268	305
Rao-Scott adjusted Poly-3 test	P = 0.127N	P = 0.582N	P = 0.051N	P = 0.203N
Litter C-A/Fisher's test	–	–	–	–

(T) = terminal euthanasia.
[a]Number of neoplasm-bearing animals/number of animals examined. Denominator is number of animals examined microscopically for adrenal gland, liver, pancreatic islets, pituitary gland, and thyroid gland; for other tissues, denominator is number of animals necropsied.
[b]Number of litters with tumor-bearing animals/number of litters examined at site.
[c]Poly-3 estimated neoplasm incidence after adjustment for intercurrent mortality.
[d]Observed incidence at terminal euthanasia.
[e]Beneath the sham control incidence is the P value associated with the trend test. Beneath the exposed group incidence are the P values corresponding to pairwise comparisons between the sham controls and that exposed group. The Poly-3 test accounts for differential mortality in animals that do not reach terminal euthanasia. The Rao-Scott test adjusts the Poly-3 test (which accounts for differential mortality in animals that do not reach terminal euthanasia) for within-litter correlation. A negative trend or a lower incidence in an exposure group is indicated by N.
[f]The Litter Cochran-Armitage and Fishers exact tests directly compare the litter incidence rates.
[g]Not applicable; no neoplasms in animal group.
[h]Value of statistic cannot be computed.

Table D-3. Historical Incidence of Malignant Schwannoma in Control Female Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Heart: Malignant Schwannoma	All Organs: Malignant Schwannoma
Historical Incidence: All Studies		
p-Chloro-α,α,α-trifluorotoluene (January 2011)	0/50	0/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	0/49	2/50
Indole-3-carbinol (March 2007)	0/50	2/50
Radiofrequency radiation (September 2012)	0/90	4/90
Overall Historical Incidence		
Total (%)	0/239	8/240 (3.3%)

Study (Study Start)	Heart: Malignant Schwannoma	All Organs: Malignant Schwannoma
Mean ± standard deviation	–	3.1% ± 2.1%
Range	–	0%–4%

[a]Data as of November 2017.

Table D-4. Historical Incidence of Malignant Glioma of the Brain in Control Female Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Incidence in Controls
Historical Incidence: All Studies	
p-Chloro-α,α,α-trifluorotoluene (January 2011)	1/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	0/50
Radiofrequency radiation (September 2012)	0/90
Overall Historical Incidence	
Total (%)	1/190 (0.5%)
Mean ± standard deviation	0.7% ± 1.2%
Range	0%–2%

[a]Data as of November 2017; due to differences in sectioning method, indole-3-carbinol was excluded from evaluation of brain lesions.

Table D-5. Historical Incidence of Pituitary Gland (Pars Distalis) Neoplasms in Control Female Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Adenoma	Carcinoma	Adenoma or Carcinoma
Historical Incidence: All Studies			
p-Chloro-α,α,α-trifluorotoluene (January 2011)	18/50	0/50	18/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	19/50	0/50	19/50
Indole-3-carbinol (March 2007)	18/50	0/50	18/50
Radiofrequency radiation (September 2012)	43/90	1/90	44/90
Overall Historical Incidence			
Total (%)	98/240 (40.8%)	1/240 (0.4%)	99/240 (41.3%)
Mean ± standard deviation	39.4% ± 5.6%	0.3% ± 0.6%	39.7% ± 6.2%
Range	36%–48%	0%–1%	36%–49%

[a]Data as of November 2017.

Table D-6. Historical Incidence of Hepatocellular Neoplasms in Control Female Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Adenoma	Carcinoma	Adenoma or Carcinoma
Historical Incidence: All Studies			
p-Chloro-α,α,α-trifluorotoluene (January 2011)	2/50	0/50	2/50
2-Hydroxy-4-methoxybenzophenone (November 2010)	0/50	0/50	0/50
Indole-3-carbinol (March 2007)	2/50	0/50	2/50
Radiofrequency radiation (September 2012)	7/90	0/90	7/90
Overall Historical Incidence			
Total (%)	11/240 (4.6%)	0/240	11/240 (4.6%)
Mean ± standard deviation	3.9% ± 3.2%	–	3.9% ± 3.2%
Range	0%–8%	–	0%–8%

[a]Data as of November 2017.

Table D-7. Historical Incidence of Adrenal Medulla Neoplasms in Control Female Hsd:Sprague Dawley® SD® Rats[a]

Study (Study Start)	Benign Pheochromocytoma	Malignant Pheochromocytoma	Complex Pheochromocytoma	Benign, Malignant, or Complex Pheochromocytoma
Historical Incidence: All Studies				
p-Chloro-α,α,α-trifluorotoluene (January 2011)	0/49	0/49	0/49	0/49
2-Hydroxy-4-methoxybenzophenone (November 2010)	3/50	2/50	0/50	5/50
Indole-3-carbinol (March 2007)	0/50	0/50	0/50	0/50
Radiofrequency radiation (September 2012)	1/86	0/86	0/86	1/86
Overall Historical Incidence				
Total (%)	4/235 (1.7%)	2/235 (0.9%)	0/235	6/235 (2.6%)
Mean ± standard deviation	1.8% ± 2.9%	1.0% ± 2.0%	–	2.8% ± 4.8%
Range	0%-6%	0%-4%	–	0%-10%

[a]Data as of November 2017.

Table D-8. Summary of the Incidence of Non-neoplastic Lesions in Female Rats Exposed to CDMA-modulated Cell Phone RFR for Two Years[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Disposition Summary				
Animals initially in study	105	105	105	105
Fourteen-week interim evaluation	15	15	15	15
Early deaths				
Accidental death	1	–	–	–
Moribund	30	29	28	16
Natural deaths	11	15	12	13
Survivors				
Died last week of study	1	2	–	–
Terminal euthanasia	47	44	50	61
Animals examined microscopically	100	100	100	100
Fourteen-week Interim Evaluation				
Alimentary System				
Esophagus	(10)	(10)	(10)	(10)
Intestine large, cecum	(10)	(10)	(10)	(10)
Intestine large, colon	(10)	(10)	(10)	(10)
Inflammation, chronic active	–	–	–	1 (10%)
Intestine large, rectum	(10)	(10)	(10)	(10)
Lymphoid tissue, hyperplasia	1 (10%)	–	2 (20%)	1 (10%)
Intestine small, duodenum	(10)	(10)	(10)	(10)
Intestine small, ileum	(10)	(10)	(10)	(10)
Intestine small, jejunum	(10)	(10)	(10)	(10)
Liver	(10)	(10)	(10)	(10)
Infiltration cellular, mixed cell	1 (10%)	1 (10%)	–	2 (20%)
Inflammation, chronic active	–	–	–	1 (10%)
Pancreas	(10)	(10)	(10)	(10)
Inflammation, chronic active	–	–	–	1 (10%)
Salivary glands	(10)	(10)	(10)	(10)
Inflammation, chronic active	–	–	–	1 (10%)
Stomach, forestomach	(10)	(10)	(10)	(10)
Stomach, glandular	(10)	(10)	(10)	(10)
Cardiovascular System				
Aorta	(10)	(10)	(10)	(10)
Inflammation, chronic active	–	–	–	1 (10%)
Heart	(10)	(10)	(10)	(10)
Cardiomyopathy	–	–	2 (20%)	2 (20%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Endocardium, inflammation, chronic active	–	–	–	1 (10%)
Ventricle right, cardiomyopathy	–	–	1 (10%)	1 (10%)
Endocrine System				
Adrenal cortex	(10)	(10)	(10)	(10)
Adrenal medulla	(10)	(10)	(10)	(10)
Islets, pancreatic	(10)	(10)	(10)	(10)
Parathyroid gland	(10)	(10)	(9)	(9)
Pituitary gland	(10)	(10)	(10)	(10)
Pars distalis, cyst	2 (20%)	–	–	–
Pars intermedia, cyst	1 (10%)	–	2 (20%)	–
Rathke's cleft, cyst	–	1 (10%)	–	–
Thyroid gland	(10)	(10)	(10)	(10)
Ectopic thymus	–	–	1 (10%)	–
Genital System				
Clitoral gland	(10)	(10)	(10)	(10)
Inflammation, chronic active	4 (40%)	4 (40%)	4 (40%)	2 (20%)
Ovary	(10)	(10)	(10)	(10)
Follicle, cyst	1 (10%)	–	1 (10%)	–
Uterus	(10)	(10)	(10)	(10)
Hematopoietic System				
Bone marrow	(10)	(10)	(10)	(10)
Lymph node	(0)	(0)	(1)	(0)
Pigment	–	–	1 (100%)	–
Lymph node, mandibular	(10)	(10)	(10)	(9)
Lymph node, mesenteric	(10)	(10)	(10)	(10)
Spleen	(10)	(10)	(10)	(10)
Extramedullary hematopoiesis	–	–	–	1 (10%)
Thymus	(10)	(10)	(10)	(10)
Hemorrhage	1 (10%)	2 (20%)	1 (10%)	–
Hyperplasia, epithelial	–	–	–	1 (10%)
Integumentary System				
Mammary gland	(10)	(10)	(10)	(10)
Skin	(10)	(10)	(10)	(10)
Inflammation, chronic active	–	–	–	1 (10%)
Musculoskeletal System				
Bone	(10)	(10)	(10)	(10)
Skeletal muscle	(10)	(10)	(10)	(10)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Inflammation, chronic active	–	–	–	1 (10%)
Respiratory System				
Lung	(10)	(10)	(10)	(10)
Congestion	–	–	–	1 (10%)
Alveolus, infiltration cellular, histiocyte	2 (20%)	–	–	–
Nose	(10)	(10)	(10)	(10)
Trachea	(10)	(10)	(10)	(10)
Special Senses System				
Eye	(10)	(10)	(10)	(10)
Conjunctiva, inflammation, chronic active	–	–	–	1 (10%)
Harderian gland	(10)	(10)	(10)	(10)
Infiltration cellular, lymphocyte	1 (10%)	–	–	–
Inflammation, chronic	–	–	–	1 (10%)
Urinary System				
Kidney	(10)	(10)	(10)	(10)
Infiltration cellular, mixed cell	–	–	–	1 (10%)
Inflammation, chronic active	–	–	–	1 (10%)
Nephropathy, chronic progressive	3 (30%)	3 (30%)	4 (40%)	4 (40%)
Urinary bladder	(10)	(10)	(10)	(10)
Systems Examined with No Lesions Observed				
General Body System				
Nervous System				
Two-year Study				
Alimentary System				
Esophagus	(90)	(90)	(90)	(90)
Dilation	–	2 (2%)	–	–
Inflammation, acute	–	–	–	1 (1%)
Muscularis, degeneration	–	1 (1%)	–	–
Intestine large, cecum	(84)	(82)	(86)	(80)
Ulcer	–	–	1 (1%)	–
Artery, inflammation, chronic active	–	2 (2%)	–	–
Intestine large, colon	(89)	(89)	(88)	(88)
Diverticulum	–	–	1 (1%)	–
Artery, inflammation, chronic active	–	1 (1%)	–	–
Intestine large, rectum	(90)	(88)	(87)	(88)
Hyperplasia, lymphocyte	–	–	3 (3%)	–
Artery, inflammation, chronic active	–	1 (1%)	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Intestine small, duodenum	(88)	(86)	(87)	(85)
Intestine small, ileum	(86)	(83)	(84)	(83)
Hyperplasia, lymphocyte	1 (1%)	–	–	–
Intestine small, jejunum	(83)	(81)	(84)	(79)
Liver	(90)	(90)	(90)	(90)
Angiectasis	6 (7%)	3 (3%)	9 (10%)	3 (3%)
Basophilic focus	11 (12%)	11 (12%)	7 (8%)	15 (17%)
Clear cell focus	2 (2%)	4 (4%)	7 (8%)	3 (3%)
Congestion	–	–	–	1 (1%)
Eosinophilic focus	9 (10%)	17 (19%)	10 (11%)	9(10%)
Extramedullary hematopoiesis	15 (17%)	11 (12%)	13 (14%)	13 (14%)
Hepatodiaphragmatic nodule	1 (1%)	–	–	3 (3%)
Infiltration cellular, histiocyte	–	1 (1%)	–	–
Infiltration cellular, mixed cell	1 (1%)	2 (2%)	4 (4%)	2 (2%)
Inflammation, granulomatous	–	1 (1%)	–	–
Mitotic alteration	–	–	–	1 (1%)
Mixed cell focus	29 (32%)	17 (19%)	29 (32%)	35 (39%)
Pigment	–	1 (1%)	–	–
Artery, inflammation, chronic active	–	1 (1%)	–	–
Bile duct, cyst	11 (12%)	14 (16%)	6 (7%)	9 (10%)
Bile duct, fibrosis	1 (1%)	1 (1%)	4 (4%)	–
Bile duct, hyperplasia	9 (10%)	10 (11%)	12 (13%)	7 (8%)
Hepatocyte, hypertrophy	2 (2%)	2 (2%)	1 (1%)	1 (1%)
Hepatocyte, increased mitoses	2 (2%)	–	–	–
Hepatocyte, necrosis	4 (4%)	9 (10%)	7 (8%)	4 (4%)
Hepatocyte, vacuolation, cytoplasmic	1 (1%)	5 (6%)	5 (6%)	9 (10%)
Kupffer cell, hyperplasia	3 (3%)	–	–	1 (1%)
Kupffer cell, hypertrophy	2 (2%)	–	–	–
Kupffer cell, pigment	–	–	1 (1%)	1 (1%)
Periductal, cholangiofibrosis	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Serosa, inflammation, chronic active	1 (1%)	–	–	–
Mesentery	(4)	(3)	(11)	(4)
Hemorrhage	–	–	1 (9%)	–
Inflammation, chronic active	1 (25%)	–	–	1 (25%)
Necrosis	1 (25%)	1 (33%)	5 (45%)	2 (50%)
Artery, inflammation, chronic active	–	2 (67%)	2 (18%)	–
Vein, degeneration	–	–	1 (9%)	–
Vein, inflammation, chronic active	–	–	1 (9%)	1 (25%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Oral mucosa	(1)	(1)	(0)	(0)
Inflammation, chronic active	–	1 (100%)	–	–
Pancreas	(90)	(90)	(90)	(89)
Ectopic liver	1 (1%)	–	1 (1%)	–
Inflammation, chronic active	1 (1%)	–	–	3 (3%)
Necrosis	–	–	–	1 (1%)
Acinus, atrophy	5 (6%)	2 (2%)	6 (7%)	2 (2%)
Acinus, hyperplasia	1 (1%)	4 (4%)	2 (2%)	2 (2%)
Artery, inflammation, chronic active	–	5 (6%)	–	1 (1%)
Periductal, cholangiofibrosis	–	3 (3%)	2 (2%)	1 (1%)
Salivary glands	(90)	(90)	(90)	(90)
Degeneration	–	1 (1%)	–	–
Artery, inflammation, chronic active	–	3 (3%)	–	–
Duct, parotid gland, dilation	1 (1%)	1 (1%)	2 (2%)	–
Duct, parotid gland, fibrosis	–	–	1 (1%)	–
Parotid gland, atrophy	4 (4%)	7 (8%)	9 (10%)	1 (1%)
Parotid gland, fibrosis	–	–	2 (2%)	3 (3%)
Parotid gland, inflammation, suppurative	–	1 (1%)	–	–
Parotid gland, inflammation, acute	–	1 (1%)	1 (1%)	–
Parotid gland, mineral	–	–	–	1 (1%)
Parotid gland, vacuolation, cytoplasmic	–	–	1 (1%)	–
Sublingual gland, atrophy	–	2 (2%)	3 (3%)	–
Sublingual gland, fibrosis	–	–	1 (1%)	–
Sublingual gland, metaplasia	–	1 (1%)	–	1 (1%)
Submandibular gland, atrophy	–	–	1 (1%)	1 (1%)
Stomach, forestomach	(90)	(90)	(90)	(90)
Cyst, squamous	–	–	–	1 (1%)
Edema	2 (2%)	2 (2%)	2 (2%)	3 (3%)
Erosion	2 (2%)	1 (1%)	–	–
Fibrosis	1 (1%)	1 (1%)	1 (1%)	–
Inflammation, acute	–	–	–	1 (1%)
Inflammation, chronic active	4 (4%)	5 (6%)	2 (2%)	1 (1%)
Ulcer	1 (1%)	3 (3%)	3 (3%)	3 (3%)
Epithelium, hyperplasia	10 (11%)	11 (12%)	8 (9%)	8 (9%)
Epithelium, hyperplasia, basal cell	1 (1%)	1 (1%)	2 (2%)	–
Stomach, glandular	(90)	(90)	(89)	(88)
Cyst	–	1 (1%)	–	–
Erosion	1 (1%)	–	1 (1%)	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Artery, inflammation, chronic active	–	1 (1%)	–	–
Tongue	(1)	(0)	(0)	(0)
Cardiovascular System				
Aorta	(90)	(90)	(90)	(90)
Blood vessel	(0)	(0)	(0)	(1)
Pulmonary artery, degeneration	–	–	–	1 (100%)
Heart	(90)	(90)	(90)	(90)
Cardiomyopathy	40 (44%)	43 (48%)	33 (37%)	45 (50%)
Artery, inflammation, chronic	–	1 (1%)	–	–
Artery, mineral	–	1 (1%)	–	–
Artery, necrosis	–	1 (1%)	–	–
Atrium, endocardium, hyperplasia, Schwann cell	–	–	1 (1%)	–
Endocardium, hyperplasia, Schwann cell	–	1 (1%)	–	1 (1%)
Epicardium, inflammation, acute	–	–	1 (1%)	–
Ventricle right, cardiomyopathy	4 (4%)	7 (8%)	9 (10%)	9 (10%)
Endocrine System				
Adrenal cortex	(90)	(90)	(90)	(90)
Accessory adrenal cortical nodule	5 (6%)	7 (8%)	6 (7%)	12 (13%)
Atrophy	1 (1%)	–	2 (2%)	–
Degeneration, cystic	22 (24%)	19 (21%)	18 (20%)	19 (21%)
Extramedullary hematopoiesis	–	–	1 (1%)	–
Hyperplasia	14 (16%)	31 (34%)	26 (29%)	19 (21%)
Hypertrophy	52 (58%)	55 (61%)	56 (62%)	50 (56%)
Necrosis	2 (2%)	2 (2%)	2 (2%)	4 (4%)
Pigment	1 (1%)	–	1 (1%)	–
Vacuolation, cytoplasmic	18 (20%)	17 (19%)	11 (12%)	14 (16%)
Adrenal medulla	(86)	(89)	(87)	(88)
Hyperplasia	13 (15%)	20 (22%)	20 (23%)	18 (20%)
Hypertrophy	–	–	–	1 (1%)
Necrosis	1 (1%)	–	–	1 (1%)
Islets, pancreatic	(90)	(89)	(90)	(88)
Hyperplasia	15 (17%)	12 (13%)	14 (16%)	13 (15%)
Parathyroid gland	(87)	(80)	(85)	(85)
Cyst	–	–	2 (2%)	–
Fibrosis	13 (15%)	11 (14%)	6 (7%)	10 (12%)
Hyperplasia	–	2 (3%)	–	3 (4%)
Hyperplasia, focal	3 (3%)	–	2 (2%)	–
Hypertrophy, focal	–	–	–	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Pituitary gland	(90)	(89)	(89)	(90)
Angiectasis	–	1 (1%)	–	–
Atrophy	–	–	1 (1%)	–
Cyst	1 (1%)	–	1 (1%)	–
Fibrosis	–	–	1 (1%)	–
Pigment	–	–	2 (2%)	–
Pars distalis, angiectasis	2 (2%)	–	–	–
Pars distalis, cyst	7 (8%)	5 (6%)	3 (3%)	1 (1%)
Pars distalis, hyperplasia	20 (22%)	22 (25%)	26 (29%)	22 (24%)
Pars distalis, vacuolation, cytoplasmic	–	–	1 (1%)	–
Pars intermedia, cyst	3 (3%)	3 (3%)	1 (1%)	3 (3%)
Pars intermedia, hyperplasia	1 (1%)	–	1 (1%)	–
Pars nervosa, developmental malformation	–	–	–	1 (1%)
Thyroid gland	(90)	(90)	(90)	(89)
C-cell, hyperplasia	28 (31%)	30 (33%)	34 (38%)	38 (43%)
C-cell, hypoplasia	–	–	–	1 (1%)
Follicle, cyst	1 (1%)	–	1 (1%)	–
Follicular cell, hyperplasia	–	1 (1%)	–	–
General Body System				
Tissue NOS	(8)	(11)	(8)	(6)
Cyst	–	1 (9%)	–	–
Inflammation, chronic active	1 (13%)	1 (9%)	–	–
Abdominal, fat, necrosis	–	5 (45%)	3 (38%)	2 (33%)
Fat, necrosis	6 (75%)	4 (36%)	4 (50%)	3 (50%)
Mediastinum, cyst	–	–	–	1 (17%)
Mediastinum, hemorrhage	–	1 (9%)	–	–
Mediastinum, inflammation, chronic	–	1 (9%)	–	–
Genital System				
Clitoral gland	(87)	(88)	(89)	(86)
Hyperplasia	–	1 (1%)	–	–
Inflammation, suppurative	1 (1%)	1 (1%)	–	–
Inflammation, granulomatous	–	–	–	1 (1%)
Inflammation, acute	–	1 (1%)	–	–
Inflammation, chronic	–	–	1 (1%)	1 (1%)
Inflammation, chronic active	28 (32%)	43 (49%)	35 (39%)	42 (49%)
Duct, dilation	47 (54%)	64 (73%)	65 (73%)	60 (70%)
Duct, hyperplasia	–	3 (3%)	4 (4%)	1 (1%)
Ovary	(90)	(90)	(89)	(90)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Atrophy	72 (80%)	69 (77%)	56 (63%)	77 (86%)
Congestion	1 (1%)	–	–	–
Cyst	22 (24%)	27 (30%)	23 (26%)	34 (38%)
Fibrosis	–	1 (1%)	1 (1%)	1 (1%)
Hemorrhage	–	–	–	1 (1%)
Inflammation, chronic	–	1 (1%)	1 (1%)	–
Inflammation, chronic active	–	–	1 (1%)	–
Pigment	–	–	–	1 (1%)
Bursa, dilation	4 (4%)	4 (4%)	2 (2%)	1 (1%)
Follicle, cyst	–	–	–	1 (1%)
Periovarian tissue, cyst	–	–	1 (1%)	–
Rete ovarii, cyst	–	–	1 (1%)	–
Rete ovarii, hyperplasia	15 (17%)	17 (19%)	14 (16%)	11 (12%)
Oviduct	(1)	(0)	(0)	(0)
Cyst	1 (100%)	–	–	–
Uterus	(90)	(90)	(90)	(90)
Adenomyosis	–	2 (2%)	2 (2%)	–
Angiectasis	1 (1%)	–	–	–
Cyst	5 (6%)	6 (7%)	7 (8%)	11 (12%)
Dilation	8 (9%)	10 (11%)	11 (12%)	8 (9%)
Fibrosis	1 (1%)	–	1 (1%)	
Hemorrhage	–	–	1 (1%)	4 (4%)
Infiltration cellular, plasma cell	–	1 (1%)	–	–
Inflammation, suppurative	4 (4%)	11 (12%)	8 (9%)	12 (13%)
Inflammation, acute	1 (1%)	1 (1%)	1 (1%)	1 (1%)
Inflammation, chronic active	–	–	4 (4%)	1 (1%)
Thrombus	1 (1%)	1 (1%)	–	–
Cervix, hyperplasia, stromal	2 (2%)	1 (1%)	1 (1%)	1 (1%)
Cervix, thrombus	–	1 (1%)	–	–
Cervix, epithelium, hyperplasia	–	1 (1%)	–	–
Cervix, serosa, fibrosis	1 (1%)	–	–	–
Endometrium, hyperplasia, cystic	37 (41%)	43 (48%)	35 (39%)	46 (51%)
Epithelium, metaplasia, squamous	48 (53%)	39 (43%)	28 (31%)	46 (51%)
Glands, dilation	–	1 (1%)	–	–
Vagina	(2)	(1)	(0)	(1)
Cyst	–	–	–	1 (100%)
Hematopoietic System				
Bone marrow	(90)	(90)	(90)	(90)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Fibrosis	–	2 (2%)	–	–
Hypercellularity	56 (62%)	52 (58%)	43 (48%)	43 (48%)
Lymph node	(13)	(8)	(11)	(20)
Erythrophagocytosis	–	1 (13%)	–	–
Axillary, erythrophagocytosis	–	–	–	1 (5%)
Axillary, proliferation, plasma cell	1 (8%)	–	–	–
Bronchial, erythrophagocytosis	–	–	1 (9%)	–
Bronchial, proliferation, plasma cell	–	–	1 (9%)	–
Deep cervical, erythrophagocytosis	–	–	–	1 (5%)
Iliac, erythrophagocytosis	3 (23%)	3 (38%)	1 (9%)	3 (15%)
Iliac, hyperplasia, lymphocyte	1 (8%)	–	1 (9%)	6 (30%)
Iliac, infiltration cellular, histiocyte	–	–	–	1 (5%)
Iliac, inflammation, acute	1 (8%)	–	–	–
Iliac, pigment	1 (8%)	–	–	3 (15%)
Iliac, proliferation, plasma cell	6 (46%)	1 (13%)	2 (18%)	5 (25%)
Iliac, lymphatic sinus, ectasia	–	1 (13%)	1 (9%)	5 (25%)
Inguinal, erythrophagocytosis	1 (8%)	–	–	–
Inguinal, hyperplasia, lymphocyte	–	–	1 (9%)	–
Inguinal, pigment	–	–	1 (9%)	–
Inguinal, proliferation, plasma cell	1 (8%)	–	–	–
Inguinal, lymphatic sinus, ectasia	1 (8%)	1 (13%)	1 (9%)	–
Lumbar, erythrophagocytosis	1 (8%)	2 (25%)	–	–
Lumbar, hyperplasia, lymphocyte	–	–	–	1 (5%)
Lumbar, lymphatic sinus, ectasia	–	–	–	1 (5%)
Lymphatic sinus, renal, ectasia	–	1 (13%)	–	–
Mediastinal, congestion	1 (8%)	–	–	–
Mediastinal, erythrophagocytosis	–	2 (25%)	4 (36%)	4 (20%)
Mediastinal, proliferation, plasma cell	1 (8%)	–	–	–
Pancreatic, erythrophagocytosis	1 (8%)	–	1 (9%)	–
Pancreatic, infiltration cellular, histiocyte	–	–	–	1 (5%)
Renal, erythrophagocytosis	–	2 (25%)	–	–
Lymph node, mandibular	(90)	(90)	(89)	(90)
Congestion	–	2 (2%)	–	1 (1%)
Erythrophagocytosis	–	1 (1%)	2 (2%)	3 (3%)
Hemorrhage	1 (1%)	–	–	–
Hyperplasia, lymphocyte	46 (51%)	49 (54%)	45 (51%)	43 (48%)
Infiltration cellular, histiocyte	–	–	–	1 (1%)
Pigment	–	–	–	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Proliferation, plasma cell	68 (76%)	68 (76%)	58 (65%)	56 (62%)
Lymphatic sinus, ectasia	1 (1%)	2 (2%)	2 (2%)	3 (3%)
Lymph node, mesenteric	(90)	(90)	(90)	(89)
Atrophy	1 (1%)	–	–	–
Erythrophagocytosis	1 (1%)	3 (3%)	2 (2%)	–
Hyperplasia, lymphocyte	–	–	1 (1%)	–
Infiltration cellular, histiocyte	2 (2%)	–	–	1 (1%)
Lymphatic sinus, ectasia	–	–	1 (1%)	1 (1%)
Spleen	(90)	(90)	(90)	(90)
Accessory spleen	–	1 (1%)	–	–
Extramedullary hematopoiesis	80 (89%)	74 (82%)	79 (88%)	82 (91%)
Fibrosis	–	1 (1%)	–	–
Hemorrhage	–	1 (1%)	–	1 (1%)
Hyperplasia, lymphocyte	–	1 (1%)	–	–
Hyperplasia, stromal	1 (1%)	–	1 (1%)	–
Pigment	74 (82%)	79 (88%)	77 (86%)	79 (88%)
Red pulp, atrophy	7 (8%)	11 (12%)	13 (14%)	6 (7%)
White pulp, atrophy	3 (3%)	3 (3%)	4 (4%)	1 (1%)
Thymus	(87)	(83)	(87)	(87)
Atrophy	75 (86%)	67 (81%)	74 (85%)	63 (72%)
Cyst	39 (45%)	34 (41%)	34 (39%)	45 (52%)
Ectopic parathyroid gland	1 (1%)	2 (2%)	2 (2%)	2 (2%)
Hemorrhage	2 (2%)	5 (6%)	5 (6%)	3 (3%)
Hyperplasia, epithelial	55 (63%)	59 (71%)	54 (62%)	38 (44%)
Hyperplasia, lymphocyte	–	–	1 (1%)	–
Artery, inflammation, chronic active	–	2 (2%)	1 (1%)	–
Integumentary System				
Mammary gland	(90)	(90)	(90)	(90)
Galactocele	24 (27%)	17 (19%)	17 (19%)	10 (11%)
Hyperplasia	49 (54%)	50 (56%)	46 (51%)	34 (38%)
Inflammation, granulomatous	–	–	2 (2%)	–
Inflammation, acute	–	–	–	1 (1%)
Inflammation, chronic active	–	–	2 (2%)	1 (1%)
Duct, dilation	56 (62%)	61 (68%)	51 (57%)	70 (78%)
Skin	(90)	(90)	(90)	(90)
Cyst epithelial inclusion	1 (1%)	1 (1%)	3 (3%)	1 (1%)
Hyperkeratosis	–	1 (1%)	–	–
Inflammation, chronic active	1 (1%)	–	–	1 (1%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Ulcer	–	–	1 (1%)	–
Epidermis, hyperplasia	2 (2%)	–	–	–
Lymphatic, subcutaneous tissue, angiectasis	–	–	–	1 (1%)
Subcutaneous tissue, inflammation, chronic active	–	1 (1%)	–	–
Musculoskeletal System				
Bone	(90)	(90)	(90)	(90)
Fibrous osteodystrophy	–	1 (1%)	–	–
Cranium, fracture	1 (1%)	–	–	–
Mandible, fracture	1 (1%)	–	–	–
Maxilla, fracture	1 (1%)	–	–	–
Skeletal muscle	(90)	(90)	(90)	(90)
Degeneration	3 (3%)	7 (8%)	10 (11%)	2 (2%)
Diaphragm, hernia	–	–	–	1 (1%)
Nervous System				
Brain	(90)	(90)	(90)	(90)
Compression	26 (29%)	31 (34%)	16 (18%)	20 (22%)
Congestion	1 (1%)	–	–	–
Cyst	–	1 (1%)	–	–
Edema	2 (2%)	1 (1%)	–	–
Hemorrhage	–	1 (1%)	–	–
Mineral	–	1 (1%)	1 (1%)	–
Pigment	–	–	1 (1%)	–
Cerebrum, degeneration	–	–	1 (1%)	–
Choroid plexus, mineral	–	1 (1%)	–	–
Glial cell, hyperplasia	–	–	1 (1%)	1 (1%)
Meninges, hyperplasia	1 (1%)	–	1 (1%)	–
Meninges, hyperplasia, granular cell	1 (1%)	–	–	1 (1%)
Meninges, mineral	–	–	1 (1%)	–
Neuron, necrosis	–	1 (1%)	–	–
Pineal gland, infiltration cellular, mononuclear cell	–	1 (1%)	–	–
Pineal gland, mineral	1 (1%)	–	–	–
Pineal gland, vacuolation, cytoplasmic	1 (1%)	–	2 (2%)	–
Nerve trigeminal	(84)	(84)	(85)	(84)
Degeneration	64 (76%)	70 (83%)	64 (75%)	72 (86%)
Gliosis	–	–	1 (1%)	–
Peripheral nerve, sciatic	(90)	(90)	(90)	(90)
Degeneration	80 (89%)	83 (92%)	83 (92%)	89 (99%)
Infiltration cellular, mixed cell	1 (1%)	–	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Peripheral nerve, tibial	(90)	(90)	(89)	(89)
Degeneration	77 (86%)	77 (86%)	83 (93%)	86 (97%)
Spinal cord, cervical	(90)	(90)	(90)	(90)
Degeneration	24 (27%)	29 (32%)	22 (24%)	35 (39%)
Spinal cord, lumbar	(90)	(90)	(89)	(90)
Degeneration	10 (11%)	11 (12%)	15 (17%)	12 (13%)
Nerve, degeneration	74 (82%)	77 (86%)	77 (87%)	80 (89%)
Spinal cord, thoracic	(90)	(90)	(90)	(90)
Degeneration	59 (66%)	64 (71%)	59 (66%)	70 (78%)
Trigeminal ganglion	(81)	(77)	(81)	(75)
Degeneration	33 (41%)	21 (27%)	22 (27%)	28 (37%)
Respiratory System				
Lung	(90)	(90)	(90)	(90)
Congestion	3 (3%)	12 (13%)	9 (10%)	5 (6%)
Foreign body	–	1 (1%)	–	1 (1%)
Hemorrhage	1 (1%)	6 (7%)	–	1 (1%)
Inflammation, suppurative	2 (2%)	–	–	1 (1%)
Inflammation, granulomatous	1 (1%)	5 (6%)	1 (1%)	2 (2%)
Inflammation, chronic active	6 (7%)	6 (7%)	6 (7%)	11 (12%)
Alveolar epithelium, hyperplasia	–	1 (1%)	–	–
Alveolar epithelium, metaplasia, squamous	–	1 (1%)	–	2 (2%)
Alveolus, infiltration cellular, histiocyte	71 (79%)	77 (86%)	84 (93%)	81 (90%)
Artery, inflammation, chronic active	1 (1%)	–	–	–
Artery, muscularis, hyperplasia	–	–	–	1 (1%)
Bronchus, hyperplasia	–	1 (1%)	–	–
Epithelium alveolus, hyperplasia	2 (2%)	2 (2%)	3 (3%)	1 (1%)
Pleura, inflammation, acute	–	–	1 (1%)	–
Nose	(90)	(89)	(90)	(89)
Foreign body	–	1 (1%)	1 (1%)	2 (2%)
Inflammation, suppurative	1 (1%)	3 (3%)	1 (1%)	3 (3%)
Inflammation, acute	–	1 (1%)	–	–
Inflammation, chronic active	–	1 (1%)	–	–
Nerve, degeneration	–	1 (1%)	–	–
Olfactory epithelium, accumulation, hyaline droplet	89 (99%)	89 (100%)	86 (96%)	86 (97%)
Olfactory epithelium, hyperplasia	–	–	1 (1%)	–
Olfactory epithelium, metaplasia, respiratory	1 (1%)	–	–	2 (2%)
Olfactory epithelium, metaplasia, squamous	–	1 (1%)	–	–

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Respiratory epithelium, accumulation, hyaline droplet	12 (13%)	19 (21%)	22 (24%)	11 (12%)
Respiratory epithelium, hyperplasia	–	1 (1%)	–	3 (3%)
Respiratory epithelium, metaplasia, squamous	–	–	–	1 (1%)
Trachea	(89)	(88)	(89)	(89)
Inflammation, chronic active	1 (1%)	1 (1%)	–	–
Epithelium, hyperplasia	–	1 (1%)	–	–
Epithelium, metaplasia, squamous	–	1 (1%)	–	–
Glands, cyst	1 (1%)	1 (1%)	–	2 (2%)
Special Senses System				
Ear	(0)	(0)	(1)	(1)
Eye	(88)	(86)	(88)	(86)
Anterior chamber, exudate	–	1 (1%)	–	–
Anterior chamber, inflammation, acute	–	–	1 (1%)	2 (2%)
Anterior chamber, iris, synechia	–	1 (1%)	–	–
Choroid, inflammation, chronic active	–	–	1 (1%)	–
Cornea, fibrosis	–	1 (1%)	–	–
Cornea, inflammation, acute	1 (1%)	2 (2%)	1 (1%)	2 (2%)
Cornea, inflammation, chronic active	–	1 (1%)	–	–
Cornea, neovascularization	–	1 (1%)	–	–
Cornea, ulcer	–	–	–	1 (1%)
Cornea, epithelium, hyperplasia	1 (1%)	2 (2%)	–	1 (1%)
Lens, cataract	1 (1%)	3 (3%)	–	–
Retina, atrophy	18 (20%)	17 (20%)	18 (20%)	18 (21%)
Retina, dysplasia	1 (1%)	1 (1%)	1 (1%)	3 (3%)
Harderian gland	(90)	(90)	(90)	(90)
Atrophy	13 (14%)	15 (17%)	16 (18%)	17 (19%)
Cyst	–	–	–	1 (1%)
Hyperplasia	–	–	–	1 (1%)
Hypertrophy	–	–	–	1 (1%)
Infiltration cellular, lymphocyte	2 (2%)	–	–	1 (1%)
Inflammation, granulomatous	7 (8%)	6 (7%)	4 (4%)	9 (10%)
Inflammation, chronic	7 (8%)	1 (1%)	1 (1%)	2 (2%)
Inflammation, chronic active	1 (1%)	4 (4%)	2 (2%)	–
Urinary System				
Kidney	(90)	(90)	(90)	(89)
Inflammation, acute	1 (1%)	–	–	–
Nephropathy, chronic progressive	74 (82%)	76 (84%)	76 (84%)	65 (73%)

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Artery, inflammation, chronic active	1 (1%)	–	–	–
Pelvis, dilation	3 (3%)	–	2 (2%)	–
Pelvis, inflammation, suppurative	–	2 (2%)	–	–
Pelvis, mineral	–	–	–	1 (1%)
Pelvis, urothelium, hyperplasia	–	1 (1%)	–	–
Renal tubule, cyst	3 (3%)	2 (2%)	–	–
Renal tubule, hyperplasia	–	–	1 (1%)	–
Renal tubule, necrosis	–	1 (1%)	–	–
Urinary bladder	(88)	(88)	(90)	(90)
Dilation	1 (1%)	–	–	–
Edema	–	3 (3%)	–	–
Fibrosis	–	–	1 (1%)	–
Hemorrhage	–	1 (1%)	1 (1%)	–
Infiltration cellular, histiocyte	–	1 (1%)	1 (1%)	–
Inflammation, acute	3 (3%)	2 (2%)	–	–
Inflammation, chronic active	–	1 (1%)	–	–
Necrosis	1 (1%)	–	–	–
Artery, inflammation, chronic active	–	1 (1%)	–	–
Urothelium, hyperplasia	1 (1%)	–	–	–

[a]Number of animals examined microscopically at the site and the number of animals with lesion.

Appendix E. Genetic Toxicology

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E.1. Collection of Tissue Samples for Genotoxicity Testing

Exposures ceased at 7 a.m. on the day of necropsy at 14 weeks after weaning. Thirty-five male rats (five sham controls, 15 that were exposed to CDMA, and 15 that were exposed to GSM) were necropsied approximately 2 to 4 hours after cessation of exposure and 35 female rats (five sham controls, 15 that were exposed to CDMA, and 15 that were exposed to GSM) were necropsied approximately 5 to 7 hours after cessation of exposure. Animals were necropsied in the following order: one animal from each exposure group starting with the sham control group, moving through each of the exposed groups for each of the radiofrequency modulations in turn, then rotating back to the sham control group; animals were necropsied in numerical order within each exposure group. Five different tissues (cerebrum, frontal cortex, hippocampus, liver, and blood leukocytes) were collected from each animal for the comet assay. Because blood was examined in both the micronucleus and the comet assays, a single tube of blood was collected per animal by retroorbital bleeding, and the sample was divided into two aliquots, one that was processed for the comet assay and the other for the micronucleus assay.

E.2. Comet Assay

For preparation of samples for the comet assay, a 50 µL sample of blood was transferred to a tube containing 1 mL of freshly prepared cold mincing buffer [Mg+2, Ca+2, and phenol free Hank's Balanced Salt Solution (Life Technologies, Carlsbad, CA) with 20 mM ethylenediaminetetraacetic acid (EDTA) pH 7.3 to 7.5 and 10% v/v fresh dimethyl sulfoxide (DMSO)]. The liver and the hippocampus, cerebellum, and frontal cortex sections of the brain were rinsed with cold mincing buffer to remove residual blood and held on ice briefly (≤5 minutes) until processed. Small portions (3 to 4 mm) of the left lobe of the liver and each brain section were placed in tubes containing cold mincing solution and rapidly minced until finely dispersed. All samples prepared for the comet assay were immediately flash frozen in liquid nitrogen[211] and subsequently transferred to a –80°C freezer for storage until shipment by overnight courier on dry ice to the analytical laboratory. Upon receipt, all samples were immediately placed in a –80°C freezer for storage until further processing.

Blood and tissue samples were thawed on ice and maintained on ice during slide preparation. Just prior to use, each cell suspension was shaken gently to mix the cells and placed back on ice for 15 to 30 seconds to allow clumps to settle. A portion of the supernatant was empirically diluted with 0.5% low melting point agarose (Lonza, Walkersville, MD) dissolved in Dulbecco's phosphate buffer (Ca+2, Mg+2, and phenol free) at 37°C and layered onto each well of a 2-well CometSlide™ (Trevigen, Gaithersburg, MD). Slides were immersed in cold lysing solution [2.5 M NaCl, 100 mM Na2EDTA, 10 mM tris(hydroxymethyl)aminomethane (Tris), pH 10, containing freshly added 10% DMSO (Fisher Scientific, Pittsburgh, PA) and 1% Triton X-100] overnight in a refrigerator, protected from light. The following day, the slides were rinsed in 0.4 M Trizma base (pH 7.5), randomly placed onto the platform of a horizontal electrophoresis unit and treated with cold alkali solution (300 mM NaOH, 1 mM Na2EDTA, pH > 13) for 20 minutes to allow DNA unwinding, then electrophoresed at 4º to 9ºC for 20 minutes at 25 V (0.7 V/cm), with a current of approximately 300 mA. Following electrophoresis, slides were neutralized with 0.4 M Trizma base (pH 7.5) for 5 minutes and then dehydrated by immersion in absolute ethanol (Pharmco-AAPER, Shelbyville, KY) for at least 5 minutes and allowed to air dry. Slides were prepared in a laboratory with a relative humidity of no more than 60% and

stored at room temperature in a desiccator with a relative humidity of no more than 60% until stained and scored; stained slides were stored in a desiccator. NaCl, Na2EDTA, Triton X-100, and Trizma base were purchased from Sigma-Aldrich (St. Louis, MO); NaOH was purchased from Fisher Scientific (Pittsburgh, PA).

After staining with SYBR® Gold (Molecular Probes, Life Technologies, Grand Island, NY), slides, independently coded to mask treatment, were scored using Comet Assay IV Imaging Software, Version 4.3.1 (Perceptive Instruments, Ltd., Suffolk, UK) validated for GLP Part 11 compliance. In the alkaline (pH > 13) comet assay, when damaged nuclear DNA fragments, it undergoes unidirectional migration through the agarose gel within an electrical field, forming an image that resembles a comet, and the greater the amount of fragmentation, the greater the amount of DNA migration that will occur. The image analysis software partitions the intensity of the fluorescent signal of the DNA in the entire comet image into the percent that is attributable to the comet head and the percent attributable to the tail. Manual adjustment of the automated detection of head and tail features is sometimes required. To evaluate DNA damage levels, the extent of DNA migration was characterized for 100 scorable comet figures per animal/tissue as percent tail DNA (intensity of all tail pixels divided by the total intensity of all pixels in the comet, expressed as a percentage).

Comet figures are classified during the scoring process as scorable (evaluated for percent tail DNA), non-scorable (due to inability to evaluate percent tail DNA, e.g., if comets overlapped), and "hedgehog." Hedgehogs either have no defined head, i.e., all DNA appears to be in the tail, or the head and tail appear to be separated. Hedgehogs may represent cells that have sustained high levels of DNA damage and are apoptotic, although certain data suggest they may represent cells with high levels of repairable DNA damage[212; 213]. The frequency of hedgehogs (%HH) was determined by tabulating the number observed in a separate group of 100 cells per animal/tissue.

When rat samples were scored, a marked interanimal variation in percent tail DNA and high %HH values were observed in some tissues, yet the range of percent tail DNA values appeared to be truncated at approximately 65%. To better understand these observations, rat slides were reanalyzed by scoring 150 cells/tissue per animal, as recommended by the OECD guideline[184]. In this rescoring of the rat samples, all scorable cells were included in the sample of 150 analyzed cells, regardless of the apparent level of DNA damage estimated by the scorer prior to software analysis of the images; highly damaged cells that were unscorable using the software (true HH) were not included. For the 150-cell scoring method, the %HH was not independently determined due to limitations at the time in the comet assay software arising from the added number of cells scored. Therefore, %HH was estimated by dividing the number of comets having greater than or equal to 90% tail DNA by 150.

Although there was no concurrent positive control group in these cell phone RFR studies, slides were made with human TK6 cells treated with ethyl methanesulfonate (standard positive control compound for the comet assay) and were included in each electrophoresis run with each slide set as an internal technical positive control.

E.3. Micronucleus Assay

For the micronucleus assay, sampling schedules were as described for the comet assay. At 14 weeks after weaning, blood samples (approximately 200 μL) obtained by retroorbital bleeding

(one sample per rat) were placed into EDTA tubes and immediately refrigerated. The samples were sent on the day of collection to the analytical laboratory well insulated on cold packs via overnight delivery. Upon arrival, blood samples were diluted in anticoagulant (heparin) and fixed in ice cold methanol (Sigma-Aldrich, St. Louis, MO) according to instructions provided with the MicroFlowPLUS Kit (Litron Laboratories, Rochester, NY). Fixed blood samples were stored in a –80°C freezer for at least 3 days prior to analysis by flow cytometry.

Flow cytometric analysis of red blood cell samples was performed using MicroFlowPLUS Kit reagents and a FACSCalibur™ dual-laser bench top system (Becton Dickinson Biosciences, San Jose, CA) as described by MacGregor et al.[214] and Witt et al.[215]. Both mature [normochromatic erythrocytes (NCEs)] and immature [reticulocytes; polychromatic erythrocytes (PCEs)] erythrocytes were analyzed for the presence of micronuclei. Immature erythrocytes are distinguished by the presence of an active transferrin receptor (CD-71) on the cell surface. For each sample, 20,000 (± 2,000) immature CD71-positive erythrocytes were analyzed by flow cytometry to determine the frequency of micronucleated PCEs. Aggregates were excluded on the basis of forward and side scatter, platelets were excluded based on staining with an anti-CD61 antibody, and nucleated leukocytes were excluded on the basis of intense propidium iodide staining. Typically, more than one million NCEs (CD-71 negative) were enumerated concurrently during PCE analysis, allowing for calculation of the percentage of PCEs (% PCEs) among total erythrocytes as a measure of bone marrow toxicity.

E.4. Data Analysis for the Comet and Micronucleus Assays

Data from both the comet and the micronucleus assays were analyzed using the same statistical methods[216]. Mean percent tail DNA was calculated for each cell type for each animal; likewise, mean micronucleated PCEs/1,000 PCEs and micronucleated NCEs/1,000 NCEs, as well as % PCEs, were calculated for each animal. These data are summarized in the tables as mean ± standard error of the mean. Levene's test was used to determine if variances among exposed groups were equal at $P = 0.05$. When variances were equal, linear regression analysis was used to test for linear trend and Williams' test was used to evaluate pairwise differences of each exposed group with the sham control group. When variances were unequal, nonparametric methods were used to analyze the data; Jonckheere's test was used to evaluate linear trend and Dunn's test was used to assess the significance of pairwise differences of each exposed group with the sham control group. To maintain the overall significance level at 0.05, the trend as well as the pairwise differences from the sham control group were declared statistically significant if $P < 0.025$. A result was considered positive if the trend test was significant and if at least one exposed group was significantly elevated over the sham control group, or if two or more exposed groups were significantly increased over the corresponding sham control group. A response was considered equivocal if only the trend test was significant or if only a single exposed group was significantly increased over the sham control.

E.5. Results

Twenty tissue samples obtained from animals in the 14-week interim evaluation study were evaluated for DNA damage using the comet assay (two sexes, two cell phone RFR modulations, five tissues). Results are reported based on the standard 100-cell scoring approach in use at the time these data were collected; data obtained using a 150-cell scoring approach, recommended in

a recently adopted international guideline for the in vivo comet assay, are noted for comparison. The only clear positive result was observed in hippocampus cells of male rats exposed to the CDMA modulation (Table E-1). Data obtained using the 150-cell scoring approach did not meet the statistical criteria for a positive result, although the mean % tail DNA values were elevated over the sham controls in all exposure groups, and the values increased with increasing dose level (Table E-3). An exposure-related increase in DNA damage was seen in the cells of the frontal cortex of male rats exposed to the CDMA modulation (Table E-1). However, although the trend test was significant (P = 0.004), no individual exposure groups were significantly elevated over the sham control group and the result was therefore judged to be equivocal. Data obtained using the 150-cell scoring approach showed a similar pattern of response in the male frontal cortex (CDMA) and were also considered to be equivocal based on a significant trend test (P = 0.005) (Table E-3). For male rat blood leukocytes exposed to either the CDMA or GSM modulation (Table E-1 and Table E-2), results from scoring 100 cells were negative; however, these leukocyte samples showed equivocal responses with the 150-cell method due to a significant trend test (P = 0.012) or pairwise test (P = 0.021) for CDMA- and GSM-exposed rats, respectively (Table E-3 and Table E-4). No statistically significant increases in the percent tail DNA were observed in any of the female rat samples scored with the 100-cell approach (Table E-5 and Table E-6). The 150-cell scoring approach yielded a significant trend test (P = 0.013) in peripheral blood leukocytes of female rats exposed to the CDMA modulation, but these results were driven by data from a single animal (Table E-7).

In contrast to what was seen in the mice, a high degree of interanimal variability was observed in the % tail DNA values in rats within a treatment group, and this level of variability reduced the statistical power to detect increases in DNA migration, although the magnitudes of the increases observed in some rats suggested these were treatment-related effects. To rule out any influence from technical artifacts or protocol features, % tail DNA values and %HH were correlated to the position of slides in the electrophoresis chambers, the interval from exposure cessation to tissue collection, and the date of slide preparation; no patterns emerged for any of these variables and the level of DNA damage observed.

The possibility that the longer interval from exposure cessation to tissue collection for the female rats may have been a factor in the absence of any detectable exposure-related increases in DNA damage cannot be ruled out due to the increased opportunity for DNA repair during this interval.

Similar to what was seen in the mice, no significant increases in micronucleated red blood cells or in the % PCEs were observed in rats of either sex exposed to either modulation of cell phone RFR (Table E-9).

Table E-1. DNA Damage in Male Rats Exposed to CDMA-modulated Cell Phone RFR for 19 Weeks (100-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b]
Frontal Cortex				
Sham Control[d]	0	6.18 ± 0.72		2.00 ± 0.71
CDMA	1.5	6.00 ± 0.48	1.000	1.00 ± 0.77
	3	9.51 ± 1.17	0.081	10.60 ± 3.89
	6	12.78 ± 3.96	0.049	12.20 ± 6.84
		P = 0.004[e]		
Hippocampus				
Sham Control	0	5.88 ± 0.39		3.40 ± 1.21
CDMA	1.5	8.06 ± 1.20	0.135	3.80 ± 2.33
	3	8.16 ± 0.98	0.151	6.20 ± 2.56
	6	10.42 ± 2.18	0.019	4.40 ± 2.98
		P = 0.014		
Cerebellum				
Sham Control	0	5.57 ± 0.92		0.40 ± 0.24
CDMA	1.5	5.60 ± 0.71	1.000	1.80 ± 0.80
	3	10.70 ± 3.66	0.504	9.40 ± 6.81
	6	10.58 ± 3.52	0.731	8.00 ± 3.91
		P = 0.156		
Liver				
Sham Control	0	13.81 ± 2.88		33.60 ± 17.89
CDMA	1.5	22.99 ± 2.77	0.081	68.60 ± 15.70
	3	16.04 ± 2.14	0.098	7.80 ± 0.86
	6	20.79 ± 3.10	0.057	41.10 ± 14.80
		P = 0.154		
Peripheral Blood				
Sham Control	0	1.48 ± 0.29		0.20 ± 0.20
CDMA	1.5	1.22 ± 0.45	0.596	0.80 ± 0.80
	3	2.13 ± 0.34	0.156	0.40 ± 0.40
	6	2.08 ± 0.43	0.166	1.40 ± 1.17
		P = 0.071		

[a]Study was performed at ILS, Inc. The detailed protocol is presented by Recio et al.[211]. Groups of five rats per exposure group; exposure began in utero on gestation day 6. Statistical analysis runs Levene's test to determine if variances of exposed groups are equal (P = 0.05). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.
[b]Mean ± standard error.
[c]Pairwise comparison with the sham control group; exposed group values are significant at P ≤ 0.025 by Williams' or Dunn's test.
[d]No exposure to CDMA-modulated cell phone RFR.
[e]Dose-related trend derived from one-tailed linear regression or Jonckheere's test; the trend is significant when P ≤ 0.025.

Table E-2. DNA Damage in Male Rats Exposed to GSM-modulated Cell Phone RFR for 19 Weeks (100-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b]
Frontal Cortex				
Sham Control[d]	0	6.18 ± 0.72		2.00 ± 0.71
GSM	1.5	6.98 ± 0.42	0.465	1.40 ± 0.51
	3	8.66 ± 1.96	0.247	8.20 ± 2.69
	6	6.30 ± 0.32	1.000	3.00 ± 1.55
		P = 0.343[e]		
Hippocampus				
Sham Control	0	5.88 ± 0.39		3.40 ± 1.21
GSM	1.5	11.82 ± 2.68	0.092	4.80 ± 2.84
	3	9.64 ± 1.27	0.111	4.80 ± 1.53
	6	11.69 ± 3.92	0.072	10.20 ± 7.98
		P = 0.103		
Cerebellum				
Sham Control	0	5.57 ± 0.92		0.40 ± 0.24
GSM	1.5	7.36 ± 2.48	0.295	2.40 ± 1.91
	3	6.37 ± 0.77	0.354	3.40 ± 1.17
	6	8.48 ± 1.85	0.149	5.00 ± 2.86
		P = 0.132		
Liver				
Sham Control	0	13.81 ± 2.88		33.60 ± 17.89
GSM	1.5	13.26 ± 2.38	0.547	21.00 ± 12.30
	3	13.09 ± 2.32	0.634	28.40 ± 15.07
	6	14.49 ± 2.71	0.536	24.80 ± 16.13
		P = 0.404		
Peripheral Blood				
Sham Control	0	1.48 ± 0.29		0.20 ± 0.20
GSM	1.5	1.83 ± 0.63	0.352	3.20 ± 2.71
	3	1.78 ± 0.33	0.419	1.20 ± 0.49
	6	1.50 ± 0.27	0.446	0.40 ± 0.24
		P = 0.550		

[a]Study was performed at ILS, Inc. The detailed protocol is presented by Recio et al.[211]. Groups of five rats per exposure group; exposure began in utero on gestation day 6. Statistical analysis runs Levene's test to determine if variances of exposed groups are equal (P = 0.05). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.
[b]Mean ± standard error.
[c]Pairwise comparison with the sham control group; exposed group values are significant at P ≤ 0.025 by Williams' or Dunn's test.
[d]No exposure to GSM-modulated cell phone RFR.
[e]Dose-related trend derived from one-tailed linear regression or Jonckheere's test; the trend is significant when P ≤ 0.025.

Table E-3. DNA Damage in Male Rats Exposed to CDMA-modulated Cell Phone RFR for 19 Weeks (150-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b,d]
Frontal Cortex				
Sham Control[e]	0	9.73 ± 0.81		0.27 ± 0.27
CDMA	1.5	8.24 ± 0.39	1.000	0.13 ± 0.13
	3	18.77 ± 3.27	0.043	2.53 ± 1.29
	6	23.62 ± 8.66	0.092	3.20 ± 1.72
		P = 0.005[f]		
Hippocampus				
Sham Control	0	8.99 ± 1.55		1.07 ± 0.45
CDMA	1.5	12.27 ± 2.21	0.244	0.40 ± 0.27
	3	15.46 ± 2.25	0.107	2.53 ± 0.90
	6	16.77 ± 5.44	0.069	2.40 ± 1.44
		P = 0.043		
Cerebellum				
Sham Control	0	4.90 ± 0.82		0.00 ± 0.00
CDMA	1.5	6.33 ± 1.00	0.681	0.27 ± 0.16
	3	13.75 ± 6.01	0.504	2.93 ± 2.20
	6	15.86 ± 5.91	0.163	2.40 ± 1.07
		P = 0.061		
Liver				
Sham Control	0	25.71 ± 8.71		1.73 ± 1.73
CDMA	1.5	55.41 ± 7.91	0.136	14.67 ± 5.57
	3	19.11 ± 2.28	0.164	0.80 ± 0.49
	6	40.01 ± 7.90	0.114	9.07 ± 7.10
		P = 0.385		
Peripheral Blood				
Sham Control	0	0.69 ± 0.20		0.00 ± 0.00
CDMA	1.5	1.16 ± 0.47	0.295	0.00 ± 0.00
	3	1.83 ± 0.74	0.121	0.13 ± 0.13
	6	2.57 ± 0.80	0.026	0.00 ± 0.00
		P = 0.012		

[a]Study was performed at ILS, Inc. The detailed protocol is presented by Recio et al.[211] and OECD[184]. Groups of five rats per exposure group; exposure began in utero on gestation day 6. Statistical analysis runs Levene's test to determine if variances of exposed groups are equal (P = 0.05). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.
[b]Mean ± standard error.
cPairwise comparison with the sham control group; exposed group values are significant at P ≤ 0.025 by Williams' or Dunn's test.
[d]Percent hedgehogs = estimated as the number of comets with ≥90% tail DNA/150.
[e]No exposure to CDMA-modulated cell phone RFR.
[f]Dose-related trend derived from one-tailed linear regression or Jonckheere's test; the trend is significant when P ≤ 0.025.

Table E-4. DNA Damage in Male Rats Exposed to GSM-modulated Cell Phone RFR for 19 Weeks (150-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b,d]
Frontal Cortex				
Sham Control[e]	0	9.73 ± 0.81		0.27 ± 0.27
GSM	1.5	11.96 ± 1.65	0.634	0.40 ± 0.27
	3	17.98 ± 5.12	0.545	1.20 ± 0.57
	6	9.57 ± 1.57	1.000	0.13 ± 0.13
		P = 0.500[f]		
Hippocampus				
Sham Control	0	8.99 ± 1.55		1.07 ± 0.45
GSM	1.5	17.24 ± 4.09	0.186	0.27 ± 0.16
	3	14.77 ± 2.54	0.227	1.47 ± 0.57
	6	21.32 ± 9.55	0.080	3.60 ± 2.03
		P = 0.076		
Cerebellum				
Sham Control	0	4.90 ± 0.82		0.00 ± 0.00
GSM	1.5	9.43 ± 4.69	0.190	1.33 ± 1.17
	3	8.66 ± 2.17	0.232	1.47 ± 0.68
	6	12.11 ± 3.89	0.088	1.07 ± 1.07
		P = 0.076		
Liver				
Sham Control	0	25.71 ± 8.71		1.73 ± 1.73
GSM	1.5	23.27 ± 9.43	0.539	4.13 ± 3.64
	3	25.15 ± 8.43	0.604	0.40 ± 0.40
	6	28.25 ± 10.55	0.534	4.93 ± 3.94
		P = 0.390		
Peripheral Blood				
Sham Control	0	0.69 ± 0.20		0.00 ± 0.00
GSM	1.5	3.97 ± 2.75	0.146	0.27 ± 0.27
	3	1.97 ± 0.35	0.021	0.00 ± 0.00
	6	1.28 ± 0.23	0.272	0.00 ± 0.00
		P = 0.089		

[a]Study was performed at ILS, Inc. The detailed protocol is presented by Recio et al.[211] and OECD[184]. Groups of five rats per exposure group; exposure began in utero on gestation day 6. Statistical analysis runs Levene's test to determine if variances of exposed groups are equal (P = 0.05). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.
[b]Mean ± standard error.
[c]Pairwise comparison with the sham control group; exposed group values are significant at P ≤ 0.025 by Williams' or Dunn's test.
[d]Percent hedgehogs = estimated as the number of comets with ≥90% tail DNA/150.
[e]No exposure to GSM-modulated cell phone RFR.
[f]Dose-related trend derived from one-tailed linear regression or Jonckheere's test; the trend is significant when P ≤ 0.025.

Table E-5. DNA Damage in Female Rats Exposed to CDMA-modulated Cell Phone RFR for 19 Weeks (100-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b]
Frontal Cortex				
Sham Control[d]	0	7.03 ± 1.21		3.80 ± 1.46
CDMA	1.5	12.70 ± 5.15	0.205	19.00 ± 15.04
	3	9.50 ± 2.27	0.249	9.80 ± 5.12
	6	13.00 ± 3.63	0.150	25.40 ± 11.44
		P = 0.166[e]		
Hippocampus				
Sham Control	0[f]	13.14 ± 1.20		9.00 ± 2.58
CDMA	1.5	14.94 ± 0.70	0.346	8.40 ± 1.96
	3	15.24 ± 1.97	0.379	9.40 ± 2.89
	6	19.11 ± 5.27	0.126	21.20 ± 11.12
		P = 0.080		
Cerebellum				
Sham Control	0	5.94 ± 0.98		3.80 ± 1.07
CDMA	1.5	4.91 ± 0.58	0.671	2.00 ± 1.05
	3	5.46 ± 0.83	0.747	2.00 ± 0.63
	6	5.86 ± 0.84	0.650	1.20 ± 0.37
		P = 0.421		
Liver				
Sham Control	0	10.09 ± 0.87		7.00 ± 1.87
CDMA	1.5	15.26 ± 3.35	0.634	33.40 ± 15.11
	3	11.49 ± 2.05	1.000	12.40 ± 3.59
	6	18.35 ± 3.44	0.163	31.40 ± 12.33
		P = 0.113		
Peripheral Blood				
Sham Control	0	3.15 ± 0.40		0.20 ± 0.20
CDMA	1.5	3.77 ± 1.19	0.371	1.20 ± 0.80
	3	4.13 ± 0.54	0.361	0.40 ± 0.40
	6	6.06 ± 2.18	0.082	9.80 ± 8.81
		P = 0.048		

[a]Study was performed at ILS, Inc. The detailed protocol is presented by Recio et al.[211]. Groups of five rats per exposure group; exposure began in utero on gestation day 6. Statistical analysis runs Levene's test to determine if variances of exposed groups are equal (P = 0.05). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.
[b]Mean ± standard error.
[c]Pairwise comparison with the sham control group; exposed group values are significant at P ≤ 0.025 by Williams' or Dunn's test.
[d]No exposure to CDMA-modulated cell phone RFR.
[e]Dose-related trend derived from one-tailed linear regression or Jonckheere's test; the trend is significant when P ≤ 0.025.
[f]n = 4.

Table E-6. DNA Damage in Female Rats Exposed to GSM-modulated Cell Phone RFR for 19 Weeks (100-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b]
Frontal Cortex				
Sham Control[d]	0	7.03 ± 1.21		3.80 ± 1.46
GSM	1.5	4.87 ± 0.47	0.820	2.20 ± 0.73
	3	6.18 ± 0.67	0.843	5.60 ± 2.36
	6	6.74 ± 0.74	0.723	6.40 ± 2.73
		P = 0.386[e]		
Hippocampus				
Sham Control	0[f]	13.14 ± 1.20		9.00 ± 2.58
GSM	1.5[f]	13.22 ± 1.56	0.936	7.25 ± 3.20
	3[f]	17.67 ± 3.64	0.351	19.50 ± 7.89
	6	13.21 ± 1.03	1.000	10.00 ± 3.81
		P = 0.334		
Cerebellum				
Sham Control	0	5.94 ± 0.98		3.80 ± 1.07
GSM	1.5	5.69 ± 0.75	0.662	2.00 ± 0.71
	3	4.62 ± 0.85	0.749	0.60 ± 0.24
	6	6.62 ± 0.96	0.381	2.40 ± 1.03
		P = 0.302		
Liver				
Sham Control	0	10.09 ± 0.87		7.00 ± 1.87
GSM	1.5	9.91 ± 2.60	1.000	13.20 ± 11.23
	3	9.46 ± 2.07	1.000	17.00 ± 14.76
	6	18.99 ± 6.20	1.000	35.20 ± 19.42
		P = 0.394		
Peripheral Blood				
Sham Control	0	3.15 ± 0.40		0.20 ± 0.20
GSM	1.5	2.80 ± 0.33	0.593	0.80 ± 0.49
	3	3.39 ± 0.68	0.447	0.60 ± 0.24
	6	3.93 ± 0.63	0.203	1.00 ± 0.32
		P = 0.093		

[a]Study was performed at ILS, Inc. The detailed protocol is presented by Recio et al.[211]. Groups of five rats per exposure group; exposure began in utero on gestation day 6. Statistical analysis runs Levene's test to determine if variances of exposed groups are equal (P = 0.05). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.
[b]Mean ± standard error.
[c]Pairwise comparison with the sham control group; exposed group values are significant at P ≤ 0.025 by Williams' or Dunn's test.
[d]No exposure to GSM-modulated cell phone RFR.
[e]Dose-related trend derived from one-tailed linear regression or Jonckheere's test; the trend is significant when P ≤ 0.025.
[f]n = 4.

Table E-7. DNA Damage in Female Rats Exposed to CDMA-modulated Cell Phone RFR for 19 Weeks (150-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b,d]
Frontal Cortex				
Sham Control[e]	0	12.23 ± 2.18		0.40 ± 0.16
CDMA	1.5	25.37 ± 12.96	0.782	8.67 ± 7.67
	3	18.70 ± 5.28	0.634	1.87 ± 0.88
	6	33.49 ± 11.14	0.092	7.20 ± 5.62
		P = 0.035[f]		
Hippocampus				
Sham Control	0[g]	18.08 ± 1.30		0.83 ± 0.32
CDMA	1.5	20.58 ± 2.06	0.531	1.07 ± 0.34
	3	20.63 ± 1.92	0.382	1.33 ± 0.21
	6	29.55 ± 9.44	0.218	6.53 ± 5.23
		P = 0.068		
Cerebellum				
Sham Control	0	4.93 ± 1.09		0.00 ± 0.00
CDMA	1.5	4.61 ± 1.61	0.621	0.53 ± 0.53
	3	3.89 ± 0.43	0.709	0.13 ± 0.13
	6	5.88 ± 0.63	0.342	0.27 ± 0.16
		P = 0.249		
Liver				
Sham Control	0	12.41 ± 1.64		0.13 ± 0.13
CDMA	1.5	26.15 ± 8.57	0.145	4.00 ± 3.67
	3	16.17 ± 2.17	0.176	0.67 ± 0.42
	6	26.65 ± 6.91	0.059	2.00 ± 1.17
		P = 0.102		
Peripheral Blood				
Sham Control	0	3.32 ± 0.09		0.13 ± 0.13
CDMA	1.5	4.45 ± 1.53	1.000	0.40 ± 0.27
	3	3.94 ± 0.40	0.465	0.13 ± 0.13
	6	12.76 ± 7.59	0.028	2.93 ± 2.77
		P = 0.013		

[a]Study was performed at ILS, Inc. The detailed protocol is presented by Recio et al.[211] and OECD[184]. Groups of five rats per exposure group; exposure began in utero on gestation day 6. Statistical analysis runs Levene's test to determine if variances of exposed groups are equal (P = 0.05). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.
[b]Mean ± standard error.
[c]Pairwise comparison with the sham control group; exposed group values are significant at P ≤ 0.025 by Williams' or Dunn's test.
[d]Percent hedgehogs = estimated as the number of comets with ≥90% tail DNA/150.
[e]No exposure to CDMA-modulated cell phone RFR.
[f]Dose-related trend derived from one-tailed linear regression or Jonckheere's test; the trend is significant when P ≤ 0.025.
[g]n = 4.

Table E-8. DNA Damage in Female Rats Exposed to GSM-modulated Cell Phone RFR for 19 Weeks (150-Cell Method)[a]

	Dose (W/kg)	Percent Tail DNA[b]	P Value[c]	Percent Hedgehogs[b,d]
Frontal Cortex				
Sham Controle	0	12.23 ± 2.18		0.40 ± 0.16
GSM	1.5	6.28 ± 1.00	0.856	0.00 ± 0.00
	3	9.83 ± 1.11	0.877	0.67 ± 0.21
	6	13.74 ± 2.79	0.376	0.13 ± 0.13
		P = 0.137[f]		
Hippocampus				
Sham Control	0[g]	18.08 ± 1.30		0.83 ± 0.32
GSM	1.5[g]	17.54 ± 3.59	1.000	1.50 ± 1.29
	3[g]	28.08 ± 7.0	0.662	3.66 ± 1.40
	6	18.19 ± 3.35	1.000	2.93 ± 1.53
		P = 0.534		
Cerebellum				
Sham Control	0	4.93 ± 1.09		0.00 ± 0.00
GSM	1.5	5.11 ± 0.63	0.731	0.00 ± 0.00
	3	3.51 ± 0.74	1.000	0.00 ± 0.00
	6	6.54 ± 2.33	1.000	0.27 ± 0.16
		P = 0.705		
Liver				
Sham Control	0	12.41 ± 1.64		0.13 ± 0.13
GSM	1.5	17.05 ± 7.24	1.000	0.93 ± 0.62
	3	14.06 ± 5.68	1.000	0.27 ± 0.16
	6	26.03 ± 10.69	1.000	4.00 ± 3.23
		P = 0.580		
Peripheral Blood				
Sham Control	0	3.32 ± 0.09		0.13 ± 0.13
GSM	1.5	3.07 ± 0.43	1.000	0.27 ± 0.16
	3	2.82 ± 0.52	1.000	0.13 ± 0.13
	6	3.86 ± 0.76	1.000	0.40 ± 0.16
		P = 0.580		

[a]Study was performed at ILS, Inc. The detailed protocol is presented by Recio et al.[211] and OECD[184]. Groups of five rats per exposure group; exposure began in utero on gestation day 6. Statistical analysis runs Levene's test to determine if variances of exposed groups are equal (P = 0.05). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.
[b]Mean ± standard error.
[c]Pairwise comparison with the sham control group; exposed group values are significant at P ≤ 0.025 by Williams' or Dunn's test.
[d]Percent hedgehogs = estimated as the number of comets with ≥90% tail DNA/150.
[e]No exposure to GSM-modulated cell phone RFR.
[f]Dose-related trend derived from one-tailed linear regression or Jonckheere's test; the trend is significant when P ≤ 0.025.
[g]n = 4.

Table E-9. Frequency of Micronuclei in Peripheral Blood Erythrocytes of Rats Following Exposure to CDMA- or GSM-modulated Cell Phone RFR for 19 Weeks[a]

	Dose (W/kg)	Number of Rats with Erythrocytes Scored	Micronucleated PCEs/ 1,000 PCEs[b]	P Value[c]	Micronucleated NCEs/ 1,000 NCEs[b]	P Value[c]	PCEs[b] (%)	P Value[c]
Male								
Sham Control[d]	0	5	0.84 ± 0.10		0.33 ± 0.11		0.95 ± 0.05	
CDMA	1.5	5	0.56 ± 0.02	0.989	0.12 ± 0.02	1.000	0.83 ± 0.07	0.588
	3	5	0.55 ± 0.06	0.997	0.13 ± 0.02	1.000	0.88 ± 0.05	0.700
	6	5	0.43 ± 0.07	0.998	0.13 ± 0.05	1.000	0.99 ± 0.07	0.741
			P = 0.999[e]		P = 0.970		P = 0.389	
GSM	1.5	5	0.61 ± 0.11	0.920	0.14 ± 0.04	1.000	1.03 ± 0.03	0.352
	3	5	0.60 ± 0.11	0.961	0.08 ± 0.02	1.000	1.00 ± 0.06	0.425
	6	5	0.49 ± 0.08	0.972	0.13 ± 0.02	1.000	1.08 ± 0.06	0.114
			P = 0.985		P = 0.911		P = 0.123	
Female								
Sham Control	0	5	0.62 ± 0.07		0.13 ± 0.04		0.66 ± 0.08	
CDMA	1.5	5	0.54 ± 0.08	1.000	0.18 ± 0.03	0.263	0.92 ± 0.17	0.337
	3	5	0.72 ± 0.12	0.778	0.16 ± 0.02	0.316	0.73 ± 0.12	0.406
	6	5	0.51 ± 0.04	1.000	0.19 ± 0.06	0.219	0.82 ± 0.05	0.297
			P = 0.541		P = 0.212		P = 0.430	
GSM	1.5	5	0.61 ± 0.10	0.519	0.20 ± 0.04	0.377	0.76 ± 0.07	0.376
	3	5	0.70 ± 0.08	0.495	0.11 ± 0.02	0.447	0.74 ± 0.09	0.455
	6	5	0.59 ± 0.07	0.525	0.13 ± 0.03	0.476	0.99 ± 0.03	0.010
			P = 0.566		P = 0.737		P = 0.008	

[a]Study was performed at ILS, Inc. The detailed protocol is presented by Witt et al.[215]. Exposure began in utero on gestation day 6; NCE = normochromatic erythrocyte; PCE = polychromatic erythrocyte. Statistical analysis runs Levene's test to determine if variances of exposed groups are equal (P = 0.05). If variances are significantly different, Jonckheere's test is used for trend with Dunn's test for pairwise comparisons; otherwise, linear regression is used to test for trend and Williams' test for pairwise comparisons.
[b]Mean ± standard error.
[c]Pairwise comparison with the sham control group; exposed group values are significant at P ≤ 0.025 by Williams' or Dunn's test.
[d]No exposure to CDMA- or GSM-modulated cell phone RFR.
[e]Dose-related trend derived from one-tailed linear regression or Jonckheere's test; the trend is significant when P ≤ 0.025.

Appendix F. Clinical Pathology Results

Tables

Table F-1. Clinical Pathology Data for Rats at the 14-week Interim Evaluation in the Two-year GSM-modulated Cell Phone RFR Study F-2
Table F-2. Clinical Pathology Data for Rats at the 14-week Interim Evaluation in the Two-year CDMA-modulated Cell Phone RFR Study F-4

Table F-1. Clinical Pathology Data for Rats at the 14-week Interim Evaluation in the Two-year GSM-modulated Cell Phone RFR Study[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Male				
Hematology				
Hematocrit (%)	52.0 ± 0.3	51.7 ± 0.2	51.8 ± 0.6	52.4 ± 0.4
Manual hematocrit (%)	50 ± 0	50 ± 0	50 ± 1	51 ± 1
Hemoglobin (g/dL)	16.5 ± 0.1	16.5 ± 0.1	16.6 ± 0.2	16.7 ± 0.2
Erythrocytes (106/µL)	8.88 ± 0.07	8.92 ± 0.05	9.15 ± 0.10	8.94 ± 0.08
Reticulocytes (106/µL)	270.4 ± 3.9	259.4 ± 7.3	258.2 ± 7.0	265.6 ± 9.5
Nucleated erythrocytes/100 leukocytes	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.10 ± 0.10
Mean cell volume (fL)	58.5 ± 0.4	58.0 ± 0.5	56.7 ± 0.5	58.6 ± 0.5
Mean cell hemoglobin (pg)	18.6 ± 0.1	18.5 ± 0.1	18.2 ± 0.2	18.8 ± 0.2
Mean cell hemoglobin concentration (g/dL)	31.7 ± 0.1	31.9 ± 0.1	32.0 ± 0.2	32.0 ± 0.2
Platelets (103/µL)	894 ± 41	896 ± 34	954 ± 57	896 ± 24
Leukocytes (103/µL)	9.34 ± 0.50	9.23 ± 0.45	9.38 ± 0.52	9.63 ± 0.65
Segmented neutrophils (103/µL)	1.19 ± 0.09	1.02 ± 0.06	1.29 ± 0.11	1.33 ± 0.11
Lymphocytes (103/µL)	7.66 ± 0.48	7.78 ± 0.43	7.68 ± 0.53	7.87 ± 0.59
Monocytes (103/µL)	0.26 ± 0.03	0.22 ± 0.02	0.20 ± 0.02	0.22 ± 0.02
Basophils (103/µL)	0.05 ± 0.01	0.05 ± 0.00	0.05 ± 0.01	0.05 ± 0.01
Eosinophils (103/µL)	0.10 ± 0.01	0.08 ± 0.01	0.09 ± 0.01	0.09 ± 0.01
Large unstained cells (103/µL)	0.09 ± 0.01	0.09 ± 0.01	0.07 ± 0.01	0.08 ± 0.01
Clinical Chemistry				
Urea nitrogen (mg/dL)	18.1 ± 0.6	17.6 ± 0.4	17.7 ± 0.4	18.1 ± 0.5
Creatinine (mg/dL)	0.50 ± 0.01	0.54 ± 0.02	0.54 ± 0.01*	0.56 ± 0.01**
Glucose (mg/dL)	135 ± 6	134 ± 4	127 ± 2	128 ± 3
Total protein (g/dL)	6.1 ± 0.1	6.2 ± 0.1	6.1 ± 0.1	6.3 ± 0.1
Albumin (g/dL)	3.6 ± 0.0	3.7 ± 0.1	3.7 ± 0.0	3.7 ± 0.0
Cholesterol (mg/dL)	92 ± 2	86 ± 3	87 ± 3	88 ± 4
Triglycerides (mg/dL)	88 ± 6	88 ± 10	98 ± 6	96 ± 6
Alanine aminotransferase (IU/L)	55 ± 2	49 ± 1	57 ± 4	51 ± 2
Alkaline phosphatase (IU/L)	284 ± 12	316 ± 19	315 ± 19	318 ± 16
Creatine kinase (IU/L)	277 ± 83	467 ± 161	284 ± 68	664 ± 266
Sorbitol dehydrogenase (IU/L)	33 ± 1	33 ± 1	34 ± 1	33 ± 1
Bile salt/acids (µmol/L)	27.3 ± 3.8	20.5 ± 3.1	33.0 ± 4.8	29.6 ± 4.8

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Female				
Hematology				
Hematocrit (%)	48.1 ± 0.5	49.1 ± 0.5	48.6 ± 0.4	48.7 ± 0.6
Manual hematocrit (%)	47 ± 0	49 ± 0*	49 ± 0**	49 ± 0*
Hemoglobin (g/dL)	15.6 ± 0.2	15.9 ± 0.1	15.8 ± 0.1	15.8 ± 0.1
Erythrocytes (106/µL)	8.15 ± 0.07	8.33 ± 0.12	8.29 ± 0.06	8.18 ± 0.11
Reticulocytes (106/µL)	245.7 ± 14.9	232.8 ± 6.3	241.9 ± 13.8	273.1 ± 17.6
Nucleated erythrocytes/100 leukocytes	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Mean cell volume (fL)	58.9 ± 0.3	59.0 ± 0.6	58.7 ± 0.6	59.5 ± 0.5
Mean cell hemoglobin (pg)	19.1 ± 0.1	19.2 ± 0.2	19.0 ± 0.2	19.3 ± 0.2
Mean cell hemoglobin concentration (g/dL)	32.4 ± 0.1	32.5 ± 0.2	32.5 ± 0.2	32.3 ± 0.2
Platelets (103/µL)	963 ± 40	933 ± 33	1,022 ± 38	954 ± 55
Leukocytes (103/µL)	8.80 ± 0.50	7.90 ± 0.67	6.86 ± 0.51*	7.34 ± 0.41
Segmented neutrophils (103/µL)	1.15 ± 0.12	0.88 ± 0.09	0.93 ± 0.13	1.02 ± 0.10
Lymphocytes (103/µL)	7.22 ± 0.40	6.66 ± 0.61	5.60 ± 0.40**	6.00 ± 0.37
Monocytes (103/µL)	0.19 ± 0.02	0.15 ± 0.01	0.16 ± 0.02	0.14 ± 0.01
Basophils (103/µL)	0.05 ± 0.01	0.04 ± 0.01	0.03 ± 0.00*	0.03 ± 0.00*
Eosinophils (103/µL)	0.11 ± 0.02	0.10 ± 0.01	0.08 ± 0.01	0.09 ± 0.01
Large unstained cells (103/µL)	0.09 ± 0.01	0.07 ± 0.01	0.06 ± 0.01**	0.06 ± 0.01**
Clinical Chemistry				
Urea nitrogen (mg/dL)	17.5 ± 0.9	18.1 ± 0.8	16.9 ± 0.5	15.9 ± 0.9
Creatinine (mg/dL)	0.57 ± 0.03	0.56 ± 0.03	0.56 ± 0.02	0.63 ± 0.02
Glucose (mg/dL)	151 ± 9	146 ± 9	141 ± 5	146 ± 6
Total protein (g/dL)	6.2 ± 0.1	6.2 ± 0.1	6.3 ± 0.1	6.3 ± 0.1
Albumin (g/dL)	3.8 ± 0.0	3.8 ± 0.0	3.9 ± 0.0	3.9 ± 0.1
Cholesterol (mg/dL)	78 ± 4	72 ± 3	69 ± 3	64 ± 3**
Triglycerides (mg/dL)	70 ± 9	51 ± 4	61 ± 8	44 ± 5*
Alanine aminotransferase (IU/L)	48 ± 2	41 ± 2	43 ± 2	41 ± 2
Alkaline phosphatase (IU/L)	277 ± 24	273 ± 16	265 ± 13	226 ± 12
Creatine kinase (IU/L)	209 ± 35	295 ± 54	218 ± 27	275 ± 68
Sorbitol dehydrogenase (IU/L)	31 ± 4	28 ± 5	28 ± 4	32 ± 4
Bile salt/acids (µmol/L)	33.7 ± 6.1	22.8 ± 3.7	28.0 ± 4.5	31.5 ± 6.3

*Significantly different ($P \leq 0.05$) from the sham control group by Dunn's or Shirley's test.
**$P \leq 0.01$.
[a]Data are presented as mean ± standard error. Statistical analysis performed by Jonckheere (trend) and Shirley's or Dunn's tests.

Table F-2. Clinical Pathology Data for Rats at the 14-week Interim Evaluation in the Two-year CDMA-modulated Cell Phone RFR Study[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Male				
Hematology				
Hematocrit (%)	52.0 ± 0.3	51.9 ± 0.4	51.4 ± 0.5	51.9 ± 0.8
Manual hematocrit (%)	50 ± 0	50 ± 1	50 ± 1	50 ± 1[b]
Hemoglobin (g/dL)	16.5 ± 0.1	16.6 ± 0.1	16.6 ± 0.1	16.7 ± 0.3
Erythrocytes (106/µL)	8.88 ± 0.07	8.92 ± 0.07	8.91 ± 0.10	8.82 ± 0.13
Reticulocytes (106/µL)	270.4 ± 3.9	266.8 ± 5.7	257.4 ± 5.6	274.8 ± 15.1
Nucleated erythrocytes/100 leukocytes	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Mean cell volume (fL)	58.5 ± 0.4	58.2 ± 0.4	57.7 ± 0.5	58.9 ± 0.5
Mean cell hemoglobin (pg)	18.6 ± 0.1	18.7 ± 0.1	18.6 ± 0.1	18.9 ± 0.1
Mean cell hemoglobin concentration (g/dL)	31.7 ± 0.1	32.0 ± 0.2	32.3 ± 0.2	32.1 ± 0.2
Platelets (103/µL)	894 ± 41	839 ± 26	911 ± 22	926 ± 35
Leukocytes (103/µL)	9.34 ± 0.50	10.46 ± 0.64	10.08 ± 0.55	9.52 ± 0.88
Segmented neutrophils (103/µL)	1.19 ± 0.09	1.06 ± 0.14	1.11 ± 0.06	1.03 ± 0.10
Lymphocytes (103/µL)	7.66 ± 0.48	8.94 ± 0.64	8.49 ± 0.52	8.09 ± 0.82
Monocytes (103/µL)	0.26 ± 0.03	0.22 ± 0.03	0.21 ± 0.03	0.20 ± 0.03
Basophils (103/µL)	0.05 ± 0.01	0.06 ± 0.01	0.05 ± 0.00	0.05 ± 0.01
Eosinophils (103/µL)	0.10 ± 0.01	0.10 ± 0.01	0.13 ± 0.04	0.07 ± 0.01
Large unstained cells (103/µL)	0.09 ± 0.01	0.08 ± 0.01	0.09 ± 0.01	0.07 ± 0.01
Clinical Chemistry				
Urea nitrogen (mg/dL)	18.1 ± 0.6	17.2 ± 0.4	17.5 ± 0.4	18.5 ± 0.6
Creatinine (mg/dL)	0.50 ± 0.01	0.50 ± 0.01	0.54 ± 0.02	0.59 ± 0.02**
Glucose (mg/dL)	135 ± 6	133 ± 5	149 ± 8	154 ± 11
Total protein (g/dL)	6.1 ± 0.1	6.1 ± 0.0	6.3 ± 0.1	6.2 ± 0.1
Albumin (g/dL)	3.6 ± 0.0	3.6 ± 0.0	3.6 ± 0.0	3.7 ± 0.0
Cholesterol (mg/dL)	92 ± 2	89 ± 2	92 ± 3	88 ± 3
Triglycerides (mg/dL)	88 ± 6	84 ± 7	87 ± 5	72 ± 5
Alanine aminotransferase (IU/L)	55 ± 2	48 ± 2	52 ± 3	50 ± 2
Alkaline phosphatase (IU/L)	284 ± 12	287 ± 16	300 ± 15	309 ± 26
Creatine kinase (IU/L)	277 ± 83	265 ± 31	387 ± 109	309 ± 64
Sorbitol dehydrogenase (IU/L)	33 ± 1	31 ± 1	33 ± 2	35 ± 1
Bile salt/acids (µmol/L)	27.3 ± 3.8	36.3 ± 4.5	33.7 ± 5.0	30.1 ± 5.4

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Female				
Hematology				
Hematocrit (%)	48.1 ± 0.5	48.9 ± 0.3	48.7 ± 0.4	48.4 ± 0.4
Manual hematocrit (%)	47 ± 0	48 ± 0	48 ± 0*	48 ± 0
Hemoglobin (g/dL)	15.6 ± 0.2	15.8 ± 0.1	15.8 ± 0.1	15.7 ± 0.1
Erythrocytes (106/µL)	8.15 ± 0.07	8.13 ± 0.08	8.23 ± 0.03	8.17 ± 0.08
Reticulocytes (106/µL)	245.7 ± 14.9	282.5 ± 10.3	268.0 ± 19.8	267.9 ± 14.4
Nucleated erythrocytes/100 leukocytes	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
Mean cell volume (fL)	58.9 ± 0.3	60.2 ± 0.4	59.2 ± 0.5	59.3 ± 0.7
Mean cell hemoglobin (pg)	19.1 ± 0.1	19.5 ± 0.2	19.2 ± 0.1	19.3 ± 0.2
Mean cell hemoglobin concentration (g/dL)	32.4 ± 0.1	32.4 ± 0.1	32.4 ± 0.2	32.6 ± 0.2
Platelets (103/µL)	963 ± 40	978 ± 36	989 ± 38	983 ± 33
Leukocytes (103/µL)	8.80 ± 0.50	8.12 ± 0.83	7.82 ± 0.56	8.83 ± 0.97
Segmented neutrophils (103/µL)	1.15 ± 0.12	0.96 ± 0.15	0.98 ± 0.11	1.28 ± 0.28
Lymphocytes (103/µL)	7.22 ± 0.40	6.75 ± 0.67	6.47 ± 0.48	7.12 ± 0.68
Monocytes (103/µL)	0.19 ± 0.02	0.20 ± 0.03	0.18 ± 0.03	0.19 ± 0.03
Basophils (103/µL)	0.05 ± 0.01	0.04 ± 0.01	0.03 ± 0.00	0.04 ± 0.01
Eosinophils (103/µL)	0.11 ± 0.02	0.09 ± 0.01	0.09 ± 0.01	0.12 ± 0.03
Large unstained cells (103/µL)	0.09 ± 0.01	0.07 ± 0.01	0.06 ± 0.01*	0.08 ± 0.01
Clinical Chemistry				
Urea nitrogen (mg/dL)	17.5 ± 0.9	17.9 ± 0.5	17.0 ± 0.6	16.7 ± 0.5
Creatinine (mg/dL)	0.57 ± 0.03	0.58 ± 0.03	0.56 ± 0.01	0.59 ± 0.02
Glucose (mg/dL)	151 ± 9	145 ± 9	140 ± 5	141 ± 7
Total protein (g/dL)	6.2 ± 0.1	6.2 ± 0.1	6.3 ± 0.1	6.2 ± 0.1
Albumin (g/dL)	3.8 ± 0.0	3.8 ± 0.0	3.9 ± 0.0	3.8 ± 0.0
Cholesterol (mg/dL)	78 ± 4	71 ± 2	73 ± 3	71 ± 2
Triglycerides (mg/dL)	70 ± 9	61 ± 5	54 ± 3	70 ± 8
Alanine aminotransferase (IU/L)	48 ± 2	43 ± 2	44 ± 2	45 ± 1
Alkaline phosphatase (IU/L)	277 ± 24	248 ± 16	257 ± 18	252 ± 17
Creatine kinase (IU/L)	209 ± 35	212 ± 36	217 ± 41	418 ± 97
Sorbitol dehydrogenase (IU/L)	31 ± 4	31 ± 4	28 ± 4	34 ± 5
Bile salt/acids (µmol/L)	33.7 ± 6.1	36.8 ± 5.1	32.7 ± 5.8	29.9 ± 5.6

*Significantly different ($P \leq 0.05$) from the sham control group by Dunn's or Shirley's test.
**$P \leq 0.01$.
[a]Data are presented as mean ± standard error. Statistical analysis performed by Jonckheere (trend) and Shirley's or Dunn's tests.
[b]n = 9..

Appendix G. Mean Body Temperatures, Organ Weights, and Organ-Weight-to-Body-Weight Ratios

Tables

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Table G-1. Mean Body Temperatures for Rats by Litter in the 28-day GSM-modulated Cell Phone RFR Study[a]

Day[b]	Sham Control		3 W/kg		6 W/kg		9 W/kg	
	Temperature (°C)	No. Litters Measured	Temperature (°C)	No. Litters Measured	Temperature (°C)	No. Litters Measured	Temperature (°C)	No. Litters Measured
Male								
16	37.3 ± 0.1	4	37.1 ± 0.1	4	37.3 ± 0.1	4	37.3 ± 0.1	4
20	37.6 ± 0.1	4	37.0 ± 0.1**	4	37.3 ± 0.1	4	37.4 ± 0.1	4
27	37.2 ± 0.1	4	37.0 ± 0.1	4	37.2 ± 0.1	3	37.4 ± 0.1	4
16–27[c]	37.4 ± 0.1	4	37.0 ± 0.1*	4	37.3 ± 0.1	4[d]	37.4 ± 0.1	4
Female								
16	37.9 ± 0.2	4	37.0 ± 0.2**	4	37.1 ± 0.1*	4	37.4 ± 0.1	4
20	38.0 ± 0.2	4	37.5 ± 0.2	4	37.1 ± 0.1**	4	37.6 ± 0.2	4
27	37.9 ± 0.2	4	38.0 ± 0.2	4	37.4 ± 0.3	4	37.6 ± 0.2	4
16–27[c]	37.9 ± 0.1▲	4	37.5 ± 0.1	4	37.2 ± 0.1**	4	37.5 ± 0.1	4

▲Significant trend ($P \leq 0.05$) by Jonckheere's test.
*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Temperatures are given as mean ± standard error. Statistical analysis for linear trends was performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dosed groups to the sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method.
[b]Postnatal day.
[c]Average was calculated as the mean of the litter means of the individual animal averages over the time range.
[d]There were three litters on day 27.

Table G-2. Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats by Litter in the 28-day GSM-modulated Cell Phone RFR Study[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	4	4	4	4
Males				
Necropsy body wt.	251 ± 6	230 ± 2*	227 ± 2*	202 ± 7**
R. Adrenal gland				
Absolute	0.0253 ± 0.0012	0.0236 ± 0.0009	0.0240 ± 0.0006	0.0198 ± 0.0021
Relative	0.10 ± 0.00	0.10 ± 0.00	0.11 ± 0.00	0.10 ± 0.01
Brain				
Absolute	1.76 ± 0.02	1.78 ± 0.02	1.75 ± 0.03	1.66 ± 0.05
Relative	7.02 ± 0.08	7.73 ± 0.12*	7.73 ± 0.11*	8.25 ± 0.21**
Heart				
Absolute	1.02 ± 0.03	0.94 ± 0.02	0.88 ± 0.03*	0.80 ± 0.04**
Relative	4.06 ± 0.08	4.07 ± 0.11	3.89 ± 0.11	3.97 ± 0.09
R. Kidney				
Absolute	1.10 ± 0.05	1.04 ± 0.01	0.98 ± 0.03	0.84 ± 0.03**
Relative	4.36 ± 0.12	4.52 ± 0.02	4.30 ± 0.12	4.16 ± 0.06
Liver				
Absolute	12.05 ± 0.35	11.04 ± 0.36	10.77 ± 0.16	9.75 ± 0.40**
Relative	47.93 ± 0.66	47.99 ± 1.17	47.47 ± 0.69	48.30 ± 1.18
Lung				
Absolute	2.23 ± 0.13	1.93 ± 0.07	1.77 ± 0.09	1.81 ± 0.17
Relative	8.93 ± 0.69	8.40 ± 0.36	7.82 ± 0.43	9.00 ± 0.96
R. Testis				
Absolute	1.473 ± 0.056	1.504 ± 0.038	1.455 ± 0.047	1.324 ± 0.028
Relative	5.86 ± 0.14	6.54 ± 0.13*	6.42 ± 0.23*	6.56 ± 0.10*
Thymus				
Absolute	0.742 ± 0.060	0.520 ± 0.027*	0.672 ± 0.068	0.584 ± 0.017
Relative	2.95 ± 0.23	2.26 ± 0.10	2.96 ± 0.31	2.91 ± 0.14
Females				
Necropsy body wt.	168 ± 3	161 ± 5	167 ± 5	155 ± 4
R. Adrenal gland				
Absolute	0.0299 ± 0.0027	0.0263 ± 0.0019	0.0280 ± 0.0004	0.0253 ± 0.0017
Relative	0.18 ± 0.01	0.16 ± 0.01	0.17 ± 0.01	0.16 ± 0.01
Brain				
Absolute	1.64 ± 0.01	1.64 ± 0.02	1.68 ± 0.03	1.61 ± 0.02
Relative	9.77 ± 0.22	10.27 ± 0.30	10.05 ± 0.22	10.36 ± 0.16
Heart				
Absolute	0.73 ± 0.02	0.69 ± 0.02	0.71 ± 0.02	0.68 ± 0.02

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Relative	4.35 ± 0.09	4.31 ± 0.07	4.27 ± 0.04	4.37 ± 0.06
R. Kidney				
Absolute	0.76 ± 0.02	0.73 ± 0.02	0.75 ± 0.02	0.68 ± 0.01
Relative	4.49 ± 0.10	4.53 ± 0.10	4.47 ± 0.07	4.38 ± 0.04
Liver				
Absolute	7.65 ± 0.20	7.29 ± 0.28	7.74 ± 0.21	7.10 ± 0.21
Relative	45.53 ± 0.65	45.36 ± 0.78	46.28 ± 0.22	45.71 ± 0.88
Lung				
Absolute	1.52 ± 0.08	1.52 ± 0.03	1.50 ± 0.10	1.38 ± 0.04
Relative	9.03 ± 0.51	9.49 ± 0.51	8.93 ± 0.56	8.92 ± 0.43
Thymus				
Absolute	0.453 ± 0.059	0.344 ± 0.015	0.444 ± 0.032	0.405 ± 0.035
Relative	2.71 ± 0.38	2.13 ± 0.05	2.64 ± 0.15	2.61 ± 0.23

*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Organ weights (absolute weights) and body weights are given in grams as means of litter means; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error). Statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dosed groups to the sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method.

Table G-3. Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats in the 28-day GSM-modulated Cell Phone RFR Study[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	10	10	10	10
Males				
Necropsy body wt.	249 ± 3	231 ± 2*	227 ± 2*	205 ± 6**
R. Adrenal gland				
Absolute	0.0247 ± 0.0011	0.0237 ± 0.0012	0.0238 ± 0.0009	0.0206 ± 0.0013
Relative	0.10 ± 0.00	0.10 ± 0.00	0.11 ± 0.00	0.10 ± 0.01
Brain				
Absolute	1.75 ± 0.01	1.77 ± 0.02	1.75 ± 0.02	1.69 ± 0.03
Relative	7.05 ± 0.07	7.68 ± 0.12*	7.73 ± 0.08*	8.29 ± 0.17**
Heart				
Absolute	1.02 ± 0.02	0.94 ± 0.02	0.90 ± 0.02*	0.82 ± 0.03**
Relative	4.09 ± 0.06	4.06 ± 0.08	3.95 ± 0.06	4.02 ± 0.08
R. Kidney				
Absolute	1.08 ± 0.03	1.04 ± 0.01	0.98 ± 0.02	0.85 ± 0.03**
Relative	4.32 ± 0.09	4.51 ± 0.04	4.33 ± 0.08	4.17 ± 0.05
Liver				
Absolute	11.85 ± 0.22	11.25 ± 0.22	10.86 ± 0.18	9.81 ± 0.30**

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Relative	47.66 ± 0.63	48.61 ± 0.66	47.84 ± 0.56	47.98 ± 0.77
Lung				
Absolute	2.24 ± 0.12	1.90 ± 0.09	1.81 ± 0.08	1.87 ± 0.12
Relative	9.05 ± 0.56	8.24 ± 0.40	8.00 ± 0.37	9.25 ± 0.70
R. Testis				
Absolute	1.464 ± 0.036	1.516 ± 0.039	1.482 ± 0.029	1.330 ± 0.041
Relative	5.88 ± 0.11	6.55 ± 0.14*	6.54 ± 0.14*	6.50 ± 0.09*
Thymus				
Absolute	0.757 ± 0.041	0.536 ± 0.019*	0.637 ± 0.036	0.576 ± 0.013
Relative	3.04 ± 0.15	2.31 ± 0.07	2.81 ± 0.16	2.83 ± 0.10
Females				
Necropsy body wt.	167 ± 2	163 ± 4	168 ± 3	156 ± 3
R. Adrenal gland				
Absolute	0.0285 ± 0.0019	0.0266 ± 0.0016	0.0280 ± 0.0012	0.0259 ± 0.0017
Relative	0.17 ± 0.01	0.16 ± 0.01	0.17 ± 0.01	0.17 ± 0.01
Brain				
Absolute	1.64 ± 0.01	1.64 ± 0.02	1.67 ± 0.02	1.60 ± 0.02
Relative	9.86 ± 0.15	10.13 ± 0.23	9.96 ± 0.14	10.31 ± 0.14
Heart				
Absolute	0.72 ± 0.01	0.70 ± 0.02	0.72 ± 0.02	0.68 ± 0.01
Relative	4.34 ± 0.07	4.26 ± 0.05	4.25 ± 0.05	4.34 ± 0.05
R. Kidney				
Absolute	0.74 ± 0.02	0.73 ± 0.02	0.74 ± 0.02	0.68 ± 0.01
Relative	4.46 ± 0.06	4.48 ± 0.07	4.43 ± 0.05	4.38 ± 0.07
Liver				
Absolute	7.55 ± 0.14	7.44 ± 0.24	7.80 ± 0.19	7.04 ± 0.14
Relative	45.35 ± 0.44	45.56 ± 0.64	46.38 ± 0.40	45.18 ± 0.42
Lung				
Absolute	1.52 ± 0.07	1.50 ± 0.05	1.54 ± 0.09	1.36 ± 0.04
Relative	9.11 ± 0.40	9.22 ± 0.37	9.16 ± 0.44	8.78 ± 0.38
Thymus				
Absolute	0.474 ± 0.036	0.349 ± 0.017	0.444 ± 0.025	0.386 ± 0.021
Relative	2.86 ± 0.23	2.13 ± 0.07	2.63 ± 0.12	2.48 ± 0.13

*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error). Statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dosed groups to the sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method.

Table G-4. Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats at the 14-week Interim Evaluation in the Two-year GSM-modulated Cell Phone RFR Study[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Males				
Necropsy body wt.	444 ± 9	426 ± 10	430 ± 7	410 ± 7*
Brain				
Absolute	1.92 ± 0.02	1.94 ± 0.02	1.94 ± 0.02	1.88 ± 0.02
Relative	4.34 ± 0.08	4.58 ± 0.11	4.53 ± 0.06	4.59 ± 0.06
R. Epididymis				
Absolute	0.6423 ± 0.0278	0.6370 ± 0.0191	0.6245 ± 0.0173	0.6332 ± 0.0312
Relative	1.45 ± 0.06	1.50 ± 0.05	1.46 ± 0.06	1.54 ± 0.06
L. Epididymis				
Absolute	0.6404 ± 0.0208	0.6430 ± 0.0178	0.6219 ± 0.0168	0.6435 ± 0.0290
Relative	1.44 ± 0.05	1.51 ± 0.04	1.45 ± 0.05	1.56 ± 0.05
Heart				
Absolute	1.52 ± 0.03	1.46 ± 0.03	1.42 ± 0.04	1.44 ± 0.03
Relative	3.42 ± 0.06	3.42 ± 0.04	3.30 ± 0.05	3.51 ± 0.04
R. Kidney				
Absolute	1.59 ± 0.04	1.43 ± 0.06*	1.50 ± 0.02	1.41 ± 0.04*
Relative	3.58 ± 0.09	3.37 ± 0.11	3.50 ± 0.06	3.44 ± 0.07
L. Kidney				
Absolute	1.58 ± 0.04	1.43 ± 0.05*	1.47 ± 0.02*	1.35 ± 0.05**
Relative	3.57 ± 0.09	3.34 ± 0.09	3.42 ± 0.05	3.28 ± 0.09*
Liver				
Absolute	16.49 ± 0.31	14.97 ± 0.46	15.23 ± 0.35	14.85 ± 0.59*
Relative	37.17 ± 0.61	35.12 ± 0.63	35.47 ± 0.73	36.12 ± 0.97
Lung				
Absolute	2.20 ± 0.06	2.08 ± 0.06	2.03 ± 0.06	2.00 ± 0.08
Relative	4.96 ± 0.15	4.89 ± 0.13	4.73 ± 0.16	4.88 ± 0.17
R. Testis				
Absolute	2.071 ± 0.051	1.982 ± 0.049	2.055 ± 0.041	1.930 ± 0.056
Relative	4.68 ± 0.16	4.66 ± 0.11	4.80 ± 0.15	4.70 ± 0.10
L. Testis				
Absolute	2.083 ± 0.059	2.028 ± 0.051	2.049 ± 0.039	1.954 ± 0.055
Relative	4.71 ± 0.17	4.77 ± 0.11	4.79 ± 0.15	4.76 ± 0.08
Thymus				
Absolute	0.349 ± 0.023	0.361 ± 0.026	0.372 ± 0.024	0.360 ± 0.017
Relative	0.79 ± 0.05	0.84 ± 0.04	0.86 ± 0.05	0.87 ± 0.03

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Females				
Necropsy body wt.	274 ± 8	249 ± 6*	265 ± 8	244 ± 5**
Brain				
Absolute	1.79 ± 0.02	1.80 ± 0.03	1.81 ± 0.02	1.78 ± 0.02
Relative	6.58 ± 0.18	7.28 ± 0.14**	6.90 ± 0.18	7.33 ± 0.11**
Heart				
Absolute	1.02 ± 0.03	0.95 ± 0.02	0.98 ± 0.03	0.94 ± 0.03
Relative	3.74 ± 0.09	3.84 ± 0.08	3.70 ± 0.06	3.87 ± 0.04
R. Kidney				
Absolute	0.97 ± 0.03	0.91 ± 0.02	0.89 ± 0.02*	0.85 ± 0.02**
Relative	3.54 ± 0.08	3.65 ± 0.05	3.37 ± 0.10	3.50 ± 0.07
L. Kidney				
Absolute	0.97 ± 0.04	0.87 ± 0.02*	0.88 ± 0.02*	0.83 ± 0.02**
Relative	3.53 ± 0.11	3.51 ± 0.04	3.33 ± 0.08	3.41 ± 0.08
Liver				
Absolute	10.07 ± 0.56	8.41 ± 0.25*	9.36 ± 0.40	8.03 ± 0.22**
Relative	36.55 ± 1.16	33.85 ± 0.61	35.27 ± 0.63	32.94 ± 0.46**
Lung				
Absolute	1.85 ± 0.05	1.67 ± 0.05	1.90 ± 0.07	1.56 ± 0.03**
Relative	6.79 ± 0.24	6.74 ± 0.18	7.22 ± 0.29	6.42 ± 0.11
R. Ovary				
Absolute	0.0598 ± 0.0042	0.0581 ± 0.0047	0.0569 ± 0.0038	0.0565 ± 0.0025
Relative	0.22 ± 0.02	0.23 ± 0.02	0.21 ± 0.01	0.23 ± 0.01
L. Ovary				
Absolute	0.0511 ± 0.0031	0.0496 ± 0.0040	0.0532 ± 0.0045	0.0521 ± 0.0035
Relative	0.19 ± 0.01	0.20 ± 0.01	0.20 ± 0.01	0.21 ± 0.01
Thymus				
Absolute	0.299 ± 0.016	0.244 ± 0.013*	0.274 ± 0.020	0.234 ± 0.013*
Relative	1.10 ± 0.06	0.98 ± 0.05	1.03 ± 0.07	0.96 ± 0.04

*Significantly different ($P \leq 0.05$) from the sham control group by Williams' or Dunnett's test.
**$P \leq 0.01$.
[a]Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error).

Table G-5. Mean Body Temperatures for Rats by Litter in the 28-day CDMA-modulated Cell Phone RFR Study[a]

Day[b]	Sham Control		3 W/kg		6 W/kg		9 W/kg	
	Temperature (°C)	No. Litters Measured	Temperature (°C)	No. Litters Measured	Temperature (°C)	No. Litters Measured	Temperature (°C)	No. Litters Measured
Male								
16	37.3 ± 0.1	4	37.0 ± 0.1	4	37.1 ± 0.1	4	37.3 ± 0.1	4
20	37.6 ± 0.1	4	37.2 ± 0.1	4	37.1 ± 0.1	4	37.5 ± 0.2	4
27	37.2 ± 0.1	4	36.8 ± 0.1	4	37.0 ± 0.1	4	37.4 ± 0.2	4
16–27[c]	37.4 ± 0.1	4	37.0 ± 0.1	4	37.1 ± 0.1	4	37.5 ± 0.2	4
Female								
16	37.9 ± 0.2	4	37.2 ± 0.2*	3	37.5 ± 0.1	4	37.5 ± 0.1	4
20	38.0 ± 0.2▲	4	38.0 ± 0.1	4	37.6 ± 0.2	4	37.6 ± 0.2	4
27	37.9 ± 0.2	4	37.1 ± 0.2	4	38.0 ± 0.2	4	38.0 ± 0.2	4
16–27[c]	37.9 ± 0.1	4	37.4 ± 0.1	4d	37.7 ± 0.1	4	37.7 ± 0.1	4

▲Significant trend ($P \leq 0.05$) by Jonckheere's test.
*Significantly different ($P \leq 0.05$) from the sham control group.
[a]Temperatures are given as mean ± standard error. Statistical analysis for linear trends was performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dosed groups to the sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method.
[b]Postnatal day.
[c]Average was calculated as the mean of the litter means of the individual animal averages over the time range.
[d]There were three litters on day 16.

Table G-6. Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats by Litter in the 28-day CDMA-modulated Cell Phone RFR Study[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	4	4	4	4
Males				
Necropsy body wt.	251 ± 6	232 ± 4	234 ± 7	216 ± 3**
R. Adrenal gland				
Absolute	0.0253 ± 0.0012	0.0242 ± 0.0021	0.0249 ± 0.0019	0.0228 ± 0.0008
Relative	0.10 ± 0.00	0.10 ± 0.01	0.11 ± 0.00	0.11 ± 0.00
Brain				
Absolute	1.76 ± 0.02	1.75 ± 0.02	1.74 ± 0.02	1.72 ± 0.02
Relative	7.02 ± 0.08	7.53 ± 0.13*	7.47 ± 0.19	7.98 ± 0.06**
Heart				
Absolute	1.02 ± 0.03	0.92 ± 0.01	0.95 ± 0.05	0.89 ± 0.04
Relative	4.06 ± 0.08	3.95 ± 0.06	4.05 ± 0.12	4.12 ± 0.13
R. Kidney				
Absolute	1.10 ± 0.05	1.04 ± 0.02	1.00 ± 0.03	0.91 ± 0.04*
Relative	4.36 ± 0.12	4.46 ± 0.04	4.27 ± 0.09	4.20 ± 0.13
Liver				
Absolute	12.05 ± 0.35	10.97 ± 0.22	11.25 ± 0.38	10.23 ± 0.17**
Relative	47.93 ± 0.66	47.20 ± 0.99	48.11 ± 0.68	47.49 ± 0.74
Lung				
Absolute	2.23 ± 0.13	1.98 ± 0.16	1.94 ± 0.09	1.96 ± 0.04
Relative	8.93 ± 0.69	8.53 ± 0.76	8.30 ± 0.49	9.13 ± 0.26
R. Testis				
Absolute	1.473 ± 0.056	1.444 ± 0.024	1.456 ± 0.050	1.410 ± 0.038
Relative	5.86 ± 0.14	6.22 ± 0.08	6.23 ± 0.04	6.54 ± 0.08**
Thymus				
Absolute	0.742 ± 0.060	0.639 ± 0.011	0.646 ± 0.023	0.553 ± 0.025**
Relative	2.95 ± 0.23	2.75 ± 0.04	2.77 ± 0.11	2.57 ± 0.12
Females				
Necropsy body wt.	168 ± 3	165 ± 5	169 ± 8	161 ± 4
R. Adrenal gland				
Absolute	0.0299 ± 0.0027	0.0250 ± 0.0003	0.0268 ± 0.0009	0.0242 ± 0.0016
Relative	0.18 ± 0.01	0.15 ± 0.00	0.16 ± 0.01	0.15 ± 0.01
Brain				
Absolute	1.64 ± 0.01	1.68 ± 0.02	1.63 ± 0.01	1.63 ± 0.03
Relative	9.77 ± 0.22	10.23 ± 0.21	9.76 ± 0.42	10.14 ± 0.14

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Heart				
Absolute	0.73 ± 0.02	0.71 ± 0.02	0.72 ± 0.03	0.70 ± 0.01
Relative	4.35 ± 0.09	4.34 ± 0.07	4.29 ± 0.10	4.33 ± 0.08
R. Kidney				
Absolute	0.76 ± 0.02	0.75 ± 0.02	0.73 ± 0.03	0.69 ± 0.02*
Relative	4.49 ± 0.10	4.53 ± 0.03	4.32 ± 0.07	4.26 ± 0.08
Liver				
Absolute	7.65 ± 0.20	7.50 ± 0.16	7.87 ± 0.42	7.25 ± 0.07
Relative	45.53 ± 0.65	45.64 ± 1.19	46.54 ± 0.53	45.11 ± 0.70
Lung				
Absolute	1.52 ± 0.08	1.48 ± 0.06	1.46 ± 0.06	1.41 ± 0.04
Relative	9.03 ± 0.51	8.98 ± 0.45	8.78 ± 0.73	8.76 ± 0.34
Thymus				
Absolute	0.453 ± 0.059	0.444 ± 0.030	0.388 ± 0.024	0.380 ± 0.024
Relative	2.71 ± 0.38	2.69 ± 0.10	2.31 ± 0.17	2.36 ± 0.09

*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Organ weights (absolute weights) and body weights are given in grams as means of litter means; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error). Statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dosed groups to the sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method.

Table G-7. Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats in the 28-day CDMA-modulated Cell Phone RFR Study[a]

	Sham Control	3 W/kg	6 W/kg	9 W/kg
n	10	10	10	10
Males				
Necropsy body wt.	249 ± 3	231 ± 3	236 ± 6	215 ± 3**
R. Adrenal gland				
Absolute	0.0247 ± 0.0011	0.0254 ± 0.0016	0.0253 ± 0.0013	0.0223 ± 0.0007
Relative	0.10 ± 0.00	0.11 ± 0.01	0.11 ± 0.00	0.10 ± 0.00
Brain				
Absolute	1.75 ± 0.01	1.75 ± 0.01	1.75 ± 0.01	1.72 ± 0.02
Relative	7.05 ± 0.07	7.59 ± 0.11*	7.43 ± 0.14	8.01 ± 0.07**
Heart				
Absolute	1.02 ± 0.02	0.91 ± 0.01	0.97 ± 0.04	0.90 ± 0.02
Relative	4.09 ± 0.06	3.95 ± 0.05	4.08 ± 0.09	4.16 ± 0.09
R. Kidney				
Absolute	1.08 ± 0.03	1.03 ± 0.02	1.00 ± 0.03	0.91 ± 0.03*
Relative	4.32 ± 0.09	4.46 ± 0.05	4.22 ± 0.07	4.24 ± 0.08

	Sham Control	3 W/kg	6 W/kg	9 W/kg
Liver				
Absolute	11.85 ± 0.22	11.05 ± 0.21	11.38 ± 0.33	10.29 ± 0.17**
Relative	47.66 ± 0.63	47.80 ± 0.58	48.13 ± 0.59	47.88 ± 0.47
Lung				
Absolute	2.24 ± 0.12	1.92 ± 0.10	1.91 ± 0.09	1.95 ± 0.08
Relative	9.05 ± 0.56	8.34 ± 0.49	8.07 ± 0.35	9.10 ± 0.42
R. Testis				
Absolute	1.464 ± 0.036	1.438 ± 0.023	1.473 ± 0.036	1.407 ± 0.034
Relative	5.88 ± 0.11	6.22 ± 0.08	6.23 ± 0.06	6.54 ± 0.12**
Thymus				
Absolute	0.757 ± 0.041	0.639 ± 0.017	0.658 ± 0.026	0.568 ± 0.022**
Relative	3.04 ± 0.15	2.77 ± 0.06	2.79 ± 0.10	2.64 ± 0.11
Females				
Necropsy body wt.	167 ± 2	163 ± 4	172 ± 6	159 ± 3
R. Adrenal gland				
Absolute	0.0285 ± 0.0019	0.0250 ± 0.0007	0.0265 ± 0.0012	0.0236 ± 0.0011
Relative	0.17 ± 0.01	0.15 ± 0.00	0.16 ± 0.01	0.15 ± 0.01
Brain				
Absolute	1.64 ± 0.01	1.67 ± 0.01	1.64 ± 0.01	1.61 ± 0.01
Relative	9.86 ± 0.15	10.33 ± 0.19	9.62 ± 0.33	10.15 ± 0.16
Heart				
Absolute	0.72 ± 0.01	0.71 ± 0.02	0.74 ± 0.02	0.69 ± 0.01
Relative	4.34 ± 0.07	4.34 ± 0.06	4.29 ± 0.08	4.33 ± 0.06
R. Kidney				
Absolute	0.74 ± 0.02	0.73 ± 0.02	0.74 ± 0.02	0.67 ± 0.01*
Relative	4.46 ± 0.06	4.51 ± 0.03	4.32 ± 0.06	4.23 ± 0.06
Liver				
Absolute	7.55 ± 0.14	7.52 ± 0.16	8.04 ± 0.32	7.23 ± 0.17
Relative	45.35 ± 0.44	46.25 ± 0.64	46.77 ± 0.55	45.45 ± 0.74
Lung				
Absolute	1.52 ± 0.07	1.50 ± 0.07	1.44 ± 0.04	1.41 ± 0.06
Relative	9.11 ± 0.40	9.23 ± 0.37	8.45 ± 0.41	8.85 ± 0.37
Thymus				
Absolute	0.474 ± 0.036	0.432 ± 0.021	0.400 ± 0.025	0.369 ± 0.017
Relative	2.86 ± 0.23	2.65 ± 0.08	2.35 ± 0.16	2.32 ± 0.10

*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error). Statistical analysis is for linear trends performed using mixed models with continuous dose and dam ID (litter) as a random effect. Multiple pairwise comparisons of dosed groups to the sham control group were performed using mixed models with categorical dose and dam ID (litter) as a random effect with Dunnett-Hsu adjustment method.

Table G-8. Organ Weights and Organ-Weight-to-Body-Weight Ratios for Rats at the 14-week Interim Evaluation in the Two-year CDMA-modulated Cell Phone RFR Study[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Males				
Necropsy body wt.	444 ± 9	440 ± 8	435 ± 4	411 ± 8**
Brain				
Absolute	1.92 ± 0.02	1.96 ± 0.02	1.97 ± 0.04	1.92 ± 0.02
Relative	4.34 ± 0.08	4.47 ± 0.08	4.53 ± 0.10	4.67 ± 0.08*
R. Epididymis				
Absolute	0.6423 ± 0.0278	0.6298 ± 0.0256	0.6370 ± 0.0162b	0.5817 ± 0.0353
Relative	1.45 ± 0.06	1.44 ± 0.07	1.46 ± 0.04b	1.42 ± 0.09
L. Epididymis				
Absolute	0.6404 ± 0.0208	0.6673 ± 0.0137	0.6491 ± 0.0155	0.5866 ± 0.0428
Relative	1.44 ± 0.05	1.52 ± 0.05	1.49 ± 0.03	1.43 ± 0.11
Heart				
Absolute	1.52 ± 0.03	1.52 ± 0.04	1.51 ± 0.03	1.43 ± 0.04
Relative	3.42 ± 0.06	3.46 ± 0.06	3.47 ± 0.06	3.49 ± 0.07
R. Kidney				
Absolute	1.59 ± 0.04	1.51 ± 0.04	1.51 ± 0.05	1.38 ± 0.05**
Relative	3.58 ± 0.09	3.45 ± 0.09	3.47 ± 0.11	3.36 ± 0.13
L. Kidney				
Absolute	1.58 ± 0.04	1.53 ± 0.04	1.49 ± 0.04	1.35 ± 0.04**
Relative	3.57 ± 0.09	3.49 ± 0.06	3.43 ± 0.09	3.29 ± 0.12
Liver				
Absolute	16.49 ± 0.31	15.66 ± 0.39	15.57 ± 0.37	14.13 ± 0.36**
Relative	37.17 ± 0.61	35.64 ± 0.57	35.79 ± 0.64	34.35 ± 0.45**
Lung				
Absolute	2.20 ± 0.06	2.06 ± 0.07	2.29 ± 0.12	2.09 ± 0.09
Relative	4.96 ± 0.15	4.70 ± 0.18	5.26 ± 0.25	5.12 ± 0.30
R. Testis				
Absolute	2.071 ± 0.051	2.104 ± 0.045	2.087 ± 0.044	1.784 ± 0.152
Relative	4.68 ± 0.16	4.80 ± 0.14	4.81 ± 0.12	4.37 ± 0.38
L. Testis				
Absolute	2.083 ± 0.059	2.116 ± 0.055	2.102 ± 0.036	1.836 ± 0.160
Relative	4.71 ± 0.17	4.83 ± 0.14	4.84 ± 0.10	4.50 ± 0.40
Thymus				

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
Absolute	0.349 ± 0.023	0.368 ± 0.018	0.387 ± 0.026	0.364 ± 0.031
Relative	0.79 ± 0.05	0.84 ± 0.04	0.89 ± 0.06	0.88 ± 0.07
Females				
Necropsy body wt.	274 ± 8	264 ± 5	264 ± 6	252 ± 4*
Brain				
Absolute	1.79 ± 0.02	1.79 ± 0.02	1.85 ± 0.02	1.77 ± 0.02
Relative	6.58 ± 0.18	6.81 ± 0.13	7.02 ± 0.15	7.05 ± 0.09
Heart				
Absolute	1.02 ± 0.03	0.97 ± 0.01	0.98 ± 0.02	0.94 ± 0.02*
Relative	3.74 ± 0.09	3.67 ± 0.05	3.73 ± 0.07	3.73 ± 0.08
R. Kidney				
Absolute	0.97 ± 0.03	0.91 ± 0.02	0.91 ± 0.02	0.84 ± 0.02**
Relative	3.54 ± 0.08	3.45 ± 0.07	3.47 ± 0.06	3.35 ± 0.08
L. Kidney				
Absolute	0.97 ± 0.04	0.87 ± 0.02*	0.89 ± 0.02*	0.82 ± 0.02**
Relative	3.53 ± 0.11	3.28 ± 0.05	3.38 ± 0.06	3.26 ± 0.09
Liver				
Absolute	10.07 ± 0.56	8.92 ± 0.28	9.19 ± 0.28	8.76 ± 0.20*
Relative	36.55 ± 1.16	33.72 ± 0.59	34.80 ± 0.64	34.82 ± 0.76
Lung				
Absolute	1.85 ± 0.05	1.80 ± 0.07	1.75 ± 0.08	1.72 ± 0.06
Relative	6.79 ± 0.24	6.83 ± 0.26	6.68 ± 0.35	6.84 ± 0.25
R. Ovary				
Absolute	0.0598 ± 0.0042	0.0578 ± 0.0039	0.0587 ± 0.0029	0.0566 ± 0.0053
Relative	0.22 ± 0.02	0.22 ± 0.01	0.22 ± 0.01	0.22 ± 0.02
L. Ovary				
Absolute	0.0511 ± 0.0031	0.0521 ± 0.0030	0.0547 ± 0.0035	0.0495 ± 0.0055
Relative	0.19 ± 0.01	0.20 ± 0.01	0.21 ± 0.01	0.20 ± 0.02
Thymus				
Absolute	0.299 ± 0.016	0.282 ± 0.008	0.274 ± 0.015	0.278 ± 0.015
Relative	1.10 ± 0.06	1.07 ± 0.03	1.04 ± 0.05	1.11 ± 0.06

*Significantly different ($P \leq 0.05$) from the sham control group.
**$P \leq 0.01$.
[a]Organ weights (absolute weights) and body weights are given in grams; organ-weight-to-body-weight ratios (relative weights) are given as mg organ weight/g body weight (mean ± standard error). Statistical analysis performed by Jonckheere's (trend) and Williams' or Dunnett's (pairwise) tests.
[b]n = 9.

Appendix H. Reproductive Tissue Evaluations

Tables

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Table H-1. Summary of Reproductive Tissue Evaluations for Male Rats Exposed to GSM-modulated Cell Phone RFR for 14 Weeks[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Weights (g)				
Necropsy body wt	444 ± 9	426 ± 10	430 ± 7	410 ± 7*
L. Cauda epididymis	0.266 ± 0.011	0.271 ± 0.008	0.257 ± 0.009	0.269 ± 0.011
L. Epididymis	0.640 ± 0.021	0.643 ± 0.018	0.622 ± 0.017	0.643 ± 0.029
L. Testis	2.083 ± 0.059	2.028 ± 0.051	2.049 ± 0.039	1.954 ± 0.055
Spermatid measurements				
Spermatid heads (106/testis)	305.1 ± 11.2	280.3 ± 8.9	263.4 ± 12.3	297.4 ± 12.5
Spermatid heads (106/g testis)	147.3 ± 6.0	138.6 ± 4.2	128.5 ± 5.2	152.7 ± 6.8
Epididymal spermatozoal measurements				
Sperm motility (%)	91.5 ± 1.4	74.0 ± 8.0	91.5 ± 1.4	89.4 ± 2.2
Sperm (106/cauda epididymis)	247.7 ± 68.9	285.5 ± 71.3	283.7 ± 57.5	220.3 ± 34.9
Sperm (106/g cauda epididymis)	909.3 ± 243.5	1,081.1 ± 282.6	1,085.0 ± 210.7	834.8 ± 139.5

*Significantly different ($P \leq 0.05$) from the sham control group by Williams' or Dunnett's test.
[a]Data are presented as mean ± standard error. Differences from the sham control group are not significant by Williams' or Dunnett's test (tissue weights) or by Shirley's or Dunn's test (spermatid and epididymal spermatozoal measurements).

Table H-2. Summary of Reproductive Tissue Evaluations for Male Rats Exposed to CDMA-modulated Cell Phone RFR for 14 Weeks[a]

	Sham Control	1.5 W/kg	3 W/kg	6 W/kg
n	10	10	10	10
Weights (g)				
Necropsy body wt	444 ± 9	440 ± 8	435 ± 4	411 ± 8**
L. Cauda epididymis	0.266 ± 0.011	0.284 ± 0.008	0.274 ± 0.009	0.249 ± 0.017
L. Epididymis	0.640 ± 0.021	0.667 ± 0.014	0.649 ± 0.015	0.587 ± 0.043
L. Testis	2.083 ± 0.059	2.116 ± 0.055	2.102 ± 0.036	1.836 ± 0.160
Spermatid measurements				
Spermatid heads (106/testis)	305.1 ± 11.2	281.0 ± 12.5	280.7 ± 10.1	253.7 ± 30.1
Spermatid heads (106/g testis)	147.3 ± 6.0	133.9 ± 7.6	133.6 ± 4.5	129.5 ± 12.9
Epididymal spermatozoal measurements				
Sperm motility (%)	91.5 ± 1.4	90.9 ± 1.0	88.7 ± 4.0	81.9 ± 9.2
Sperm (106/cauda epididymis)	247.7 ± 68.9	206.0 ± 36.4	243.9 ± 36.4	201.8 ± 29.7
Sperm (106/g cauda epididymis)	909.3 ± 243.5	742.9 ± 140.7	906.2 ± 144.4	775.8 ± 106.6

**Significantly different ($P \leq 0.01$) from the sham control group by Williams' test.
[a]Data are presented as mean ± standard error. Differences from the sham control group are tested for significance by Williams' or Dunnett's test (tissue weights) or by Shirley's or Dunn's test (spermatid and epididymal spermatozoal measurements).

Appendix I. GSM- and CDMA-modulated Cell Phone RFR Exposure Data

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Table I-8. Summary of CDMA-modulated Cell Phone RFR Exposure Data in Rats – H-Field I-58

I.1. Overview

Exposure data include SAR (W/kg) (Table I-1 and Table I-5), chamber field strength (V/m) (Table I-2 and Table I-6), and E- and H-field measurements (V/m) (Table I-3, Table I-4, Table I-7, and Table I-8). For the medium- and high-dose GSM chambers, where a second E-field probe was used, the H-field measurements were converted from E-field measurements (E-field divided by 377). Fields were measured continuously throughout the studies and measurements automatically recorded approximately every 20 seconds. For every 20-second interval, the SAR was calculated based on the average H- and/or E-field data. The data presented for each exposure parameter include the mean and standard deviation [expressed in decibels (dB), W/kg or V/m], the total number of measurements recorded during the identified period of exposure (>50,000 calculated SAR per month, and more than 1.4 million over the course of the 2-year studies); the lowest (minimum) and highest measurement (maximum) recorded during the given exposure period; the number of measurements that were within the acceptable range; and the ratio of all measurements within range. The data reported for SAR also include the range of animal body weights (g) over the indicated time period of exposure, and the selected target SAR (W/kg) for each group. The data reported for field strengths (chamber, E-field, and H-field) include the target range of the field required to maintain appropriate SAR exposures. The minimum and maximum exposure values reported represent a single recorded measurement over the 2-year exposure period. The SAR and chamber-field in the sham and exposure chambers were within the target ranges (defined as ± 2 dB) for >99.97% of recorded measurements over the course of the 2-year study; ≥99.25% of E- and H-field exposures in the sham and exposure chambers were within the target ranges.

The dB is a mathematical transformation of a number or numerical ratio using base 10 logarithms. Multiplication of ratios is transformed into addition of dBs; raising a number to a power is transformed into multiplication of dBs.

In general, dB(power) = 10 × log(R) and dB(field) = 20 × log(R). The formulas differ by a factor of two because power or SAR varies as the square of the fields. For SAR (in W/kg), the decibel formula is calculated as:

SAR(dB) = 10 × log(SARM/SART)

where SARM is the measured value and SART is the target value, and

–2 dB = 10 × log(SARL/SART), where SARL (low) = SART × 10-0.2

+2 dB = 10 × log(SARH/SART), where SARH (high) = SART × 100.2

On this basis, the ± 2 dB range specified by the NTP translates to the following ranges for each SAR used in the 2-year study:

Target SAR (W/kg) and acceptable SAR range (W/kg; ± 2 dB)

1.5 0.95 to 2.38

3 1.89 to 4.75

6 3.79 to 9.51

I.2. Summary of GSM-modulated Exposure Data

Table I-1. Summary of GSM-modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
August 8 to 31, 2012								
Male								
Ch06 GSM High	238.7 to 355.6	6.00	5.96	0.30/0.07	2.553	11.257	42880/42893	1.000
Ch05 GSM Med	238.7 to 355.5	3.00	2.99	0.25/0.06	1.347	4.987	42890/42893	1.000
Ch09 GSM Low	239.9 to 358.9	1.50	1.49	0.23/0.05	0.654	2.544	42890/42893	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	42893/42893	1.000
September 1 to 30, 2012								
Female								
Ch17 GSM High	58.0 to 303.9	6.00	5.99	0.30/0.07	3.154	11.061	52929/52959	0.999
Ch16 GSM Med	58.7 to 303.9	3.00	3.00	0.27/0.06	1.634	5.394	52953/52959	1.000
Ch20 GSM Low	59.4 to 303.9	1.50	1.49	0.28/0.07	0.823	3.032	52938/52959	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52959/52959	1.000
Male								
Ch06 GSM High	60.9 to 290.7	6.00	5.99	0.33/0.08	1.483	25.815	52932/52959	0.999
Ch05 GSM Med	62.0 to 302.3	3.00	3.00	0.31/0.07	1.646	10.693	52926/52959	0.999
Ch09 GSM Low	63.9 to 309.8	1.50	1.50	0.30/0.07	0.737	5.732	52920/52959	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52959/52959	1.000
October 1 to 31, 2012								
Female								
Ch17 GSM High	117.8 to 202.2	6.00	5.97	0.27/0.07	4.018	9.604	55337/55339	1.000
Ch16 GSM Med	118.8 to 201.8	3.00	2.98	0.25/0.06	1.870	4.575	55338/55339	1.000
Ch20 GSM Low	120.1 to 205.4	1.50	1.49	0.25/0.06	0.947	2.556	55332/55335	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55339/55339	1.000
Male								
Ch06 GSM High	137.9 to 306.4	6.00	5.94	0.30/0.07	3.814	9.705	55349/55351	1.000
Ch05 GSM Med	139.5 to 313.7	3.00	2.98	0.26/0.06	2.110	4.395	55351/55351	1.000
Ch09 GSM Low	143.1 to 318.0	1.50	1.48	0.24/0.06	1.089	2.162	55339/55339	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55351/55351	1.000
November 1 to 30, 2012								
Female								
Ch17 GSM High	207.1 to 246.3	6.00	5.96	0.27/0.06	4.468	8.847	53853/53853	1.000
Ch16 GSM Med	208.5 to 246.7	3.00	2.98	0.23/0.05	2.407	4.141	53853/53853	1.000
Ch20 GSM Low	210.9 to 246.6	1.50	1.49	0.24/0.06	0.990	2.587	53842/53853	1.000
Ch15 Sham	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53853/53853	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Male								
Ch06 GSM High	313.9 to 389.2	6.00	5.95	0.29/0.07	4.132	8.526	53853/53853	1.000
Ch05 GSM Med	324.3 to 400.0	3.00	2.99	0.27/0.06	2.271	3.920	53853/53853	1.000
Ch09 GSM Low	329.8 to 402.5	1.50	1.49	0.22/0.05	1.178	2.057	53853/53853	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53853/53853	1.000
December 1 to 31, 2012								
Female								
Ch17 GSM High	246.3 to 259.5	6.00	5.93	0.28/0.07	3.770	8.873	55607/55608	1.000
Ch16 GSM Med	246.7 to 257.1	3.00	2.97	0.23/0.06	2.283	4.025	55608/55608	1.000
Ch20 GSM Low	246.6 to 261.6	1.50	1.49	0.20/0.05	1.164	2.069	55608/55608	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55608/55608	1.000
Male								
Ch06 GSM High	389.2 to 419.3	6.00	5.97	0.30/0.07	4.198	8.500	55608/55608	1.000
Ch05 GSM Med	400.0 to 429.8	3.00	2.99	0.28/0.07	2.109	3.863	55608/55608	1.000
Ch09 GSM Low	402.5 to 432.3	1.50	1.48	0.23/0.06	1.119	1.916	55608/55608	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55608/55608	1.000
January 1 to 31, 2013								
Female								
Ch17 GSM High	259.5 to 275.8	6.00	5.95	0.27/0.07	4.415	8.955	55618/55618	1.000
Ch16 GSM Med	257.1 to 273.1	3.00	2.98	0.23/0.06	2.271	3.975	55619/55619	1.000
Ch20 GSM Low	261.6 to 279.0	1.50	1.49	0.21/0.05	1.148	2.082	55617/55617	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55619/55619	1.000
Male								
Ch06 GSM High	419.3 to 458.6	6.00	5.97	0.32/0.08	4.278	8.641	55619/55619	1.000
Ch05 GSM Med	429.8 to 464.1	3.00	2.97	0.27/0.06	2.242	3.984	55619/55619	1.000
Ch09 GSM Low	432.3 to 469.8	1.50	1.48	0.25/0.06	1.152	2.394	55618/55619	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55619/55619	1.000
February 1 to 28, 2013								
Female								
Ch17 GSM High	275.8 to 286.0	6.00	5.96	0.26/0.06	4.471	8.310	50082/50082	1.000
Ch16 GSM Med	273.1 to 282.0	3.00	2.98	0.24/0.06	2.326	3.891	50082/50082	1.000
Ch20 GSM Low	279.0 to 287.8	1.50	1.49	0.20/0.05	1.221	1.992	50082/50082	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	50082/50082	1.000
Male								
Ch06 GSM High	458.6 to 482.5	6.00	5.98	0.33/0.08	4.282	8.327	50082/50082	1.000
Ch05 GSM Med	464.1 to 493.7	3.00	3.00	0.28/0.07	2.362	3.887	50082/50082	1.000
Ch09 GSM Low	469.8 to 496.2	1.50	1.49	0.24/0.06	1.137	1.931	50082/50082	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	50082/50082	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
March 1 to 31, 2013								
Female								
Ch17 GSM High	286.0 to 296.4	6.00	5.93	0.27/0.06	4.613	8.141	55704/55704	1.000
Ch16 GSM Med	282.0 to 292.9	3.00	2.97	0.25/0.06	2.166	3.911	55704/55704	1.000
Ch20 GSM Low	287.8 to 297.0	1.50	1.48	0.20/0.05	1.198	1.965	55704/55704	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55704/55704	1.000
Male								
Ch06 GSM High	482.5 to 503.9	6.00	5.93	0.34/0.08	4.278	8.763	55704/55704	1.000
Ch05 GSM Med	493.7 to 513.0	3.00	3.01	0.28/0.07	2.351	4.000	55704/55704	1.000
Ch09 GSM Low	496.2 to 517.8	1.50	1.49	0.24/0.06	1.150	2.068	55704/55704	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55704/55704	1.000
April 1 to 30, 2013								
Female								
Ch17 GSM High	296.4 to 305.2	6.00	5.97	0.28/0.07	4.614	8.286	53719/53719	1.000
Ch16 GSM Med	292.9 to 300.8	3.00	2.99	0.25/0.06	2.254	3.933	53719/53719	1.000
Ch20 GSM Low	297.0 to 307.9	1.50	1.49	0.20/0.05	1.215	2.087	53719/53719	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53719/53719	1.000
Male								
Ch06 GSM High	503.9 to 523.3	6.00	5.91	0.33/0.08	4.395	8.282	53721/53721	1.000
Ch05 GSM Med	513.0 to 531.8	3.00	3.00	0.29/0.07	2.310	4.013	53721/53721	1.000
Ch09 GSM Low	517.8 to 538.7	1.50	1.49	0.25/0.06	1.207	1.911	53719/53719	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53722/53722	1.000
May 1 to 31, 2013								
Female								
Ch17 GSM High	305.2 to 314.8	6.00	5.93	0.29/0.07	3.468	8.502	52758/52762	1.000
Ch16 GSM Med	300.8 to 309.8	3.00	2.98	0.26/0.06	1.676	3.984	52760/52762	1.000
Ch20 GSM Low	307.9 to 316.0	1.50	1.48	0.22/0.05	0.817	1.957	52759/52762	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52762/52762	1.000
Male								
Ch06 GSM High	523.3 to 539.7	6.00	5.96	0.35/0.08	3.356	8.858	52788/52789	1.000
Ch05 GSM Med	531.8 to 550.9	3.00	3.00	0.29/0.07	1.145	3.948	52788/52790	1.000
Ch09 GSM Low	538.7 to 557.8	1.50	1.48	0.30/0.07	0.849	2.306	52778/52788	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52790/52790	1.000
June 1 to 30, 2013								
Female								
Ch17 GSM High	314.8 to 322.9	6.00	5.98	0.30/0.07	3.712	8.162	53536/53537	1.000
Ch16 GSM Med	309.8 to 317.7	3.00	2.97	0.26/0.06	2.049	3.927	53537/53537	1.000
Ch20 GSM Low	316.0 to 324.2	1.50	1.50	0.22/0.05	1.068	1.913	53537/53537	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53537/53537	1.000
Male								
Ch06 GSM High	539.7 to 556.0	6.00	5.93	0.35/0.08	3.758	8.744	53543/53544	1.000
Ch05 GSM Med	550.9 to 566.9	3.00	2.97	0.29/0.07	2.164	4.004	53544/53544	1.000
Ch09 GSM Low	557.8 to 574.0	1.50	1.49	0.25/0.06	1.099	1.953	53537/53537	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53544/53544	1.000
July 1 to 31, 2013								
Female								
Ch17 GSM High	322.9 to 338.2	6.00	5.99	0.29/0.07	4.603	8.053	55527/55527	1.000
Ch16 GSM Med	316.3 to 334.5	3.00	3.00	0.25/0.06	2.330	3.798	55527/55527	1.000
Ch20 GSM Low	324.2 to 341.9	1.50	1.49	0.21/0.05	1.186	1.863	55527/55527	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55527/55527	1.000
Male								
Ch06 GSM High	556.0 to 585.8	6.00	5.92	0.35/0.08	4.259	8.691	55531/55531	1.000
Ch05 GSM Med	566.9 to 596.0	3.00	2.96	0.29/0.07	2.249	4.043	55531/55531	1.000
Ch09 GSM Low	574.0 to 607.9	1.50	1.48	0.27/0.06	1.119	2.044	55527/55527	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55531/55531	1.000
August 1 to 31, 2013								
Female								
Ch17 GSM High	338.2 to 344.5	6.00	5.98	0.29/0.07	4.360	7.931	55952/55952	1.000
Ch16 GSM Med	334.5 to 343.0	3.00	2.99	0.26/0.06	2.294	4.001	55952/55952	1.000
Ch20 GSM Low	341.9 to 348.4	1.50	1.49	0.20/0.05	1.195	1.858	55952/55952	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55952/55952	1.000
Male								
Ch06 GSM High	585.8 to 597.1	6.00	5.89	0.34/0.08	3.819	8.404	55955/55955	1.000
Ch05 GSM Med	596.0 to 606.8	3.00	2.98	0.28/0.07	2.303	3.856	55955/55955	1.000
Ch09 GSM Low	607.9 to 619.4	1.50	1.48	0.25/0.06	1.167	1.959	55952/55952	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55955/55955	1.000
September 1 to 30, 2013								
Female								
Ch17 GSM High	344.5 to 349.8	6.00	5.95	0.28/0.07	3.800	8.065	53696/53696	1.000
Ch16 GSM Med	343.0 to 352.4	3.00	2.98	0.28/0.07	1.822	4.020	53694/53696	1.000
Ch20 GSM Low	348.4 to 355.8	1.50	1.49	0.22/0.05	1.073	1.968	53696/53696	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53696/53696	1.000
Male								
Ch06 GSM High	597.1 to 607.0	6.00	5.94	0.34/0.08	4.168	8.242	53703/53703	1.000
Ch05 GSM Med	606.8 to 617.7	3.00	2.97	0.30/0.07	1.936	4.044	53703/53703	1.000
Ch09 GSM Low	619.4 to 633.9	1.50	1.49	0.25/0.06	1.029	1.959	53696/53696	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53703/53703	1.000
October 1 to 31, 2013								
Female								
Ch17 GSM High	349.8 to 360.2	6.00	5.96	0.36/0.09	3.397	9.676	56717/56748	0.999
Ch16 GSM Med	352.4 to 361.2	3.00	2.97	0.36/0.09	1.520	4.314	56693/56748	0.999
Ch20 GSM Low	355.8 to 366.5	1.50	1.48	0.26/0.06	0.926	1.910	56745/56747	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	56748/56748	1.000
Male								
Ch06 GSM High	607.0 to 615.0	6.00	5.93	0.38/0.09	3.232	8.512	56799/56807	1.000
Ch05 GSM Med	617.7 to 628.2	3.00	3.00	0.34/0.08	2.071	4.172	56807/56807	1.000
Ch09 GSM Low	633.9 to 642.8	1.50	1.49	0.27/0.06	1.027	2.186	56748/56748	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	56807/56807	1.000
November 1 to 30, 2013								
Female								
Ch17 GSM High	360.2 to 368.2	6.00	5.98	0.29/0.07	3.849	8.198	55323/55323	1.000
Ch16 GSM Med	361.2 to 371.8	3.00	3.00	0.28/0.07	1.907	3.925	55323/55323	1.000
Ch20 GSM Low	366.5 to 375.8	1.50	1.50	0.22/0.05	1.085	2.056	55323/55323	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55323/55323	1.000
Male								
Ch06 GSM High	615.0 to 625.1	6.00	5.94	0.33/0.08	4.105	8.973	55332/55332	1.000
Ch05 GSM Med	628.2 to 635.6	3.00	2.99	0.30/0.07	1.778	3.941	55331/55332	1.000
Ch09 GSM Low	642.8 to 648.2	1.50	1.48	0.24/0.06	1.105	2.003	55329/55329	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55332/55332	1.000
December 1 to 31, 2013								
Female								
Ch17 GSM High	368.2 to 376.2	6.00	6.00	0.30/0.07	4.541	8.484	53099/53099	1.000
Ch16 GSM Med	371.8 to 380.7	3.00	2.99	0.27/0.06	2.251	3.819	53099/53099	1.000
Ch20 GSM Low	375.8 to 383.8	1.50	1.49	0.21/0.05	1.251	1.897	53057/53057	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53099/53099	1.000
Male								
Ch06 GSM High	625.1 to 631.2	6.00	5.93	0.33/0.08	4.449	7.962	53111/53111	1.000
Ch05 GSM Med	635.6 to 641.1	3.00	2.98	0.32/0.08	2.021	3.936	53111/53111	1.000
Ch09 GSM Low	648.2 to 653.2	1.50	1.49	0.22/0.05	1.207	1.908	53099/53099	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53111/53111	1.000
January 1 to 31, 2014								
Female								
Ch17 GSM High	376.2 to 390.8	6.00	5.98	0.30/0.07	4.226	8.481	55626/55626	1.000
Ch16 GSM Med	380.7 to 392.7	3.00	2.97	0.26/0.06	1.924	3.798	55626/55626	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch20 GSM Low	383.8 to 394.6	1.50	1.48	0.21/0.05	1.081	2.172	55621/55621	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55626/55626	1.000
Male								
Ch06 GSM High	631.2 to 641.5	6.00	5.90	0.33/0.08	4.292	8.752	55629/55629	1.000
Ch05 GSM Med	641.1 to 651.7	3.00	2.99	0.33/0.08	2.235	4.217	55629/55629	1.000
Ch09 GSM Low	653.2 to 662.0	1.50	1.49	0.23/0.05	1.181	1.899	55627/55627	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55629/55629	1.000
February 1 to 28, 2014								
Female								
Ch17 GSM High	390.8 to 401.7	6.00	5.96	0.30/0.07	4.639	8.584	51974/51974	1.000
Ch16 GSM Med	392.7 to 399.4	3.00	2.98	0.26/0.06	2.235	3.989	51974/51974	1.000
Ch20 GSM Low	394.6 to 410.5	1.50	1.50	0.20/0.05	1.226	1.894	51974/51974	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	51974/51974	1.000
Male								
Ch06 GSM High	641.5 to 650.2	6.00	5.92	0.33/0.08	4.376	8.314	51980/51980	1.000
Ch05 GSM Med	651.7 to 668.4	3.00	2.97	0.31/0.07	2.183	4.076	51980/51980	1.000
Ch09 GSM Low	662.0 to 676.6	1.50	1.49	0.23/0.05	1.192	1.888	51974/51974	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	51980/51980	1.000
March 1 to 31, 2014								
Female								
Ch17 GSM High	401.7 to 410.4	6.00	6.01	0.30/0.07	3.728	8.431	55703/55704	1.000
Ch16 GSM Med	399.4 to 408.6	3.00	2.97	0.28/0.07	1.933	3.849	55704/55704	1.000
Ch20 GSM Low	410.5 to 420.7	1.50	1.49	0.21/0.05	0.992	2.005	55704/55704	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55704/55704	1.000
Male								
Ch06 GSM High	650.2 to 658.2	6.00	5.92	0.34/0.08	4.216	9.271	55704/55704	1.000
Ch05 GSM Med	668.4 to 679.5	3.00	2.95	0.33/0.08	2.187	4.010	55704/55704	1.000
Ch09 GSM Low	676.6 to 688.8	1.50	1.50	0.23/0.05	1.190	1.982	55704/55704	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55704/55704	1.000
April 1 to 30, 2014								
Female								
Ch17 GSM High	410.4 to 422.9	6.00	5.98	0.34/0.08	3.545	8.198	53640/53644	1.000
Ch16 GSM Med	408.6 to 418.1	3.00	2.99	0.30/0.07	1.875	3.870	53643/53644	1.000
Ch20 GSM Low	420.7 to 431.1	1.50	1.48	0.21/0.05	1.096	1.840	53643/53643	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53644/53644	1.000
Male								
Ch06 GSM High	658.2 to 662.2	6.00	5.83	0.34/0.08	0.734	8.341	53646/53649	1.000
Ch05 GSM Med	676.3 to 679.5	3.00	2.97	0.32/0.08	2.265	4.191	53649/53649	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch09 GSM Low	687.5 to 688.8	1.50	1.51	0.24/0.06	1.181	1.930	53645/53645	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53649/53649	1.000
May 1 to 31, 2014								
Female								
Ch17 GSM High	422.9 to 435.5	6.00	5.96	0.32/0.08	3.891	8.517	55604/55604	1.000
Ch16 GSM Med	418.1 to 429.3	3.00	2.99	0.27/0.06	2.189	4.205	55604/55604	1.000
Ch20 GSM Low	431.1 to 437.8	1.50	1.48	0.21/0.05	1.086	1.993	55602/55602	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55604/55604	1.000
Male								
Ch06 GSM High	662.2 to 667.4	6.00	5.88	0.32/0.08	4.364	8.818	55604/55604	1.000
Ch05 GSM Med	676.3 to 679.2	3.00	2.83	0.32/0.08	2.015	4.391	55604/55604	1.000
Ch09 GSM Low	687.5 to 691.2	1.50	1.50	0.25/0.06	1.195	1.976	55604/55604	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55604/55604	1.000
June 1 to 30, 2014								
Female								
Ch17 GSM High	435.5 to 453.7	6.00	5.98	0.32/0.08	4.053	8.457	53771/53771	1.000
Ch16 GSM Med	429.3 to 440.7	3.00	2.98	0.28/0.07	1.449	3.889	53767/53771	1.000
Ch20 GSM Low	437.8 to 452.4	1.50	1.49	0.22/0.05	0.702	2.034	53763/53767	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53771/53771	1.000
Male								
Ch06 GSM High	667.3 to 670.7	6.00	5.91	0.33/0.08	3.215	9.767	53769/53773	1.000
Ch05 GSM Med	670.5 to 679.2	3.00	2.89	0.35/0.08	0.391	4.215	53750/53774	1.000
Ch09 GSM Low	672.8 to 691.2	1.50	1.51	0.25/0.06	0.292	1.988	53767/53773	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53774/53774	1.000
July 1 to 31, 2014								
Female								
Ch17 GSM High	458.1 to 465.3	6.00	5.96	0.32/0.08	3.511	8.111	55599/55601	1.000
Ch16 GSM Med	443.4 to 447.0	3.00	2.98	0.28/0.07	1.763	4.162	55600/55601	1.000
Ch20 GSM Low	451.7 to 467.3	1.50	1.49	0.21/0.05	0.990	1.973	55601/55601	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55601/55601	1.000
Male								
Ch06 GSM High	667.3 to 670.3	6.00	5.87	0.32/0.08	3.459	8.639	55602/55603	1.000
Ch05 GSM Med	659.4 to 670.5	3.00	2.96	0.32/0.08	1.787	4.205	55602/55603	1.000
Ch09 GSM Low	659.5 to 672.8	1.50	1.50	0.24/0.06	0.990	1.960	55601/55601	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55603/55603	1.000
August 1 to 31, 2014								
Female								
Ch17 GSM High	464.7 to 473.8	6.00	5.92	0.30/0.07	3.387	8.139	54345/54354	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch16 GSM Med	447.0 to 453.0	3.00	2.98	0.27/0.07	1.868	4.001	54353/54354	1.000
Ch20 GSM Low	461.2 to 475.4	1.50	1.49	0.21/0.05	1.112	1.875	54354/54354	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	54354/54354	1.000
Male								
Ch06 GSM High	655.0 to 670.3	6.00	5.90	0.32/0.08	4.391	8.661	54358/54358	1.000
Ch05 GSM Med	638.8 to 659.4	3.00	2.98	0.30/0.07	2.247	4.095	54358/54358	1.000
Ch09 GSM Low	653.6 to 660.1	1.50	1.49	0.22/0.05	1.171	1.819	54354/54354	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	54358/54358	1.000
September 1 to 30, 2014								
Female								
Ch17 GSM High	471.2 to 475.1	6.00	5.94	0.30/0.07	2.537	11.960	52084/52088	1.000
Ch16 GSM Med	453.0 to 471.6	3.00	2.98	0.26/0.06	1.802	5.349	52085/52088	1.000
Ch20 GSM Low	461.2 to 476.1	1.50	1.48	0.20/0.05	1.070	2.533	52086/52088	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52088/52088	1.000
Male								
Ch06 GSM High	650.2 to 655.0	6.00	5.95	0.30/0.07	2.313	11.724	52074/52088	1.000
Ch05 GSM Med	636.4 to 638.8	3.00	2.98	0.25/0.06	2.239	4.851	52087/52088	1.000
Ch09 GSM Low	644.0 to 653.6	1.50	1.48	0.22/0.05	0.916	2.468	52085/52088	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52088/52088	1.000
August 8, 2012 to September 30, 2014								
Female								
Ch17 GSM High	58.0 to 475.1	6.00	5.97	0.30/0.07	2.537	11.960	1400145/1400254	1.000
Ch16 GSM Med	58.7 to 471.6	3.00	2.98	0.27/0.06	1.449	5.394	1400178/1400255	1.000
Ch20 GSM Low	59.4 to 476.1	1.50	1.49	0.22/0.05	0.702	3.032	1400140/1400194	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	1400255/1400255	1.000
Male								
Ch06 GSM High	60.9 to 670.7	6.00	5.93	0.33/0.08	0.734	25.815	1400345/1400419	1.000
Ch05 GSM Med	62.0 to 679.5	3.00	2.97	0.30/0.07	0.391	10.693	1400356/1400421	1.000
Ch09 GSM Low	63.9 to 691.2	1.50	1.49	0.25/0.06	0.292	5.732	1400229/1400291	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	1400422/1400422	1.000

Ch = chamber (e.g., Ch17 = Chamber 17).

Table I-2. Summary of GSM-modulated Cell Phone RFR Exposure Data in Rats – Chamber Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
August 8 to 31, 2012							
Male							
Ch06 GSM High	246.10 to 283.80	261.74	0.30/9.31	163.93	357.66	85760/85786	1.000
Ch05 GSM Med	174.00 to 200.70	185.30	0.25/5.39	119.06	238.06	85780/85786	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch09 GSM Low	123.10 to 141.90	131.09	0.23/3.52	82.96	172.00	85780/85786	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	85786/85786	1.000
September 1 -30, 2012							
Female							
Ch17 GSM High	192.60 to 269.00	237.79	0.30/8.24	153.47	362.88	105858/105918	0.999
Ch16 GSM Med	136.20 to 190.20	167.23	0.26/5.13	105.81	241.99	105906/105918	1.000
Ch20 GSM Low	096.10 to 134.50	118.05	0.27/3.74	75.08	175.15	105876/105918	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105918/105918	1.000
Male							
Ch06 GSM High	192.60 to 266.50	232.35	0.32/8.68	106.40	443.91	105868/105918	1.000
Ch05 GSM Med	136.20 to 190.20	164.81	0.30/5.71	103.62	285.70	105858/105918	0.999
Ch09 GSM Low	096.30 to 134.50	116.84	0.29/3.90	75.03	209.17	105848/105918	0.999
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105918/105918	1.000
October 1 to 31, 2012							
Female							
Ch17 GSM High	193.80 to 232.80	214.26	0.28/6.95	164.06	288.93	110674/110678	1.000
Ch16 GSM Med	137.00 to 164.60	151.43	0.25/4.49	112.41	203.02	110676/110678	1.000
Ch20 GSM Low	098.50 to 116.40	108.14	0.25/3.20	79.98	143.57	110662/110670	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110678/110678	1.000
Male							
Ch06 GSM High	201.30 to 269.00	238.81	0.30/8.48	160.53	326.54	110698/110702	1.000
Ch05 GSM Med	142.40 to 192.20	170.55	0.26/5.18	127.50	224.85	110702/110702	1.000
Ch09 GSM Low	103.10 to 135.90	121.57	0.24/3.47	89.23	153.28	110678/110678	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110702/110702	1.000
November 1 to 30, 2012							
Female							
Ch17 GSM High	232.80 to 250.70	242.22	0.27/7.66	200.64	298.50	107706/107706	1.000
Ch16 GSM Med	164.60 to 177.30	171.90	0.24/4.71	147.26	201.43	107706/107706	1.000
Ch20 GSM Low	118.50 to 125.40	122.10	0.24/3.47	99.98	161.64	107684/107706	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107706/107706	1.000
Male							
Ch06 GSM High	271.80 to 292.20	282.73	0.30/9.97	238.55	347.86	107706/107706	1.000
Ch05 GSM Med	194.20 to 208.20	201.89	0.27/6.40	176.39	237.97	107706/107706	1.000
Ch09 GSM Low	137.40 to 148.20	143.02	0.22/3.68	123.60	164.44	107706/107706	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107706/107706	1.000
December 1 to 31, 2012							
Female							
Ch17 GSM High	250.70 to 254.90	253.13	0.28/8.43	202.44	310.55	111214/111216	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch16 GSM Med	177.30 to 180.20	179.20	0.24/4.96	155.02	209.16	111216/111216	1.000
Ch20 GSM Low	125.40 to 129.20	127.62	0.21/3.08	112.48	149.98	111216/111216	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111216/111216	1.000
Male							
Ch06 GSM High	292.20 to 298.00	295.52	0.31/10.70	248.48	353.56	111216/111216	1.000
Ch05 GSM Med	208.20 to 211.70	210.50	0.28/6.92	177.40	240.12	111216/111216	1.000
Ch09 GSM Low	148.20 to 150.20	148.91	0.24/4.13	129.81	170.40	111216/111216	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111216/111216	1.000
January 1 to 31, 2013							
Female							
Ch17 GSM High	254.90 to 261.40	257.58	0.28/8.39	220.72	312.88	111236/111236	1.000
Ch16 GSM Med	180.20 to 184.80	182.39	0.24/5.03	157.70	211.28	111238/111238	1.000
Ch20 GSM Low	129.20 to 130.70	129.57	0.21/3.21	114.20	153.83	111234/111234	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111238/111238	1.000
Male							
Ch06 GSM High	298.00 to 302.10	298.69	0.33/11.42	250.82	361.83	111238/111238	1.000
Ch05 GSM Med	211.70 to 214.00	212.38	0.28/6.89	182.92	247.58	111238/111238	1.000
Ch09 GSM Low	150.20 to 151.40	150.50	0.26/4.49	132.92	190.45	111236/111238	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111238/111238	1.000
February 1 to 28, 2013							
Female							
Ch17 GSM High	261.40 to 264.00	261.98	0.27/8.15	228.00	307.30	100164/100164	1.000
Ch16 GSM Med	184.80 to 186.70	185.40	0.25/5.38	164.18	212.70	100164/100164	1.000
Ch20 GSM Low	130.70 to 132.00	131.16	0.20/3.13	117.78	150.45	100164/100164	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	100164/100164	1.000
Male							
Ch06 GSM High	302.10 to 304.00	302.15	0.33/11.82	256.65	357.92	100164/100164	1.000
Ch05 GSM Med	214.00 to 215.50	214.86	0.28/7.04	190.61	244.53	100164/100164	1.000
Ch09 GSM Low	151.40 to 152.40	151.39	0.24/4.22	132.24	172.35	100164/100164	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	100164/100164	1.000
March 1 to 31, 2013							
Female							
Ch17 GSM High	264.00 to 266.50	264.63	0.27/8.42	231.60	307.67	111408/111408	1.000
Ch16 GSM Med	186.70 to 188.40	187.36	0.25/5.58	160.59	215.77	111408/111408	1.000
Ch20 GSM Low	132.00 to 133.20	132.42	0.21/3.18	119.43	152.94	111408/111408	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
Male							
Ch06 GSM High	304.00 to 305.60	303.27	0.35/12.40	258.25	370.03	111408/111408	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch05 GSM Med	215.50 to 216.80	216.06	0.28/7.06	190.20	250.00	111408/111408	1.000
Ch09 GSM Low	152.40 to 153.30	152.35	0.24/4.31	133.03	178.38	111408/111408	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
April 1 to 30, 2013							
Female							
Ch17 GSM High	266.50 to 269.00	267.57	0.28/8.86	235.78	314.88	107438/107438	1.000
Ch16 GSM Med	188.40 to 190.20	189.34	0.26/5.68	164.80	217.69	107438/107438	1.000
Ch20 GSM Low	133.20 to 134.50	133.66	0.21/3.22	120.28	157.64	107438/107438	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107438/107438	1.000
Male							
Ch06 GSM High	305.60 to 307.60	305.35	0.34/12.25	263.80	362.58	107442/107442	1.000
Ch05 GSM Med	216.80 to 218.30	217.81	0.29/7.44	191.48	252.37	107442/107442	1.000
Ch09 GSM Low	153.30 to 154.40	153.44	0.25/4.47	137.33	173.95	107438/107438	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107444/107444	1.000
May 1 to 31, 2013							
Female							
Ch17 GSM High	269.00 to 271.80	269.81	0.30/9.50	206.91	323.99	105516/105524	1.000
Ch16 GSM Med	190.20 to 190.20	189.49	0.27/5.91	142.11	219.08	105520/105524	1.000
Ch20 GSM Low	134.50 to 135.90	135.04	0.23/3.57	100.46	155.42	105518/105524	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105524/105524	1.000
Male							
Ch06 GSM High	307.60 to 308.80	307.28	0.36/12.95	230.79	374.97	105576/105578	1.000
Ch05 GSM Med	218.30 to 220.10	219.52	0.29/7.56	135.92	251.57	105576/105580	1.000
Ch09 GSM Low	154.40 to 155.60	154.43	0.31/5.53	117.02	191.30	105556/105576	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105580/105580	1.000
June 1 to 30, 2013							
Female							
Ch17 GSM High	271.80 to 274.70	273.16	0.31/9.87	215.41	319.42	107072/107074	1.000
Ch16 GSM Med	190.20 to 192.20	191.37	0.27/6.01	159.04	220.20	107074/107074	1.000
Ch20 GSM Low	135.90 to 137.40	136.69	0.22/3.56	115.55	154.63	107074/107074	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107074/107074	1.000
Male							
Ch06 GSM High	308.80 to 311.30	308.73	0.36/12.96	246.21	375.54	107086/107088	1.000
Ch05 GSM Med	220.10 to 221.00	220.30	0.29/7.53	188.37	256.20	107088/107088	1.000
Ch09 GSM Low	155.60 to 157.00	156.15	0.26/4.68	134.23	178.92	107074/107074	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107088/107088	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
July 1 to 31, 2013							
Female							
Ch17 GSM High	274.70 to 277.70	273.91	0.29/9.29	239.88	317.27	111054/111054	1.000
Ch16 GSM Med	192.20 to 196.40	193.72	0.25/5.73	172.83	217.88	111054/111054	1.000
Ch20 GSM Low	137.40 to 140.40	138.40	0.21/3.44	123.31	156.56	111054/111054	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111054/111054	1.000
Male							
Ch06 GSM High	311.30 to 315.30	311.45	0.36/13.11	264.23	378.17	111062/111062	1.000
Ch05 GSM Med	221.00 to 223.90	222.10	0.30/7.70	193.61	259.58	111062/111062	1.000
Ch09 GSM Low	157.00 to 159.00	157.04	0.27/5.00	136.56	186.11	111054/111054	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111062/111062	1.000
August 1 to 31, 2013							
Female							
Ch17 GSM High	277.70 to 280.80	277.49	0.29/9.56	236.42	318.87	111904/111904	1.000
Ch16 GSM Med	196.40 to 198.50	196.28	0.26/5.94	171.49	226.49	111904/111904	1.000
Ch20 GSM Low	140.40 to 140.40	139.96	0.21/3.34	125.38	156.35	111904/111904	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111904/111904	1.000
Male							
Ch06 GSM High	315.30 to 316.70	312.96	0.35/12.91	252.28	374.25	111910/111910	1.000
Ch05 GSM Med	223.90 to 224.90	222.92	0.29/7.53	197.54	253.53	111910/111910	1.000
Ch09 GSM Low	159.00 to 159.70	158.28	0.25/4.71	140.65	182.22	111904/111904	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111910/111910	1.000
September 1 to 30, 2013							
Female							
Ch17 GSM High	280.80 to 280.80	279.69	0.29/9.35	223.60	325.76	107392/107392	1.000
Ch16 GSM Med	198.50 to 200.70	198.49	0.29/6.69	154.85	229.99	107388/107392	1.000
Ch20 GSM Low	140.40 to 141.90	140.30	0.22/3.60	118.81	160.92	107392/107392	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107392/107392	1.000
Male							
Ch06 GSM High	316.70 to 318.10	314.99	0.35/12.78	265.80	370.62	107406/107406	1.000
Ch05 GSM Med	224.90 to 225.90	224.37	0.30/8.01	181.13	261.80	107406/107406	1.000
Ch09 GSM Low	159.70 to 161.00	159.38	0.26/4.76	132.09	182.21	107392/107392	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107406/107406	1.000
October 1 to 31, 2013							
Female							
Ch17 GSM High	280.80 to 286.80	281.83	0.37/12.27	215.71	356.81	113434/113496	0.999
Ch16 GSM Med	200.70 to 202.80	200.67	0.37/8.80	144.28	241.44	113388/113496	0.999
Ch20 GSM Low	141.90 to 143.40	141.96	0.27/4.41	112.11	161.75	113490/113494	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	113496/113496	1.000
Male							
Ch06 GSM High	318.10 to 319.40	316.70	0.40/14.75	20.95	379.83	113600/113614	1.000
Ch05 GSM Med	225.90 to 226.80	226.15	0.34/9.08	188.65	268.19	113614/113614	1.000
Ch09 GSM Low	161.00 to 161.70	160.60	0.27/5.14	134.24	194.14	113496/113496	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	113614/113614	1.000
November 1 to 30, 2013							
Female							
Ch17 GSM High	286.80 to 286.80	286.14	0.29/9.81	229.63	335.12	110646/110646	1.000
Ch16 GSM Med	202.80 to 204.80	203.21	0.28/6.65	161.63	233.48	110646/110646	1.000
Ch20 GSM Low	143.40 to 144.80	143.51	0.22/3.73	122.76	168.98	110646/110646	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110646/110646	1.000
Male							
Ch06 GSM High	319.40 to 320.80	318.24	0.33/12.45	263.77	389.98	110664/110664	1.000
Ch05 GSM Med	226.80 to 227.70	226.96	0.30/8.01	175.08	260.66	110662/110664	1.000
Ch09 GSM Low	161.70 to 161.70	161.02	0.25/4.62	139.23	187.46	110658/110658	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110664/110664	1.000
December 1 to 31, 2013							
Female							
Ch17 GSM High	286.80 to 289.60	287.63	0.30/10.26	251.13	340.91	106198/106198	1.000
Ch16 GSM Med	204.80 to 206.70	205.25	0.27/6.51	176.82	231.75	106198/106198	1.000
Ch20 GSM Low	144.80 to 146.10	144.94	0.21/3.57	132.18	162.32	106114/106114	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	106198/106198	1.000
Male							
Ch06 GSM High	320.80 to 322.10	319.56	0.33/12.50	276.96	370.51	106222/106222	1.000
Ch05 GSM Med	227.70 to 228.70	227.45	0.32/8.64	186.67	262.62	106222/106222	1.000
Ch09 GSM Low	161.70 to 162.40	161.63	0.22/4.24	145.51	182.95	106198/106198	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	106222/106222	1.000
January 1 to 31, 2014							
Female							
Ch17 GSM High	289.60 to 294.50	291.48	0.31/10.54	247.48	345.55	111252/111252	1.000
Ch16 GSM Med	206.70 to 208.20	206.91	0.27/6.43	166.99	234.62	111252/111252	1.000
Ch20 GSM Low	146.10 to 147.30	146.17	0.22/3.73	125.15	177.44	111242/111242	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111252/111252	1.000
Male							
Ch06 GSM High	322.10 to 323.40	320.41	0.34/12.88	272.03	391.85	111258/111258	1.000
Ch05 GSM Med	228.70 to 229.60	228.70	0.33/8.88	198.03	272.01	111258/111258	1.000
Ch09 GSM Low	162.40 to 163.00	162.49	0.23/4.34	143.93	184.14	111254/111254	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111258/111258	1.000
February 1 to 28, 2014							
Female							
Ch17 GSM High	294.50 to 296.40	295.29	0.30/10.52	260.64	355.30	103948/103948	1.000
Ch16 GSM Med	208.20 to 208.20	207.59	0.27/6.51	179.98	240.45	103948/103948	1.000
Ch20 GSM Low	147.30 to 149.00	148.04	0.20/3.53	133.39	166.89	103948/103948	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	103948/103948	1.000
Male							
Ch06 GSM High	323.40 to 324.80	322.09	0.33/12.65	277.08	381.92	103960/103960	1.000
Ch05 GSM Med	229.60 to 230.60	229.42	0.32/8.48	197.45	269.79	103960/103960	1.000
Ch09 GSM Low	163.00 to 163.70	163.19	0.23/4.39	145.88	183.61	103948/103948	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	103960/103960	1.000
March 1 to 31, 2014							
Female							
Ch17 GSM High	296.40 to 298.00	297.00	0.31/10.72	234.16	352.11	111404/111408	1.000
Ch16 GSM Med	208.20 to 209.60	208.52	0.28/6.90	168.58	237.91	111408/111408	1.000
Ch20 GSM Low	149.00 to 149.70	148.83	0.21/3.69	121.71	172.98	111408/111408	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
Male							
Ch06 GSM High	324.80 to 324.80	321.89	0.35/13.20	271.97	403.29	111408/111408	1.000
Ch05 GSM Med	230.60 to 231.50	229.35	0.33/8.95	197.64	267.59	111408/111408	1.000
Ch09 GSM Low	163.70 to 164.40	163.82	0.23/4.36	145.79	188.13	111408/111408	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
April 1 to 30, 2014							
Female							
Ch17 GSM High	298.00 to 299.30	298.09	0.34/12.08	230.03	349.80	107280/107288	1.000
Ch16 GSM Med	209.60 to 210.70	209.72	0.31/7.56	166.05	238.56	107286/107288	1.000
Ch20 GSM Low	149.70 to 150.20	149.58	0.22/3.77	128.89	166.30	107286/107286	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107288/107288	1.000
Male							
Ch06 GSM High	324.80 to 326.10	321.85	0.35/13.36	114.48	385.94	107292/107298	1.000
Ch05 GSM Med	231.50 to 231.50	229.97	0.33/8.93	201.12	273.57	107298/107298	1.000
Ch09 GSM Low	164.40 to 164.40	164.03	0.24/4.54	145.21	185.62	107290/107290	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107298/107298	1.000
May 1 to 31, 2014							
Female							
Ch17 GSM High	299.30 to 300.40	300.03	0.33/11.60	242.81	359.23	111208/111208	1.000
Ch16 GSM Med	210.70 to 211.70	211.00	0.27/6.71	180.74	250.53	111208/111208	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch20 GSM Low	150.20 to 150.20	149.77	0.22/3.81	128.27	173.78	111204/111204	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111208/111208	1.000
Male							
Ch06 GSM High	326.10 to 326.10	323.75	0.32/12.29	279.16	396.81	111208/111208	1.000
Ch05 GSM Med	231.50 to 231.50	224.46	0.33/8.72	189.71	280.03	111208/111208	1.000
Ch09 GSM Low	164.40 to 165.10	164.89	0.25/4.91	147.41	189.52	111208/111208	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111208/111208	1.000
June 1 to 30, 2014							
Female							
Ch17 GSM High	300.40 to 302.10	300.88	0.33/11.52	247.80	357.96	107542/107542	1.000
Ch16 GSM Med	211.70 to 213.10	212.09	0.29/7.21	147.07	242.76	107534/107542	1.000
Ch20 GSM Low	150.20 to 151.00	150.43	0.22/3.91	103.10	175.53	107526/107534	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107542/107542	1.000
Male							
Ch06 GSM High	326.10 to 327.40	324.69	0.34/12.95	239.60	417.63	107538/107546	1.000
Ch05 GSM Med	231.50 to 231.50	226.88	0.37/9.99	83.52	274.36	107500/107548	1.000
Ch09 GSM Low	163.70 to 165.10	164.18	0.26/4.92	72.17	190.10	107534/107546	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107548/107548	1.000
July 1 to 31, 2014							
Female							
Ch17 GSM High	302.10 to 302.70	301.45	0.33/11.55	230.64	352.78	111198/111202	1.000
Ch16 GSM Med	213.10 to 213.10	212.21	0.28/7.05	163.42	251.13	111200/111202	1.000
Ch20 GSM Low	151.00 to 151.40	150.88	0.21/3.77	122.47	172.90	111202/111202	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111202/111202	1.000
Male							
Ch06 GSM High	326.10 to 327.40	323.48	0.33/12.46	248.53	392.76	111204/111206	1.000
Ch05 GSM Med	229.60 to 231.50	229.53	0.33/8.75	178.63	274.03	111204/111206	1.000
Ch09 GSM Low	162.40 to 163.70	163.30	0.24/4.55	132.94	187.07	111202/111202	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111206/111206	1.000
August 1 to 31, 2014							
Female							
Ch17 GSM High	302.70 to 303.40	301.67	0.31/10.80	228.28	353.86	108690/108708	1.000
Ch16 GSM Med	213.10 to 213.60	212.27	0.28/6.99	168.24	246.20	108706/108708	1.000
Ch20 GSM Low	151.40 to 151.70	151.14	0.21/3.78	130.78	169.86	108708/108708	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	108708/108708	1.000
Male							
Ch06 GSM High	324.80 to 327.40	323.73	0.33/12.36	280.03	389.80	108716/108716	1.000
Ch05 GSM Med	227.70 to 229.60	228.01	0.31/8.28	198.54	268.03	108716/108716	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch09 GSM Low	162.40 to 163.00	162.22	0.22/4.20	144.61	179.04	108708/108708	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	108716/108716	1.000
September 1 to 30, 2014							
Female							
Ch17 GSM High	303.40 to 303.40	302.18	0.30/10.67	197.54	428.95	104168/104176	1.000
Ch16 GSM Med	213.60 to 214.50	212.76	0.26/6.52	166.50	286.87	104170/104176	1.000
Ch20 GSM Low	151.40 to 151.70	151.10	0.21/3.63	128.30	197.41	104172/104176	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	104176/104176	1.000
Male							
Ch06 GSM High	324.80 to 324.80	322.76	0.31/11.68	201.44	453.53	104148/104176	1.000
Ch05 GSM Med	227.70 to 227.70	226.73	0.25/6.74	196.47	289.19	104174/104176	1.000
Ch09 GSM Low	161.70 to 162.40	161.10	0.22/4.12	126.78	208.08	104170/104176	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	104176/104176	1.000
August 8, 2012 to September 30, 2014							
Female							
Ch17 GSM High	192.60 to 303.40	276.19	0.30/9.74	153.47	428.95	2800286/2800508	1.000
Ch16 GSM Med	136.20 to 214.50	195.10	0.27/6.17	105.81	286.87	2800358/2800510	1.000
Ch20 GSM Low	096.10 to 151.70	138.52	0.22/3.59	75.08	197.41	2800278/2800388	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	2800510/2800510	1.000
Male							
Ch06 GSM High	192.60 to 327.40	305.54	0.34/12.09	20.95	453.53	2800696/2800838	1.000
Ch05 GSM Med	136.20 to 231.50	217.06	0.31/7.84	83.52	289.19	2800718/2800842	1.000
Ch09 GSM Low	096.30 to 165.10	154.30	0.25/4.47	72.17	209.17	2800466/2800582	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	2800844/2800844	1.000

Ch = chamber (e.g., Ch17 = Chamber 17).

Table I-3. Summary of GSM-modulated Cell Phone RFR Exposure Data in Rats – E-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
August 8 to 31, 2012							
Male							
Ch06 GSM High	246.1 to 283.8	246.42	0.40/11.69	154.4	330.2	85692/85786	0.999
Ch05 GSM Med	174.0 to 200.7	192.97	0.37/8.36	124.3	252.8	85768/85786	1.000
Ch09 GSM Low	123.1 to 141.9	119.12	0.32/4.49	74.9	162.8	85742/85786	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	85786/85786	1.000
September 1 to 30, 2012							
Female							
Ch17 GSM High	192.6 to 269.0	231.49	0.54/14.83	141.4	395.5	105788/105918	0.999
Ch16 GSM Med	136.2 to 190.2	161.13	0.37/7.09	101.6	244.1	105894/105918	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch20 GSM Low	96.1 to 134.5	112.92	0.39/5.13	71.8	170.4	105878/105918	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105918/105918	1.000
Male							
Ch06 GSM High	192.6 to 266.5	221.52	0.45/11.71	104.2	445.8	105824/105918	0.999
Ch05 GSM Med	136.2 to 190.2	174.00	0.45/9.23	110.2	323.0	105652/105918	0.997
Ch09 GSM Low	96.3 to 134.5	109.88	0.38/4.91	68.9	195.7	105852/105918	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105918/105918	1.000
October 1 to 31, 2012							
Female							
Ch17 GSM High	193.8 to 232.8	193.51	0.44/10.00	137.3	285.2	109918/110678	0.993
Ch16 GSM Med	137.0 to 164.6	138.97	0.34/5.51	108.8	184.5	110640/110678	1.000
Ch20 GSM Low	98.5 to 116.4	106.63	0.36/4.47	75.3	141.4	110658/110670	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110678/110678	1.000
Male							
Ch06 GSM High	201.3 to 269.0	222.93	0.40/10.56	140.8	316.8	110628/110702	0.999
Ch05 GSM Med	142.4 to 192.2	173.85	0.38/7.74	128.3	226.9	110700/110702	1.000
Ch09 GSM Low	103.1 to 135.9	108.94	0.30/3.85	80.3	143.4	110474/110678	0.998
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110702/110702	1.000
November 1 to 30, 2012							
Female							
Ch17 GSM High	232.8 to 250.7	222.28	0.40/10.48	178.3	284.9	107584/107706	0.999
Ch16 GSM Med	164.6 to 177.3	156.73	0.35/6.43	129.5	196.0	107652/107706	0.999
Ch20 GSM Low	118.5 to 125.4	116.79	0.61/8.50	85.0	170.5	107364/107706	0.997
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107706/107706	1.000
Male							
Ch06 GSM High	271.8 to 292.2	263.30	0.42/13.17	212.1	339.0	107672/107706	1.000
Ch05 GSM Med	194.2 to 208.2	205.84	0.42/10.20	164.2	257.0	107706/107706	1.000
Ch09 GSM Low	137.4 to 148.2	127.09	0.31/4.62	105.4	150.3	107422/107706	0.997
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107706/107706	1.000
December 1 to 31, 2012							
Female							
Ch17 GSM High	250.7 to 254.9	232.81	0.41/11.12	177.9	303.9	110988/111216	0.998
Ch16 GSM Med	177.3 to 180.2	165.11	0.38/7.41	135.9	208.6	111180/111216	1.000
Ch20 GSM Low	125.4 to 129.2	112.68	0.32/4.18	94.9	136.5	110810/111216	0.996
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111216/111216	1.000
Male							
Ch06 GSM High	292.2 to 298.0	275.64	0.45/14.54	220.5	356.3	111140/111216	0.999
Ch05 GSM Med	208.2 to 211.7	219.30	0.44/11.41	173.0	271.3	111208/111216	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch09 GSM Low	148.2 to 150.2	132.15	0.32/4.89	113.4	153.0	110840/111216	0.997
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111216/111216	1.000
January 1 to 31, 2013							
Female							
Ch17 GSM High	254.9 to 261.4	237.27	0.41/11.43	193.2	293.0	111138/111236	0.999
Ch16 GSM Med	180.2 to 184.8	167.60	0.34/6.66	141.7	198.4	111220/111238	1.000
Ch20 GSM Low	129.2 to 130.7	115.91	0.32/4.36	99.7	140.3	111140/111234	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111238/111238	1.000
Male							
Ch06 GSM High	298.0 to 302.1	279.82	0.47/15.40	220.4	362.2	111106/111238	0.999
Ch05 GSM Med	211.7 to 214.0	221.30	0.45/11.71	179.9	275.1	111236/111238	1.000
Ch09 GSM Low	150.2 to 151.4	135.72	0.33/5.19	114.5	174.3	111180/111238	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111238/111238	1.000
February 1 to 28, 2013							
Female							
Ch17 GSM High	261.4 to 264.0	244.04	0.40/11.51	201.9	309.0	100140/100164	1.000
Ch16 GSM Med	184.8 to 186.7	174.32	0.38/7.89	145.5	216.4	100160/100164	1.000
Ch20 GSM Low	130.7 to 132.0	116.79	0.32/4.36	99.0	141.3	100012/100164	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	100164/100164	1.000
Male							
Ch06 GSM High	302.1 to 304.0	276.46	0.45/14.58	221.6	347.2	99924/100164	0.998
Ch05 GSM Med	214.0 to 215.5	227.95	0.47/12.61	186.4	279.7	100128/100164	1.000
Ch09 GSM Low	151.4 to 152.4	136.55	0.32/5.04	114.7	160.3	100106/100164	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	100164/100164	1.000
March 1 to 31, 2013							
Female							
Ch17 GSM High	264.0 to 266.5	248.07	0.39/11.49	197.3	308.4	111392/111408	1.000
Ch16 GSM Med	186.7 to 188.4	178.55	0.38/7.88	147.1	212.8	111396/111408	1.000
Ch20 GSM Low	132.0 to 133.2	117.42	0.32/4.39	99.1	143.9	111142/111408	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
Male							
Ch06 GSM High	304.0 to 305.6	278.61	0.46/15.06	224.1	380.8	111090/111408	0.997
Ch05 GSM Med	215.5 to 216.8	229.50	0.45/12.26	186.4	283.8	111380/111408	1.000
Ch09 GSM Low	152.4 to 153.3	136.42	0.31/4.94	115.9	162.5	111286/111408	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
April 1 to 30, 2013							
Female							
Ch17 GSM High	266.5 to 269.0	250.31	0.41/11.95	207.4	314.9	107404/107438	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch16 GSM Med	188.4 to 190.2	178.92	0.40/8.46	147.1	219.1	107426/107438	1.000
Ch20 GSM Low	133.2 to 134.5	119.19	0.32/4.45	101.7	149.3	107286/107438	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107438/107438	1.000
Male							
Ch06 GSM High	305.6 to 307.6	280.59	0.47/15.43	229.2	372.4	107104/107442	0.997
Ch05 GSM Med	216.8 to 218.3	231.77	0.46/12.63	189.1	288.7	107402/107442	1.000
Ch09 GSM Low	153.3 to 154.4	137.80	0.32/5.12	111.5	163.3	107268/107438	0.998
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107444/107444	1.000
May 1 to 31, 2013							
Female							
Ch17 GSM High	269.0 to 271.8	256.26	0.44/13.37	184.1	324.2	105446/105524	0.999
Ch16 GSM Med	190.2 to 190.2	179.56	0.38/8.02	138.8	219.5	105504/105524	1.000
Ch20 GSM Low	134.5 to 135.9	121.06	0.33/4.64	90.0	145.1	105262/105524	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105524/105524	1.000
Male							
Ch06 GSM High	307.6 to 308.8	278.54	0.46/15.09	206.9	359.6	104946/105578	0.994
Ch05 GSM Med	218.3 to 220.1	237.22	0.49/13.63	151.8	290.6	105468/105580	0.999
Ch09 GSM Low	154.4 to 155.6	137.77	0.43/7.00	88.7	209.9	104226/105576	0.987
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105580/105580	1.000
June 1 to 30, 2013							
Female							
Ch17 GSM High	271.8 to 274.7	252.40	0.44/13.06	194.3	343.6	106884/107074	0.998
Ch16 GSM Med	190.2 to 192.2	180.43	0.40/8.49	138.4	219.4	106978/107074	0.999
Ch20 GSM Low	135.9 to 137.4	121.62	0.33/4.72	97.5	143.3	106782/107074	0.997
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107074/107074	1.000
Male							
Ch06 GSM High	308.8 to 311.3	275.67	0.44/14.27	212.0	349.6	105800/107088	0.988
Ch05 GSM Med	220.1 to 221.0	236.86	0.48/13.34	191.9	295.4	107012/107088	0.999
Ch09 GSM Low	155.6 to 157.0	140.60	0.33/5.40	119.5	168.0	106942/107074	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107088/107088	1.000
July 1 to 31, 2013							
Female							
Ch17 GSM High	274.7 to 277.7	253.80	0.40/11.87	205.8	314.3	110994/111054	0.999
Ch16 GSM Med	192.2 to 196.4	182.06	0.39/8.41	151.1	219.7	111034/111054	1.000
Ch20 GSM Low	137.4 to 140.4	122.97	0.32/4.66	105.2	149.3	110832/111054	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111054/111054	1.000
Male							
Ch06 GSM High	311.3 to 315.3	278.80	0.44/14.64	223.1	369.0	109898/111062	0.990

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch05 GSM Med	221.0 to 223.9	237.76	0.50/14.21	189.2	300.6	110936/111062	0.999
Ch09 GSM Low	157.0 to 159.0	140.17	0.33/5.51	115.0	170.4	110664/111054	0.996
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111062/111062	1.000
August 1 to 31, 2013							
Female							
Ch17 GSM High	277.7 to 280.8	257.67	0.43/12.95	208.3	319.3	111830/111904	0.999
Ch16 GSM Med	196.4 to 198.5	182.52	0.39/8.42	151.0	222.3	111850/111904	1.000
Ch20 GSM Low	140.4 to 140.4	124.67	0.33/4.84	104.3	150.3	111676/111904	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111904/111904	1.000
Male							
Ch06 GSM High	315.3 to 316.7	279.34	0.45/14.72	215.4	372.8	110478/111910	0.987
Ch05 GSM Med	223.9 to 224.9	237.25	0.49/13.81	192.1	293.2	111860/111910	1.000
Ch09 GSM Low	159.0 to 159.7	142.43	0.32/5.41	120.6	169.9	111800/111904	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111910/111910	1.000
September 1 to 30, 2013							
Female							
Ch17 GSM High	280.8 to 280.8	262.26	0.42/13.14	204.1	327.2	107316/107392	0.999
Ch16 GSM Med	198.5 to 200.7	185.57	0.39/8.61	139.3	226.1	107296/107392	0.999
Ch20 GSM Low	140.4 to 141.9	125.73	0.33/4.86	106.5	148.1	107242/107392	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107392/107392	1.000
Male							
Ch06 GSM High	316.7 to 318.1	281.16	0.48/15.95	224.7	390.2	105756/107406	0.985
Ch05 GSM Med	224.9 to 225.9	238.83	0.50/14.05	187.3	293.1	107356/107406	1.000
Ch09 GSM Low	159.7 to 161.0	142.21	0.32/5.37	119.3	173.7	107192/107392	0.998
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107406/107406	1.000
October 1 to 31, 2013							
Female							
Ch17 GSM High	280.8 to 286.8	266.84	0.48/15.15	202.3	349.4	113156/113496	0.997
Ch16 GSM Med	200.7 to 202.8	191.15	0.50/11.29	128.8	245.9	113068/113496	0.996
Ch20 GSM Low	141.9 to 143.4	126.25	0.35/5.17	96.2	153.1	112706/113494	0.993
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	113496/113496	1.000
Male							
Ch06 GSM High	318.1 to 319.4	282.54	0.53/17.77	38.2	423.6	110868/113614	0.976
Ch05 GSM Med	225.9 to 226.8	241.27	0.54/15.50	186.8	302.8	113456/113614	0.999
Ch09 GSM Low	161.0 to 161.7	143.81	0.35/5.96	116.8	176.8	113012/113496	0.996
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	113614/113614	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
November 1 to 30, 2013							
Female							
Ch17 GSM High	286.8 to 286.8	273.00	0.45/14.54	204.0	343.4	110616/110646	1.000
Ch16 GSM Med	202.8 to 204.8	192.79	0.42/9.55	148.3	241.8	110586/110646	0.999
Ch20 GSM Low	143.4 to 144.8	127.11	0.34/5.01	108.4	153.5	110198/110646	0.996
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110646/110646	1.000
Male							
Ch06 GSM High	319.4 to 320.8	287.34	0.48/16.25	225.8	357.9	109554/110664	0.990
Ch05 GSM Med	226.8 to 227.7	241.65	0.50/14.42	165.6	302.3	110582/110664	0.999
Ch09 GSM Low	161.7 to 161.7	143.34	0.34/5.76	114.0	178.7	110244/110658	0.996
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110664/110664	1.000
December 1 to 31, 2013							
Female							
Ch17 GSM High	286.8 to 289.6	274.74	0.47/15.25	222.7	356.4	106174/106198	1.000
Ch16 GSM Med	204.8 to 206.7	194.33	0.40/9.11	158.6	234.0	106188/106198	1.000
Ch20 GSM Low	144.8 to 146.1	129.74	0.31/4.64	109.5	154.2	106008/106114	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	106198/106198	1.000
Male							
Ch06 GSM High	320.8 to 322.1	286.88	0.47/15.93	231.5	370.7	104998/106222	0.988
Ch05 GSM Med	227.7 to 228.7	241.29	0.50/14.36	188.7	304.7	106184/106222	1.000
Ch09 GSM Low	161.7 to 162.4	140.55	0.32/5.22	119.5	166.0	105262/106198	0.991
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	106222/106222	1.000
January 1 to 31, 2014							
Female							
Ch17 GSM High	289.6 to 294.5	273.60	0.44/14.08	213.1	345.2	111208/111252	1.000
Ch16 GSM Med	206.7 to 208.2	197.35	0.40/9.31	156.6	238.4	111238/111252	1.000
Ch20 GSM Low	146.1 to 147.3	131.21	0.31/4.77	110.5	164.6	111166/111242	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111252/111252	1.000
Male							
Ch06 GSM High	322.1 to 323.4	286.59	0.45/15.24	232.0	364.4	109952/111258	0.988
Ch05 GSM Med	228.7 to 229.6	241.27	0.53/15.06	187.2	311.8	111150/111258	0.999
Ch09 GSM Low	162.4 to 163.0	141.77	0.32/5.40	114.6	169.4	110336/111254	0.992
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111258/111258	1.000
February 1 to 28, 2014							
Female							
Ch17 GSM High	294.5 to 296.4	278.51	0.43/14.19	224.9	353.9	103924/103948	1.000
Ch16 GSM Med	208.2 to 208.2	198.50	0.40/9.28	165.0	239.6	103946/103948	1.000
Ch20 GSM Low	147.3 to 149.0	132.92	0.31/4.85	112.3	157.0	103880/103948	0.999

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	103948/103948	1.000
Male							
Ch06 GSM High	323.4 to 324.8	290.93	0.45/15.63	229.9	371.2	103072/103960	0.991
Ch05 GSM Med	229.6 to 230.6	241.40	0.50/14.31	197.7	300.3	103924/103960	1.000
Ch09 GSM Low	163.0 to 163.7	143.34	0.32/5.39	119.8	171.2	103444/103948	0.995
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	103960/103960	1.000
March 1 to 31, 2014							
Female							
Ch17 GSM High	296.4 to 298.0	281.01	0.42/13.93	212.2	352.2	111382/111408	1.000
Ch16 GSM Med	208.2 to 209.6	200.57	0.41/9.73	161.8	246.5	111400/111408	1.000
Ch20 GSM Low	149.0 to 149.7	133.58	0.32/4.93	106.6	154.5	111256/111408	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
Male							
Ch06 GSM High	324.8 to 324.8	290.82	0.48/16.36	223.2	381.3	110096/111408	0.988
Ch05 GSM Med	230.6 to 231.5	242.18	0.53/15.28	194.0	312.6	111336/111408	0.999
Ch09 GSM Low	163.7 to 164.4	144.23	0.32/5.33	122.6	175.8	111024/111408	0.997
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
April 1 to 30, 2014							
Female							
Ch17 GSM High	298.0 to 299.3	279.24	0.44/14.60	206.0	347.1	107100/107288	0.998
Ch16 GSM Med	209.6 to 210.7	202.29	0.43/10.22	153.5	249.1	107228/107288	0.999
Ch20 GSM Low	149.7 to 150.2	134.98	0.32/5.09	111.6	156.3	107172/107286	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107288/107288	1.000
Male							
Ch06 GSM High	324.8 to 326.1	293.70	0.49/17.15	81.1	373.8	106068/107298	0.989
Ch05 GSM Med	231.5 to 231.5	242.46	0.53/15.16	196.6	306.2	107220/107298	0.999
Ch09 GSM Low	164.4 to 164.4	144.09	0.32/5.48	119.7	170.6	106748/107290	0.995
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107298/107298	1.000
May 1 to 31, 2014							
Female							
Ch17 GSM High	299.3 to 300.4	285.77	0.45/15.19	226.9	363.3	111176/111208	1.000
Ch16 GSM Med	210.7 to 211.7	193.86	0.39/8.95	154.6	239.6	111128/111208	0.999
Ch20 GSM Low	150.2 to 150.2	134.45	0.34/5.30	108.5	158.8	111012/111204	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111208/111208	1.000
Male							
Ch06 GSM High	326.1 to 326.1	296.46	0.46/16.14	237.1	375.6	110684/111208	0.995
Ch05 GSM Med	231.5 to 231.5	207.41	0.47/11.55	162.7	306.7	110450/111208	0.993
Ch09 GSM Low	164.4 to 165.1	153.28	0.43/7.71	123.4	195.4	111094/111208	0.999

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111208/111208	1.000
June 1 to 30, 2014							
Female							
Ch17 GSM High	300.4 to 302.1	288.24	0.47/16.20	235.2	380.3	107530/107542	1.000
Ch16 GSM Med	211.7 to 213.1	200.74	0.42/9.91	138.6	261.5	107508/107542	1.000
Ch20 GSM Low	150.2 to 151.0	134.74	0.33/5.24	95.9	177.0	107376/107534	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107542/107542	1.000
Male							
Ch06 GSM High	326.1 to 327.4	295.01	0.46/16.13	225.9	406.2	106940/107546	0.994
Ch05 GSM Med	231.5 to 231.5	207.94	0.45/11.15	77.9	266.6	106192/107548	0.987
Ch09 GSM Low	163.7 to 165.1	152.64	0.39/7.06	64.5	187.5	107418/107546	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107548/107548	1.000
July 1 to 31, 2014							
Female							
Ch17 GSM High	302.1 to 302.7	289.35	0.45/15.51	220.3	362.7	111178/111202	1.000
Ch16 GSM Med	213.1 to 213.1	199.95	0.39/9.14	160.5	244.0	111182/111202	1.000
Ch20 GSM Low	151.0 to 151.4	135.12	0.31/4.98	99.5	156.3	111052/111202	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111202/111202	1.000
Male							
Ch06 GSM High	326.1 to 327.4	289.86	0.44/15.18	198.9	378.9	110010/111206	0.989
Ch05 GSM Med	229.6 to 231.5	215.61	0.41/10.50	174.4	265.3	111174/111206	1.000
Ch09 GSM Low	162.4 to 163.7	152.19	0.36/6.35	125.4	187.1	111176/111202	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111206/111206	1.000
August 1 to 31, 2014							
Female							
Ch17 GSM High	302.7 to 303.4	290.91	0.45/15.30	205.3	369.6	108668/108708	1.000
Ch16 GSM Med	213.1 to 213.6	200.65	0.39/9.18	153.8	243.3	108630/108708	0.999
Ch20 GSM Low	151.4 to 151.7	134.02	0.36/5.60	102.4	159.8	107888/108708	0.992
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	108708/108708	1.000
Male							
Ch06 GSM High	324.8 to 327.4	290.86	0.45/15.32	231.0	374.5	107704/108716	0.991
Ch05 GSM Med	227.7 to 229.6	215.05	0.41/10.32	175.0	270.0	108704/108716	1.000
Ch09 GSM Low	162.4 to 163.0	153.30	0.37/6.76	128.0	181.8	108702/108708	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	108716/108716	1.000
September 1 to 30, 2014							
Female							
Ch17 GSM High	303.4 to 303.4	294.02	0.46/15.88	194.5	436.8	104160/104176	1.000
Ch16 GSM Med	213.6 to 214.5	204.29	0.41/9.82	167.9	279.2	104168/104176	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch20 GSM Low	151.4 to 151.7	138.14	0.33/5.32	111.3	184.1	104052/104176	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	104176/104176	1.000
Male							
Ch06 GSM High	324.8 to 324.8	299.88	0.49/17.46	204.7	441.0	103900/104176	0.997
Ch05 GSM Med	227.7 to 227.7	224.63	0.61/16.43	174.7	308.7	104148/104176	1.000
Ch09 GSM Low	161.7 to 162.4	158.10	0.37/6.84	127.6	210.1	104170/104176	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	104176/104176	1.000
August 8, 2012 to September 30, 2014							
Female							
Ch17 GSM High	192.6 to 303.4	260.77	0.47/14.52	137.3	436.8	2797766/2800508	0.999
Ch16 GSM Med	136.2 to 214.5	184.16	0.41/8.99	101.6	279.2	2799216/2800510	1.000
Ch20 GSM Low	96.1 to 151.7	124.97	0.40/5.95	71.8	184.1	2794544/2800388	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	2800510/2800510	1.000
Male							
Ch06 GSM High	192.6 to 327.4	278.30	0.48/15.93	38.2	445.8	2779842/2800838	0.993
Ch05 GSM Med	136.2 to 231.5	224.32	0.65/17.31	77.9	323.0	2797270/2800842	0.999
Ch09 GSM Low	96.3 to 165.1	139.28	0.40/6.61	64.5	210.1	2792562/2800582	0.997
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	2800844/2800844	1.000

Ch = chamber (e.g., Ch17 = Chamber 17).

Table I-4. Summary of GSM-modulated Cell Phone RFR Exposure Data in Rats – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
August 8 to 31, 2012							
Female							
Ch17 GSM High	0.65 to 0.75	0.687	0.36/0.029	0.50	0.97	85734/85786	0.999
Ch16 GSM Med	0.46 to 0.53	0.505	0.30/0.018	0.40	0.67	85774/85786	1.000
Ch20 GSM Low	0.33 to 0.38	0.370	0.30/0.013	0.29	0.50	85714/85786	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	85786/85786	1.000
September 1 to 30, 2012							
Female							
Ch17 GSM High	0.51 to 0.71	0.647	0.59/0.046	0.42	0.97	105690/105918	0.998
Ch16 GSM Med	0.36 to 0.51	0.460	0.41/0.022	0.29	0.68	105834/105918	0.999
Ch20 GSM Low	0.26 to 0.36	0.327	0.37/0.014	0.21	0.50	105824/105918	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	105918/105918	1.000
Male							
Ch06 GSM High	0.51 to 0.71	0.645	0.41/0.031	0.29	1.17	105800/105918	0.999
Ch05 GSM Med	0.36 to 0.51	0.413	0.37/0.018	0.26	0.66	105892/105918	1.000
Ch09 GSM Low	0.26 to 0.36	0.328	0.38/0.015	0.21	0.59	105716/105918	0.998

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	105918/105918	1.000
October 1 to 31, 2012							
Female							
Ch17 GSM High	0.51 to 0.62	0.623	0.46/0.034	0.46	0.85	110476/110678	0.998
Ch16 GSM Med	0.36 to 0.44	0.435	0.39/0.020	0.30	0.59	110654/110678	1.000
Ch20 GSM Low	0.26 to 0.31	0.291	0.36/0.012	0.22	0.40	110646/110670	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	110678/110678	1.000
Male							
Ch06 GSM High	0.53 to 0.71	0.676	0.42/0.033	0.47	0.90	110666/110702	1.000
Ch05 GSM Med	0.38 to 0.51	0.444	0.35/0.018	0.32	0.61	110702/110702	1.000
Ch09 GSM Low	0.27 to 0.36	0.356	0.35/0.015	0.24	0.46	110620/110678	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	110702/110702	1.000
November 1 to 30, 2012							
Female							
Ch17 GSM High	0.62 to 0.67	0.695	0.45/0.037	0.56	0.92	107658/107706	1.000
Ch16 GSM Med	0.44 to 0.47	0.496	0.39/0.022	0.42	0.60	107694/107706	1.000
Ch20 GSM Low	0.31 to 0.33	0.338	0.59/0.024	0.27	0.44	107688/107706	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107706/107706	1.000
Male							
Ch06 GSM High	0.72 to 0.78	0.801	0.43/0.041	0.63	1.04	107688/107706	1.000
Ch05 GSM Med	0.52 to 0.55	0.525	0.38/0.023	0.44	0.65	107706/107706	1.000
Ch09 GSM Low	0.36 to 0.39	0.422	0.35/0.017	0.35	0.51	107682/107706	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107706/107706	1.000
December 1 to 31, 2012							
Female							
Ch17 GSM High	0.67 to 0.68	0.725	0.44/0.037	0.58	0.90	111152/111216	0.999
Ch16 GSM Med	0.47 to 0.48	0.513	0.41/0.025	0.42	0.63	111202/111216	1.000
Ch20 GSM Low	0.33 to 0.34	0.378	0.34/0.015	0.33	0.46	111172/111216	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111216/111216	1.000
Male							
Ch06 GSM High	0.78 to 0.79	0.837	0.43/0.043	0.69	1.04	111200/111216	1.000
Ch05 GSM Med	0.55 to 0.56	0.535	0.37/0.023	0.45	0.64	111216/111216	1.000
Ch09 GSM Low	0.39 to 0.40	0.439	0.37/0.019	0.36	0.52	111182/111216	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111216/111216	1.000
January 1 to 31, 2013							
Female							
Ch17 GSM High	0.68 to 0.69	0.737	0.44/0.039	0.60	0.95	111146/111236	0.999
Ch16 GSM Med	0.48 to 0.49	0.523	0.38/0.023	0.44	0.64	111226/111238	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch20 GSM Low	0.34 to 0.35	0.380	0.34/0.015	0.32	0.47	111214/111234	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111238/111238	1.000
Male							
Ch06 GSM High	0.79 to 0.80	0.842	0.44/0.043	0.70	1.05	111228/111238	1.000
Ch05 GSM Med	0.56 to 0.57	0.540	0.33/0.021	0.45	0.64	111238/111238	1.000
Ch09 GSM Low	0.40 to 0.40	0.438	0.36/0.019	0.37	0.58	111134/111238	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111238/111238	1.000
February 1 to 28, 2013							
Female							
Ch17 GSM High	0.69 to 0.70	0.742	0.43/0.037	0.61	0.91	100134/100164	1.000
Ch16 GSM Med	0.49 to 0.50	0.521	0.37/0.023	0.43	0.61	100164/100164	1.000
Ch20 GSM Low	0.35 to 0.35	0.386	0.35/0.016	0.33	0.48	100150/100164	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	100164/100164	1.000
Male							
Ch06 GSM High	0.80 to 0.81	0.870	0.47/0.049	0.72	1.08	100074/100164	0.999
Ch05 GSM Med	0.57 to 0.57	0.535	0.36/0.023	0.45	0.64	100160/100164	1.000
Ch09 GSM Low	0.40 to 0.40	0.441	0.37/0.019	0.37	0.53	100152/100164	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	100164/100164	1.000
March 1 to 31, 2013							
Female							
Ch17 GSM High	0.70 to 0.71	0.746	0.41/0.036	0.63	0.93	111390/111408	1.000
Ch16 GSM Med	0.50 to 0.50	0.520	0.37/0.023	0.43	0.63	111408/111408	1.000
Ch20 GSM Low	0.35 to 0.35	0.391	0.34/0.016	0.33	0.46	111396/111408	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111408/111408	1.000
Male							
Ch06 GSM High	0.81 to 0.81	0.870	0.46/0.047	0.72	1.06	111368/111408	1.000
Ch05 GSM Med	0.57 to 0.58	0.537	0.36/0.022	0.45	0.64	111402/111408	1.000
Ch09 GSM Low	0.40 to 0.41	0.446	0.36/0.019	0.38	0.54	111396/111408	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111408/111408	1.000
April 1 to 30, 2013							
Female							
Ch17 GSM High	0.71 to 0.71	0.756	0.43/0.038	0.62	0.91	107424/107438	1.000
Ch16 GSM Med	0.50 to 0.51	0.530	0.39/0.025	0.44	0.62	107438/107438	1.000
Ch20 GSM Low	0.35 to 0.36	0.393	0.34/0.016	0.34	0.46	107430/107438	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107438/107438	1.000
Male							
Ch06 GSM High	0.81 to 0.82	0.876	0.47/0.049	0.70	1.10	107396/107442	1.000
Ch05 GSM Med	0.58 to 0.58	0.541	0.35/0.022	0.46	0.64	107440/107442	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch09 GSM Low	0.41 to 0.41	0.449	0.37/0.019	0.39	0.54	107406/107438	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107444/107444	1.000
May 1 to 31, 2013							
Female							
Ch17 GSM High	0.71 to 0.72	0.752	0.44/0.039	0.55	0.94	105516/105524	1.000
Ch16 GSM Med	0.51 to 0.51	0.529	0.40/0.025	0.39	0.62	105522/105524	1.000
Ch20 GSM Low	0.36 to 0.36	0.395	0.33/0.015	0.29	0.46	105514/105524	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	105524/105524	1.000
Male							
Ch06 GSM High	0.82 to 0.82	0.891	0.49/0.052	0.68	1.17	105416/105578	0.998
Ch05 GSM Med	0.58 to 0.58	0.535	0.35/0.022	0.32	0.64	105564/105580	1.000
Ch09 GSM Low	0.41 to 0.41	0.454	0.41/0.022	0.36	0.60	105438/105576	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	105580/105580	1.000
June 1 to 30, 2013							
Female							
Ch17 GSM High	0.72 to 0.73	0.780	0.46/0.043	0.60	0.95	107056/107074	1.000
Ch16 GSM Med	0.51 to 0.51	0.537	0.39/0.025	0.45	0.65	107070/107074	1.000
Ch20 GSM Low	0.36 to 0.36	0.403	0.35/0.016	0.34	0.47	107056/107074	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107074/107074	1.000
Male							
Ch06 GSM High	0.82 to 0.83	0.907	0.50/0.053	0.71	1.14	106866/107088	0.998
Ch05 GSM Med	0.58 to 0.59	0.540	0.34/0.021	0.46	0.64	107084/107088	1.000
Ch09 GSM Low	0.41 to 0.42	0.455	0.37/0.020	0.37	0.54	107054/107074	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107088/107088	1.000
July 1 to 31, 2013							
Female							
Ch17 GSM High	0.73 to 0.74	0.780	0.45/0.041	0.65	0.95	111028/111054	1.000
Ch16 GSM Med	0.51 to 0.52	0.545	0.40/0.025	0.46	0.65	111054/111054	1.000
Ch20 GSM Low	0.36 to 0.37	0.408	0.34/0.016	0.34	0.48	111044/111054	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111054/111054	1.000
Male							
Ch06 GSM High	0.83 to 0.84	0.913	0.49/0.053	0.73	1.18	110836/111062	0.998
Ch05 GSM Med	0.59 to 0.59	0.548	0.35/0.023	0.45	0.66	111048/111062	1.000
Ch09 GSM Low	0.42 to 0.42	0.461	0.40/0.022	0.39	0.57	110988/111054	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111062/111062	1.000
August 1 to 31, 2013							
Female							
Ch17 GSM High	0.74 to 0.75	0.789	0.44/0.041	0.64	0.97	111878/111904	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch16 GSM Med	0.52 to 0.53	0.557	0.40/0.026	0.46	0.66	111902/111904	1.000
Ch20 GSM Low	0.37 to 0.37	0.412	0.32/0.016	0.36	0.48	111892/111904	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111904/111904	1.000
Male							
Ch06 GSM High	0.84 to 0.84	0.919	0.49/0.053	0.74	1.15	111660/111910	0.998
Ch05 GSM Med	0.59 to 0.60	0.553	0.35/0.023	0.48	0.66	111910/111910	1.000
Ch09 GSM Low	0.42 to 0.42	0.462	0.38/0.021	0.38	0.55	111884/111904	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111910/111910	1.000
September 1 to 30, 2013							
Female							
Ch17 GSM High	0.75 to 0.75	0.788	0.44/0.041	0.63	0.98	107388/107392	1.000
Ch16 GSM Med	0.53 to 0.53	0.561	0.42/0.028	0.45	0.68	107386/107392	1.000
Ch20 GSM Low	0.37 to 0.38	0.411	0.34/0.016	0.35	0.49	107382/107392	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107392/107392	1.000
Male							
Ch06 GSM High	0.84 to 0.84	0.925	0.50/0.055	0.76	1.15	107170/107406	0.998
Ch05 GSM Med	0.60 to 0.60	0.557	0.35/0.023	0.46	0.66	107386/107406	1.000
Ch09 GSM Low	0.42 to 0.43	0.468	0.38/0.021	0.37	0.56	107326/107392	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107406/107406	1.000
October 1 to 31, 2013							
Female							
Ch17 GSM High	0.75 to 0.76	0.787	0.50/0.047	0.60	1.00	113450/113496	1.000
Ch16 GSM Med	0.53 to 0.54	0.558	0.48/0.032	0.42	0.67	113476/113496	1.000
Ch20 GSM Low	0.38 to 0.38	0.418	0.39/0.019	0.33	0.49	113476/113494	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	113496/113496	1.000
Male							
Ch06 GSM High	0.84 to 0.85	0.931	0.56/0.062	0.60	1.34	113016/113614	0.995
Ch05 GSM Med	0.60 to 0.60	0.560	0.39/0.025	0.46	0.69	113554/113614	0.999
Ch09 GSM Low	0.43 to 0.43	0.471	0.38/0.021	0.39	0.59	113456/113496	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	113614/113614	1.000
November 1 to 30, 2013							
Female							
Ch17 GSM High	0.76 to 0.76	0.794	0.43/0.041	0.62	0.99	110632/110646	1.000
Ch16 GSM Med	0.54 to 0.54	0.567	0.41/0.028	0.45	0.69	110644/110646	1.000
Ch20 GSM Low	0.38 to 0.38	0.424	0.34/0.017	0.35	0.50	110622/110646	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	110646/110646	1.000
Male							
Ch06 GSM High	0.85 to 0.85	0.926	0.49/0.054	0.74	1.17	110468/110664	0.998

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch05 GSM Med	0.60 to 0.60	0.563	0.35/0.023	0.48	0.68	110664/110664	1.000
Ch09 GSM Low	0.43 to 0.43	0.474	0.35/0.020	0.41	0.58	110642/110658	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	110664/110664	1.000
December 1 to 31, 2013							
Female							
Ch17 GSM High	0.76 to 0.77	0.797	0.48/0.046	0.63	0.99	106186/106198	1.000
Ch16 GSM Med	0.54 to 0.55	0.573	0.41/0.028	0.47	0.69	106196/106198	1.000
Ch20 GSM Low	0.38 to 0.39	0.425	0.32/0.016	0.38	0.49	106106/106114	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	106198/106198	1.000
Male							
Ch06 GSM High	0.85 to 0.85	0.934	0.48/0.053	0.78	1.19	106006/106222	0.998
Ch05 GSM Med	0.60 to 0.61	0.567	0.37/0.024	0.44	0.67	106114/106222	0.999
Ch09 GSM Low	0.43 to 0.43	0.485	0.34/0.019	0.42	0.57	106114/106198	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	106222/106222	1.000
January 1 to 31, 2014							
Female							
Ch17 GSM High	0.77 to 0.78	0.821	0.46/0.045	0.68	1.04	111214/111252	1.000
Ch16 GSM Med	0.55 to 0.55	0.574	0.40/0.027	0.45	0.68	111252/111252	1.000
Ch20 GSM Low	0.39 to 0.39	0.427	0.34/0.017	0.35	0.51	111232/111242	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111252/111252	1.000
Male							
Ch06 GSM High	0.85 to 0.86	0.940	0.48/0.054	0.79	1.18	110934/111258	0.997
Ch05 GSM Med	0.61 to 0.61	0.573	0.38/0.026	0.48	0.68	111256/111258	1.000
Ch09 GSM Low	0.43 to 0.43	0.486	0.36/0.020	0.42	0.58	111152/111254	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111258/111258	1.000
February 1 to 28, 2014							
Female							
Ch17 GSM High	0.78 to 0.79	0.828	0.45/0.044	0.69	1.05	103936/103948	1.000
Ch16 GSM Med	0.55 to 0.55	0.575	0.37/0.025	0.49	0.67	103948/103948	1.000
Ch20 GSM Low	0.39 to 0.40	0.433	0.32/0.016	0.37	0.51	103938/103948	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	103948/103948	1.000
Male							
Ch06 GSM High	0.86 to 0.86	0.937	0.48/0.053	0.77	1.17	103800/103960	0.998
Ch05 GSM Med	0.61 to 0.61	0.577	0.38/0.025	0.48	0.67	103956/103960	1.000
Ch09 GSM Low	0.43 to 0.43	0.486	0.35/0.020	0.42	0.56	103898/103948	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	103960/103960	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
March 1 to 31, 2014							
Female							
Ch17 GSM High	0.79 to 0.79	0.830	0.47/0.046	0.66	1.04	111398/111408	1.000
Ch16 GSM Med	0.55 to 0.56	0.574	0.38/0.026	0.45	0.66	111408/111408	1.000
Ch20 GSM Low	0.40 to 0.40	0.435	0.32/0.016	0.36	0.54	111402/111408	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111408/111408	1.000
Male							
Ch06 GSM High	0.86 to 0.86	0.936	0.48/0.053	0.78	1.18	111174/111408	0.998
Ch05 GSM Med	0.61 to 0.61	0.574	0.37/0.025	0.49	0.68	111408/111408	1.000
Ch09 GSM Low	0.43 to 0.44	0.486	0.34/0.020	0.41	0.58	111356/111408	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111408/111408	1.000
April 1 to 30, 2014							
Female							
Ch17 GSM High	0.79 to 0.79	0.841	0.49/0.049	0.63	1.04	107254/107288	1.000
Ch16 GSM Med	0.56 to 0.56	0.576	0.41/0.027	0.46	0.69	107288/107288	1.000
Ch20 GSM Low	0.40 to 0.40	0.436	0.33/0.017	0.38	0.50	107286/107286	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107288/107288	1.000
Male							
Ch06 GSM High	0.86 to 0.87	0.928	0.46/0.050	0.39	1.17	107206/107298	0.999
Ch05 GSM Med	0.61 to 0.61	0.577	0.37/0.025	0.49	0.68	107298/107298	1.000
Ch09 GSM Low	0.44 to 0.44	0.488	0.36/0.021	0.41	0.57	107212/107290	0.999
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107298/107298	1.000
May 1 to 31, 2014							
Female							
Ch17 GSM High	0.79 to 0.80	0.834	0.47/0.047	0.67	1.03	111186/111208	1.000
Ch16 GSM Med	0.56 to 0.56	0.605	0.40/0.029	0.49	0.74	111192/111208	1.000
Ch20 GSM Low	0.40 to 0.40	0.438	0.33/0.017	0.37	0.53	111196/111204	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111208/111208	1.000
Male							
Ch06 GSM High	0.87 to 0.87	0.931	0.47/0.051	0.78	1.23	111120/111208	0.999
Ch05 GSM Med	0.61 to 0.61	0.641	0.45/0.034	0.51	0.77	111208/111208	1.000
Ch09 GSM Low	0.44 to 0.44	0.468	0.37/0.021	0.40	0.56	111204/111208	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111208/111208	1.000
June 1 to 30, 2014							
Female							
Ch17 GSM High	0.80 to 0.80	0.832	0.46/0.046	0.67	1.03	107526/107542	1.000
Ch16 GSM Med	0.56 to 0.57	0.593	0.40/0.028	0.41	0.69	107516/107542	1.000
Ch20 GSM Low	0.40 to 0.40	0.441	0.34/0.018	0.29	0.52	107508/107534	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107542/107542	1.000
Male							
Ch06 GSM High	0.87 to 0.87	0.940	0.47/0.053	0.67	1.16	107402/107546	0.999
Ch05 GSM Med	0.61 to 0.61	0.652	0.49/0.038	0.24	0.85	107538/107548	1.000
Ch09 GSM Low	0.43 to 0.44	0.466	0.35/0.019	0.21	0.55	107532/107546	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107548/107548	1.000
July 1 to 31, 2014							
Female							
Ch17 GSM High	0.80 to 0.80	0.832	0.48/0.048	0.64	1.05	111188/111202	1.000
Ch16 GSM Med	0.57 to 0.57	0.595	0.40/0.028	0.44	0.70	111200/111202	1.000
Ch20 GSM Low	0.40 to 0.40	0.442	0.33/0.017	0.37	0.52	111186/111202	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111202/111202	1.000
Male							
Ch06 GSM High	0.87 to 0.87	0.947	0.47/0.052	0.79	1.28	110960/111206	0.998
Ch05 GSM Med	0.61 to 0.61	0.646	0.45/0.034	0.46	0.80	111194/111206	1.000
Ch09 GSM Low	0.43 to 0.43	0.463	0.34/0.018	0.36	0.54	111202/111202	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111206/111206	1.000
August 1 to 31, 2014							
Female							
Ch17 GSM High	0.80 to 0.81	0.829	0.43/0.042	0.62	1.05	108694/108708	1.000
Ch16 GSM Med	0.57 to 0.57	0.594	0.40/0.028	0.47	0.71	108708/108708	1.000
Ch20 GSM Low	0.40 to 0.40	0.446	0.39/0.020	0.36	0.53	108626/108708	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	108708/108708	1.000
Male							
Ch06 GSM High	0.86 to 0.87	0.946	0.46/0.051	0.78	1.17	108550/108716	0.998
Ch05 GSM Med	0.60 to 0.61	0.639	0.43/0.032	0.53	0.78	108704/108716	1.000
Ch09 GSM Low	0.43 to 0.43	0.454	0.34/0.018	0.39	0.54	108708/108708	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	108716/108716	1.000
September 1 to 30, 2014							
Female							
Ch17 GSM High	0.81 to 0.81	0.823	0.45/0.043	0.53	1.12	104166/104176	1.000
Ch16 GSM Med	0.57 to 0.57	0.587	0.40/0.028	0.44	0.78	104168/104176	1.000
Ch20 GSM Low	0.40 to 0.40	0.435	0.33/0.017	0.37	0.56	104156/104176	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	104176/104176	1.000
Male							
Ch06 GSM High	0.86 to 0.86	0.917	0.52/0.056	0.53	1.24	104040/104176	0.999
Ch05 GSM Med	0.60 to 0.60	0.607	0.60/0.044	0.48	0.76	104174/104176	1.000
Ch09 GSM Low	0.43 to 0.43	0.435	0.34/0.018	0.33	0.56	104170/104176	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	104176/104176	1.000
August 8, 2012 to September 30, 2014							
Female							
Ch17 GSM High	0.51 to 0.81	0.774	0.49/0.045	0.42	1.12	2799438/2800508	1.000
Ch16 GSM Med	0.36 to 0.57	0.547	0.42/0.027	0.29	0.78	2800262/2800510	1.000
Ch20 GSM Low	0.26 to 0.40	0.403	0.41/0.020	0.21	0.56	2799794/2800388	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	2800510/2800510	1.000
Male							
Ch06 GSM High	0.51 to 0.87	0.883	0.49/0.051	0.29	1.34	2796716/2800838	0.999
Ch05 GSM Med	0.36 to 0.61	0.556	0.53/0.035	0.24	0.85	2800538/2800842	1.000
Ch09 GSM Low	0.26 to 0.44	0.449	0.42/0.022	0.21	0.60	2799272/2800582	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	2800844/2800844	1.000

Ch = chamber (e.g., Ch17 = Chamber 17).

Table I-5. Summary of CDMA-modulated Cell Phone RFR Exposure Data in Rats – SAR

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
August 8 to 31, 2012								
Male								
Ch07 IS95 High	237.9 to 358.4	6.00	5.97	0.17/0.04	2.109	8.613	42889/42893	1.000
Ch08 IS95 Med	238.8 to 354.1	3.00	2.99	0.17/0.04	1.077	4.524	42889/42893	1.000
Ch10 IS95 Low	239.3 to 361.1	1.50	1.50	0.17/0.04	0.593	2.223	42889/42893	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	42893/42893	1.000
September 1 to 30, 2012								
Female								
Ch19 IS95 High	55.9 to 303.9	6.00	5.99	0.19/0.04	4.373	9.406	52959/52959	1.000
Ch18 IS95 Med	59.6 to 303.9	3.00	2.99	0.19/0.05	2.018	4.860	52956/52959	1.000
Ch21 IS95 Low	59.3 to 303.9	1.50	1.50	0.20/0.05	1.103	2.524	52958/52959	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52959/52959	1.000
Male								
Ch07 IS95 High	59.2 to 294.9	6.00	5.99	0.21/0.05	4.555	15.811	52958/52959	1.000
Ch08 IS95 Med	63.0 to 306.6	3.00	3.00	0.25/0.06	2.116	10.629	52923/52959	0.999
Ch10 IS95 Low	62.1 to 309.8	1.50	1.50	0.25/0.06	0.882	5.060	52930/52959	0.999
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52959/52959	1.000
October 1 to 31, 2012								
Female								
Ch19 IS95 High	114.0 to 197.6	6.00	5.98	0.17/0.04	4.850	8.028	55335/55335	1.000
Ch18 IS95 Med	120.7 to 206.2	3.00	2.99	0.16/0.04	2.437	4.124	55335/55335	1.000
Ch21 IS95 Low	119.7 to 205.3	1.50	1.49	0.18/0.04	1.089	2.099	55335/55335	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55339/55339	1.000
Male								
Ch07 IS95 High	132.0 to 301.0	6.00	5.96	0.18/0.04	4.878	7.243	55351/55351	1.000
Ch08 IS95 Med	141.5 to 312.6	3.00	2.98	0.19/0.04	2.297	4.094	55351/55351	1.000
Ch10 IS95 Low	140.4 to 313.4	1.50	1.49	0.19/0.05	1.118	1.985	55339/55339	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55351/55351	1.000
November 1 to 30, 2012								
Female								
Ch19 IS95 High	204.3 to 239.5	6.00	5.99	0.18/0.04	4.441	9.544	53852/53853	1.000
Ch18 IS95 Med	211.8 to 248.7	3.00	2.98	0.15/0.04	2.556	3.879	53853/53853	1.000
Ch21 IS95 Low	210.9 to 249.2	1.50	1.49	0.16/0.04	1.205	2.100	53853/53853	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53853/53853	1.000
Male								
Ch07 IS95 High	310.9 to 383.3	6.00	5.99	0.17/0.04	5.166	7.287	53853/53853	1.000
Ch08 IS95 Med	323.1 to 396.1	3.00	2.99	0.17/0.04	2.432	4.100	53853/53853	1.000
Ch10 IS95 Low	324.6 to 399.7	1.50	1.49	0.17/0.04	1.251	1.926	53853/53853	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53853/53853	1.000
December 1 to 31, 2012								
Female								
Ch19 IS95 High	239.5 to 251.0	6.00	5.96	0.16/0.04	4.865	7.908	55608/55608	1.000
Ch18 IS95 Med	248.7 to 262.3	3.00	2.98	0.15/0.04	2.463	3.959	55608/55608	1.000
Ch21 IS95 Low	249.2 to 261.3	1.50	1.49	0.17/0.04	1.129	2.178	55608/55608	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55608/55608	1.000
Male								
Ch07 IS95 High	383.3 to 412.5	6.00	5.99	0.18/0.04	5.000	7.717	55608/55608	1.000
Ch08 IS95 Med	396.1 to 426.2	3.00	2.99	0.16/0.04	2.506	3.711	55608/55608	1.000
Ch10 IS95 Low	399.7 to 428.6	1.50	1.50	0.17/0.04	1.228	1.934	55608/55608	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55608/55608	1.000
January 1 to 31, 2013								
Female								
Ch19 IS95 High	250.9 to 270.7	6.00	5.98	0.16/0.04	4.937	7.523	55617/55617	1.000
Ch18 IS95 Med	262.3 to 280.6	3.00	3.00	0.28/0.07	1.426	7.073	55545/55617	0.999
Ch21 IS95 Low	261.3 to 277.7	1.50	1.49	0.17/0.04	1.188	2.066	55617/55617	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55619/55619	1.000
Male								
Ch07 IS95 High	412.5 to 452.2	6.00	6.01	0.18/0.04	4.995	7.462	55619/55619	1.000
Ch08 IS95 Med	426.2 to 463.0	3.00	2.98	0.16/0.04	2.592	3.759	55619/55619	1.000
Ch10 IS95 Low	428.6 to 462.1	1.50	1.49	0.17/0.04	1.279	1.862	55619/55619	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55619/55619	1.000
February 1 to 28, 2013								
Female								
Ch19 IS95 High	270.7 to 279.9	6.00	6.00	0.16/0.04	5.200	7.619	50082/50082	1.000
Ch18 IS95 Med	280.6 to 290.1	3.00	2.98	0.14/0.03	2.609	3.700	50082/50082	1.000
Ch21 IS95 Low	277.7 to 287.3	1.50	1.49	0.17/0.04	1.151	2.119	50082/50082	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	50082/50082	1.000
Male								
Ch07 IS95 High	452.2 to 472.2	6.00	5.98	0.18/0.04	5.098	7.180	50082/50082	1.000
Ch08 IS95 Med	463.0 to 491.2	3.00	2.99	0.17/0.04	2.585	3.770	50082/50082	1.000
Ch10 IS95 Low	462.1 to 488.9	1.50	1.49	0.17/0.04	1.253	1.964	50082/50082	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	50082/50082	1.000
March 1 to 31, 2013								
Female								
Ch19 IS95 High	279.9 to 288.7	6.00	5.98	0.16/0.04	4.893	7.540	55704/55704	1.000
Ch18 IS95 Med	290.1 to 300.4	3.00	2.99	0.15/0.03	2.491	3.827	55704/55704	1.000
Ch21 IS95 Low	287.3 to 299.3	1.50	1.48	0.17/0.04	1.183	2.207	55704/55704	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55704/55704	1.000
Male								
Ch07 IS95 High	472.2 to 495.2	6.00	6.00	0.18/0.04	5.039	7.261	55704/55704	1.000
Ch08 IS95 Med	491.2 to 512.3	3.00	3.00	0.17/0.04	2.563	3.628	55704/55704	1.000
Ch10 IS95 Low	488.9 to 510.6	1.50	1.50	0.17/0.04	1.257	1.872	55704/55704	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55704/55704	1.000
April 1 to 30, 2013								
Female								
Ch19 IS95 High	288.7 to 299.5	6.00	5.95	0.16/0.04	4.951	8.160	53719/53719	1.000
Ch18 IS95 Med	300.4 to 310.6	3.00	2.98	0.15/0.03	2.534	3.787	53719/53719	1.000
Ch21 IS95 Low	299.3 to 308.0	1.50	1.49	0.18/0.04	1.154	2.118	53719/53719	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53719/53719	1.000
Male								
Ch07 IS95 High	495.2 to 511.4	6.00	6.01	0.21/0.05	4.216	9.118	53721/53721	1.000
Ch08 IS95 Med	512.3 to 534.0	3.00	2.99	0.17/0.04	2.515	3.740	53721/53721	1.000
Ch10 IS95 Low	510.6 to 530.6	1.50	1.50	0.17/0.04	1.260	1.985	53719/53719	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53722/53722	1.000
May 1 to 31, 2013								
Female								
Ch19 IS95 High	299.5 to 308.8	6.00	5.98	0.16/0.04	4.581	7.657	52762/52762	1.000
Ch18 IS95 Med	310.6 to 320.6	3.00	3.00	0.15/0.03	2.318	3.891	52762/52762	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch21 IS95 Low	308.0 to 315.8	1.50	1.48	0.18/0.04	1.117	2.169	52762/52762	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52762/52762	1.000
Male								
Ch07 IS95 High	511.4 to 530.9	6.00	5.99	0.19/0.04	1.318	7.206	52788/52789	1.000
Ch08 IS95 Med	534.0 to 552.1	3.00	2.99	0.17/0.04	2.358	3.621	52788/52788	1.000
Ch10 IS95 Low	530.6 to 550.3	1.50	1.49	0.18/0.04	1.073	1.934	52762/52762	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52790/52790	1.000
June 1 to 30, 2013								
Female								
Ch19 IS95 High	308.8 to 316.4	6.00	5.97	0.16/0.04	5.110	7.720	53537/53537	1.000
Ch18 IS95 Med	320.6 to 332.1	3.00	3.00	0.15/0.04	2.543	4.014	53537/53537	1.000
Ch21 IS95 Low	315.8 to 328.3	1.50	1.50	0.19/0.05	1.215	2.164	53537/53537	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53537/53537	1.000
Male								
Ch07 IS95 High	530.9 to 547.2	6.00	5.93	0.19/0.04	4.899	7.410	53542/53542	1.000
Ch08 IS95 Med	552.1 to 569.1	3.00	2.95	0.27/0.06	1.304	3.920	53286/53542	0.995
Ch10 IS95 Low	550.3 to 566.3	1.50	1.48	0.17/0.04	1.274	1.860	53537/53537	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53544/53544	1.000
July 1 to 31, 2013								
Female								
Ch19 IS95 High	316.4 to 332.3	6.00	6.02	0.16/0.04	5.171	8.001	55527/55527	1.000
Ch18 IS95 Med	332.1 to 351.4	3.00	2.98	0.14/0.03	2.558	3.934	55527/55527	1.000
Ch21 IS95 Low	328.3 to 346.1	1.50	1.49	0.18/0.04	1.202	2.127	55527/55527	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55527/55527	1.000
Male								
Ch07 IS95 High	547.2 to 577.0	6.00	5.93	0.18/0.04	4.797	7.368	55528/55528	1.000
Ch08 IS95 Med	569.1 to 599.6	3.00	2.97	0.17/0.04	2.531	3.655	55528/55528	1.000
Ch10 IS95 Low	566.3 to 597.9	1.50	1.49	0.18/0.04	1.246	1.938	55527/55527	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55531/55531	1.000
August 1 to 31, 2013								
Female								
Ch19 IS95 High	332.3 to 342.8	6.00	5.99	0.16/0.04	5.082	7.893	55952/55952	1.000
Ch18 IS95 Med	351.4 to 360.3	3.00	2.97	0.15/0.03	2.477	3.781	55952/55952	1.000
Ch21 IS95 Low	346.1 to 356.6	1.50	1.48	0.18/0.04	1.168	2.076	55952/55952	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55952/55952	1.000
Male								
Ch07 IS95 High	577.0 to 590.3	6.00	5.98	0.19/0.04	5.067	7.207	55952/55952	1.000
Ch08 IS95 Med	599.6 to 613.4	3.00	2.99	0.18/0.04	2.116	4.677	55952/55952	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch10 IS95 Low	597.9 to 610.1	1.50	1.50	0.18/0.04	1.264	1.981	55952/55952	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55955/55955	1.000
September 1 to 30, 2013								
Female								
Ch19 IS95 High	342.8 to 349.6	6.00	5.97	0.17/0.04	5.099	7.749	53696/53696	1.000
Ch18 IS95 Med	360.3 to 366.3	3.00	2.99	0.15/0.03	2.569	3.855	53696/53696	1.000
Ch21 IS95 Low	356.6 to 363.7	1.50	1.48	0.18/0.04	1.149	2.224	53696/53696	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53696/53696	1.000
Male								
Ch07 IS95 High	590.3 to 594.1	6.00	5.98	0.20/0.05	3.989	7.867	53696/53696	1.000
Ch08 IS95 Med	613.4 to 623.8	3.00	2.99	0.16/0.04	2.588	3.626	53696/53696	1.000
Ch10 IS95 Low	610.1 to 620.5	1.50	1.50	0.18/0.04	1.237	1.814	53696/53696	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53703/53703	1.000
October 1 to 31, 2013								
Female								
Ch19 IS95 High	349.6 to 358.7	6.00	5.96	0.18/0.04	3.243	7.477	56746/56747	1.000
Ch18 IS95 Med	366.3 to 377.2	3.00	2.99	0.19/0.04	1.170	3.898	56706/56748	0.999
Ch21 IS95 Low	363.7 to 375.4	1.50	1.49	0.19/0.04	0.762	2.325	56746/56747	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	56748/56748	1.000
Male								
Ch07 IS95 High	594.1 to 603.9	6.00	5.95	0.19/0.05	3.609	8.358	56747/56748	1.000
Ch08 IS95 Med	623.8 to 632.5	3.00	2.97	0.18/0.04	1.622	3.648	56747/56748	1.000
Ch10 IS95 Low	620.5 to 631.5	1.50	1.49	0.19/0.04	0.769	1.850	56747/56748	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	56807/56807	1.000
November 1 to 30, 2013								
Female								
Ch19 IS95 High	358.7 to 366.5	6.00	5.96	0.21/0.05	3.622	7.586	55322/55323	1.000
Ch18 IS95 Med	377.2 to 385.8	3.00	3.00	0.19/0.05	0.910	7.500	55319/55323	1.000
Ch21 IS95 Low	375.4 to 384.9	1.50	1.49	0.23/0.05	0.811	2.262	55322/55323	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55323/55323	1.000
Male								
Ch07 IS95 High	603.9 to 611.0	6.00	5.93	0.21/0.05	2.254	7.558	55329/55330	1.000
Ch08 IS95 Med	632.5 to 640.5	3.00	2.99	0.23/0.05	2.092	4.959	55326/55329	1.000
Ch10 IS95 Low	631.5 to 641.9	1.50	1.50	0.22/0.05	1.059	2.127	55329/55329	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55332/55332	1.000
December 1 to 31, 2013								
Female								
Ch19 IS95 High	366.5 to 374.5	6.00	6.00	0.19/0.05	4.178	7.659	53057/53057	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch18 IS95 Med	385.8 to 393.5	3.00	2.97	0.16/0.04	2.156	3.649	53099/53099	1.000
Ch21 IS95 Low	384.9 to 395.7	1.50	1.49	0.19/0.05	0.995	2.069	53057/53057	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53099/53099	1.000
Male								
Ch07 IS95 High	611.0 to 614.6	6.00	5.95	0.20/0.05	0.989	7.282	53106/53108	1.000
Ch08 IS95 Med	640.5 to 646.3	3.00	2.96	0.22/0.05	1.270	4.996	53020/53106	0.998
Ch10 IS95 Low	641.9 to 645.9	1.50	1.48	0.20/0.05	1.015	1.773	53099/53099	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53111/53111	1.000
January 1 to 31, 2014								
Female								
Ch19 IS95 High	374.5 to 386.3	6.00	5.98	0.17/0.04	4.565	8.091	55621/55621	1.000
Ch18 IS95 Med	393.5 to 405.6	3.00	2.98	0.14/0.03	2.430	3.687	55626/55626	1.000
Ch21 IS95 Low	395.7 to 408.4	1.50	1.48	0.16/0.04	1.107	2.088	55621/55621	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55626/55626	1.000
Male								
Ch07 IS95 High	614.6 to 625.5	6.00	5.91	0.20/0.05	4.782	7.279	55627/55627	1.000
Ch08 IS95 Med	646.3 to 661.2	3.00	2.97	0.17/0.04	2.235	3.510	55627/55627	1.000
Ch10 IS95 Low	645.9 to 658.9	1.50	1.49	0.18/0.04	1.169	1.845	55627/55627	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55629/55629	1.000
February 1 to 28, 2014								
Female								
Ch19 IS95 High	386.3 to 401.1	6.00	5.95	0.17/0.04	4.403	7.540	51974/51974	1.000
Ch18 IS95 Med	405.6 to 417.2	3.00	2.99	0.15/0.04	2.379	3.683	51974/51974	1.000
Ch21 IS95 Low	408.4 to 423.0	1.50	1.49	0.18/0.04	1.006	2.073	51974/51974	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	51974/51974	1.000
Male								
Ch07 IS95 High	625.5 to 637.7	6.00	5.92	0.19/0.04	4.726	7.209	51974/51974	1.000
Ch08 IS95 Med	661.2 to 671.2	3.00	2.98	0.16/0.04	2.288	3.559	51974/51974	1.000
Ch10 IS95 Low	658.9 to 672.1	1.50	1.50	0.18/0.04	1.137	1.770	51974/51974	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	51980/51980	1.000
March 1 to 31, 2014								
Female								
Ch19 IS95 High	401.1 to 415.9	6.00	5.99	0.16/0.04	5.177	8.398	55704/55704	1.000
Ch18 IS95 Med	417.2 to 426.8	3.00	2.99	0.13/0.03	2.635	3.825	55704/55704	1.000
Ch21 IS95 Low	423.0 to 433.7	1.50	1.48	0.16/0.04	1.217	2.018	55704/55704	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55704/55704	1.000
Male								
Ch07 IS95 High	637.7 to 640.9	6.00	5.90	0.19/0.05	4.851	7.304	55704/55704	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch08 IS95 Med	671.2 to 679.3	3.00	2.99	0.16/0.04	2.519	3.608	55704/55704	1.000
Ch10 IS95 Low	672.1 to 678.5	1.50	1.50	0.17/0.04	1.262	1.804	55704/55704	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55704/55704	1.000
April 1 to 30, 2014								
Female								
Ch19 IS95 High	415.9 to 430.6	6.00	5.95	0.16/0.04	4.801	7.534	53643/53643	1.000
Ch18 IS95 Med	426.8 to 440.1	3.00	2.98	0.13/0.03	0.964	3.533	53643/53644	1.000
Ch21 IS95 Low	433.7 to 439.9	1.50	1.48	0.16/0.04	1.132	2.033	53643/53643	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53644/53644	1.000
Male								
Ch07 IS95 High	640.9 to 646.7	6.00	5.88	0.19/0.04	1.418	7.181	53646/53647	1.000
Ch08 IS95 Med	679.3 to 681.0	3.00	3.00	0.16/0.04	2.308	3.621	53646/53646	1.000
Ch10 IS95 Low	674.4 to 678.5	1.50	1.50	0.17/0.04	0.894	1.966	53643/53644	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53649/53649	1.000
May 1 to 31, 2014								
Female								
Ch19 IS95 High	430.6 to 435.9	6.00	5.92	0.15/0.04	4.846	7.433	55602/55602	1.000
Ch18 IS95 Med	440.1 to 447.7	3.00	2.98	0.13/0.03	2.611	3.979	55604/55604	1.000
Ch21 IS95 Low	439.9 to 448.6	1.50	1.49	0.17/0.04	1.185	2.158	55602/55602	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55604/55604	1.000
Male								
Ch07 IS95 High	640.5 to 644.4	6.00	5.91	0.17/0.04	4.656	7.371	55604/55604	1.000
Ch08 IS95 Med	681.0 to 682.7	3.00	3.01	0.16/0.04	2.370	3.513	55604/55604	1.000
Ch10 IS95 Low	674.4 to 676.1	1.50	1.49	0.17/0.04	1.210	1.822	55604/55604	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55604/55604	1.000
June 1 to 30, 2014								
Female								
Ch19 IS95 High	435.9 to 447.6	6.00	5.92	0.21/0.05	3.191	10.116	53730/53767	0.999
Ch18 IS95 Med	447.7 to 462.8	3.00	2.98	0.14/0.03	2.589	3.773	53770/53771	1.000
Ch21 IS95 Low	448.6 to 454.5	1.50	1.49	0.16/0.04	1.221	2.005	53767/53767	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53771/53771	1.000
Male								
Ch07 IS95 High	632.7 to 640.5	6.00	5.96	0.18/0.04	4.891	7.698	53773/53773	1.000
Ch08 IS95 Med	682.7 to 691.1	3.00	3.00	0.16/0.04	2.562	3.605	53773/53773	1.000
Ch10 IS95 Low	669.2 to 676.1	1.50	1.50	0.17/0.04	1.255	1.785	53771/53771	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	53774/53774	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
July 1 to 31, 2014								
Female								
Ch19 IS95 High	450.1 to 455.1	6.00	5.99	0.16/0.04	5.236	7.748	55601/55601	1.000
Ch18 IS95 Med	466.7 to 473.2	3.00	2.97	0.12/0.03	2.635	3.904	55601/55601	1.000
Ch21 IS95 Low	454.0 to 456.9	1.50	1.49	0.16/0.04	1.221	3.108	55598/55601	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55601/55601	1.000
Male								
Ch07 IS95 High	633.3 to 638.9	6.00	5.97	0.17/0.04	5.044	7.429	55601/55601	1.000
Ch08 IS95 Med	675.8 to 689.3	3.00	3.01	0.15/0.04	2.618	3.671	55601/55601	1.000
Ch10 IS95 Low	661.6 to 671.6	1.50	1.49	0.16/0.04	1.273	1.786	55601/55601	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	55603/55603	1.000
August 1 to 31, 2014								
Female								
Ch19 IS95 High	455.1 to 457.3	6.00	5.99	0.17/0.04	4.318	7.906	54354/54354	1.000
Ch18 IS95 Med	470.8 to 473.2	3.00	2.97	0.13/0.03	2.314	3.894	54354/54354	1.000
Ch21 IS95 Low	451.0 to 456.9	1.50	1.49	0.18/0.04	1.046	2.193	54354/54354	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	54354/54354	1.000
Male								
Ch07 IS95 High	622.8 to 633.3	6.00	5.94	0.18/0.04	4.629	7.561	54354/54354	1.000
Ch08 IS95 Med	659.4 to 675.8	3.00	2.98	0.17/0.04	2.209	3.803	54354/54354	1.000
Ch10 IS95 Low	649.7 to 661.6	1.50	1.49	0.16/0.04	1.090	1.855	54354/54354	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	54358/54358	1.000
September 1 to 30, 2014								
Female								
Ch19 IS95 High	454.2 to 455.7	6.00	5.99	0.18/0.04	4.062	10.317	52080/52088	1.000
Ch18 IS95 Med	467.7 to 471.7	3.00	2.97	0.14/0.03	2.195	4.559	52088/52088	1.000
Ch21 IS95 Low	445.6 to 453.5	1.50	1.49	0.16/0.04	0.880	2.711	52086/52088	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52088/52088	1.000
Male								
Ch07 IS95 High	620.1 to 622.8	6.00	5.93	0.16/0.04	4.185	9.677	52087/52088	1.000
Ch08 IS95 Med	648.3 to 659.4	3.00	2.97	0.16/0.04	2.066	4.741	52088/52088	1.000
Ch10 IS95 Low	661.1 to 665.3	1.50	1.48	0.15/0.03	1.038	2.242	52088/52088	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	52088/52088	1.000
August 8, 2012 to September 30, 2014								
Female								
Ch19 IS95 High	55.9 to 457.3	6.00	5.97	0.17/0.04	2.650	10.317	1400143/1400194	1.000
Ch18 IS95 Med	59.6 to 473.2	3.00	2.98	0.16/0.04	0.910	7.500	1400123/1400249	1.000
Ch21 IS95 Low	59.3 to 456.9	1.50	1.49	0.18/0.04	0.653	3.108	1400183/1400194	1.000

Chamber	Weight Range [g]	Target [W/kg]	Mean [W/kg]	Stdev [dB]/[W/kg]	Min [W/kg]	Max [W/kg]	In Range/Total	Ratio
Ch15 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	1400255/1400255	1.000
Male								
Ch07 IS95 High	59.2 to 646.7	6.00	5.96	0.19/0.04	0.989	15.811	1400312/1400324	1.000
Ch08 IS95 Med	63.0 to 691.1	3.00	2.98	0.18/0.04	1.077	10.629	1399933/1400319	1.000
Ch10 IS95 Low	62.1 to 678.5	1.50	1.49	0.18/0.04	0.593	5.060	1400227/1400262	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	0.00	–/0.00	0.000	0.000	1400422/1400422	1.000

Ch = chamber (e.g., Ch19 = Chamber 19).

Table I-6. Summary of CDMA-modulated Cell Phone RFR Exposure Data in Rats – Chamber Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
August 8 to 31, 2012							
Male							
Ch07 IS95 High	246.10 to 283.80	262.13	0.17/5.16	149.00	313.07	85778/85786	1.000
Ch08 IS95 Med	174.00 to 200.70	185.32	0.17/3.59	106.47	226.74	85778/85786	1.000
Ch10 IS95 Low	123.10 to 143.40	131.39	0.17/2.54	78.99	159.09	85778/85786	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	85786/85786	1.000
September 1 -30, 2012							
Female							
Ch19 IS95 High	192.60 to 269.00	237.99	0.18/4.99	178.91	298.61	105918/105918	1.000
Ch18 IS95 Med	135.90 to 190.20	167.15	0.19/3.68	125.16	210.93	105912/105918	1.000
Ch21 IS95 Low	096.30 to 134.50	118.94	0.19/2.66	87.59	154.09	105916/105918	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105918/105918	1.000
Male							
Ch07 IS95 High	192.20 to 266.50	235.04	0.21/5.62	177.28	375.72	105916/105918	1.000
Ch08 IS95 Med	136.20 to 190.20	165.57	0.23/4.50	127.08	284.85	105854/105918	0.999
Ch10 IS95 Low	096.30 to 134.50	116.96	0.23/3.16	82.06	196.54	105864/105918	0.999
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105918/105918	1.000
October 1 to 31, 2012							
Female							
Ch19 IS95 High	193.80 to 228.70	213.07	0.17/4.14	177.92	257.59	110670/110670	1.000
Ch18 IS95 Med	139.30 to 164.60	152.45	0.16/2.82	126.67	185.53	110670/110670	1.000
Ch21 IS95 Low	096.90 to 116.40	107.57	0.18/2.21	85.78	135.10	110670/110670	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110678/110678	1.000
Male							
Ch07 IS95 High	201.30 to 269.00	238.68	0.19/5.16	184.54	291.28	110702/110702	1.000
Ch08 IS95 Med	145.90 to 192.20	171.66	0.19/3.79	127.64	224.82	110702/110702	1.000
Ch10 IS95 Low	103.10 to 135.90	121.30	0.20/2.77	91.15	149.99	110678/110678	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110702/110702	1.000
November 1 to 30, 2012							
Female							
Ch19 IS95 High	232.80 to 246.10	240.74	0.18/4.97	207.64	304.40	107704/107706	1.000
Ch18 IS95 Med	167.60 to 177.30	172.96	0.15/3.08	157.53	202.06	107706/107706	1.000
Ch21 IS95 Low	118.50 to 125.40	121.96	0.16/2.33	106.52	148.67	107706/107706	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107706/107706	1.000
Male							
Ch07 IS95 High	271.80 to 292.20	282.34	0.17/5.61	252.88	319.04	107706/107706	1.000
Ch08 IS95 Med	194.20 to 208.20	201.56	0.17/4.01	176.59	233.27	107706/107706	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch10 IS95 Low	137.40 to 147.30	142.68	0.17/2.76	129.54	163.57	107706/107706	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107706/107706	1.000
December 1 to 31, 2012							
Female							
Ch19 IS95 High	246.10 to 254.90	252.54	0.16/4.78	226.29	291.17	111216/111216	1.000
Ch18 IS95 Med	177.30 to 182.70	180.85	0.15/3.18	163.62	209.74	111216/111216	1.000
Ch21 IS95 Low	125.40 to 129.20	127.68	0.17/2.52	110.99	155.56	111216/111216	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111216/111216	1.000
Male							
Ch07 IS95 High	292.20 to 298.00	296.07	0.18/6.03	268.82	336.88	111216/111216	1.000
Ch08 IS95 Med	208.20 to 211.70	210.45	0.16/3.97	191.98	235.34	111216/111216	1.000
Ch10 IS95 Low	147.30 to 149.70	148.95	0.17/2.90	134.40	168.66	111216/111216	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111216/111216	1.000
January 1 to 31, 2013							
Female							
Ch19 IS95 High	254.90 to 261.40	258.29	0.16/4.89	236.85	292.38	111234/111234	1.000
Ch18 IS95 Med	182.70 to 186.70	184.88	0.25/5.50	125.89	280.35	111090/111234	0.999
Ch21 IS95 Low	129.20 to 130.70	129.67	0.17/2.50	115.01	151.52	111234/111234	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111238/111238	1.000
Male							
Ch07 IS95 High	298.00 to 302.10	299.87	0.18/6.32	271.01	336.24	111238/111238	1.000
Ch08 IS95 Med	211.70 to 214.00	212.58	0.16/3.99	198.00	240.49	111238/111238	1.000
Ch10 IS95 Low	149.70 to 151.40	150.39	0.18/3.07	138.67	168.35	111238/111238	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111238/111238	1.000
February 1 to 28, 2013							
Female							
Ch19 IS95 High	261.40 to 261.40	261.07	0.16/4.80	243.09	294.24	100164/100164	1.000
Ch18 IS95 Med	186.70 to 188.40	187.39	0.14/3.14	174.17	209.88	100164/100164	1.000
Ch21 IS95 Low	130.70 to 132.00	131.14	0.17/2.53	115.67	155.65	100164/100164	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	100164/100164	1.000
Male							
Ch07 IS95 High	302.10 to 303.40	302.43	0.18/6.36	278.47	332.36	100164/100164	1.000
Ch08 IS95 Med	214.00 to 215.50	214.43	0.17/4.19	199.44	240.83	100164/100164	1.000
Ch10 IS95 Low	151.40 to 152.00	151.48	0.17/2.95	138.83	173.82	100164/100164	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	100164/100164	1.000
March 1 to 31, 2013							
Female							
Ch19 IS95 High	261.40 to 264.00	262.72	0.16/4.86	235.81	294.38	111408/111408	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch18 IS95 Med	188.40 to 190.20	189.26	0.15/3.30	173.24	214.74	111408/111408	1.000
Ch21 IS95 Low	132.00 to 133.20	132.32	0.17/2.63	117.29	162.11	111408/111408	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
Male							
Ch07 IS95 High	303.40 to 304.70	303.64	0.18/6.44	278.43	334.23	111408/111408	1.000
Ch08 IS95 Med	215.50 to 216.80	215.84	0.17/4.25	198.63	238.10	111408/111408	1.000
Ch10 IS95 Low	152.00 to 153.30	152.54	0.18/3.12	139.06	171.02	111408/111408	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
April 1 to 30, 2013							
Female							
Ch19 IS95 High	264.00 to 266.50	265.43	0.16/5.03	239.94	308.04	107438/107438	1.000
Ch18 IS95 Med	190.20 to 192.20	191.35	0.15/3.29	176.86	215.07	107438/107438	1.000
Ch21 IS95 Low	133.20 to 134.50	133.62	0.18/2.84	117.93	159.75	107438/107438	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107438/107438	1.000
Male							
Ch07 IS95 High	304.70 to 306.60	305.73	0.21/7.39	256.68	377.45	107442/107442	1.000
Ch08 IS95 Med	216.80 to 218.30	217.43	0.17/4.28	198.25	242.05	107442/107442	1.000
Ch10 IS95 Low	153.30 to 154.40	153.76	0.17/3.11	141.43	176.11	107438/107438	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107444/107444	1.000
May 1 to 31, 2013							
Female							
Ch19 IS95 High	266.50 to 269.00	268.19	0.16/5.04	234.94	303.73	105524/105524	1.000
Ch18 IS95 Med	192.20 to 194.20	193.45	0.15/3.32	170.21	220.53	105524/105524	1.000
Ch21 IS95 Low	134.50 to 135.90	135.05	0.18/2.89	117.44	161.67	105524/105524	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105524/105524	1.000
Male							
Ch07 IS95 High	306.60 to 308.80	307.80	0.19/6.77	144.64	338.21	105576/105578	1.000
Ch08 IS95 Med	218.30 to 220.10	219.28	0.17/4.41	195.00	241.33	105576/105576	1.000
Ch10 IS95 Low	154.40 to 155.60	155.02	0.18/3.21	131.56	176.60	105524/105524	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	105580/105580	1.000
June 1 to 30, 2013							
Female							
Ch19 IS95 High	269.00 to 271.80	271.14	0.16/5.18	251.16	308.73	107074/107074	1.000
Ch18 IS95 Med	194.20 to 196.40	195.78	0.15/3.44	180.58	226.84	107074/107074	1.000
Ch21 IS95 Low	135.90 to 137.40	136.65	0.19/3.09	123.25	164.46	107074/107074	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107074/107074	1.000
Male							
Ch07 IS95 High	308.80 to 310.00	309.00	0.19/6.87	281.09	345.71	107084/107084	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch08 IS95 Med	220.10 to 221.00	219.62	0.30/7.81	145.05	251.44	106572/107084	0.995
Ch10 IS95 Low	155.60 to 156.30	155.84	0.18/3.19	144.54	174.62	107074/107074	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107088/107088	1.000
July 1 to 31, 2013							
Female							
Ch19 IS95 High	271.80 to 277.70	274.50	0.16/5.16	254.24	316.25	111054/111054	1.000
Ch18 IS95 Med	196.40 to 200.70	198.18	0.15/3.36	184.01	227.52	111054/111054	1.000
Ch21 IS95 Low	137.40 to 140.40	138.38	0.18/2.96	124.13	165.13	111054/111054	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111054/111054	1.000
Male							
Ch07 IS95 High	310.00 to 314.00	311.52	0.19/6.78	280.42	347.54	111056/111056	1.000
Ch08 IS95 Med	221.00 to 223.90	222.27	0.17/4.39	205.37	246.82	111056/111056	1.000
Ch10 IS95 Low	156.30 to 158.40	157.32	0.18/3.25	144.08	179.70	111054/111054	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111062/111062	1.000
August 1 to 31, 2013							
Female							
Ch19 IS95 High	277.70 to 280.80	277.82	0.16/5.30	255.25	318.10	111904/111904	1.000
Ch18 IS95 Med	200.70 to 202.80	200.65	0.15/3.46	182.96	226.03	111904/111904	1.000
Ch21 IS95 Low	140.40 to 141.90	140.10	0.18/2.95	123.98	165.26	111904/111904	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111904/111904	1.000
Male							
Ch07 IS95 High	314.00 to 316.70	313.45	0.19/6.88	288.22	345.98	111904/111904	1.000
Ch08 IS95 Med	223.90 to 225.90	223.44	0.18/4.66	189.39	281.55	111904/111904	1.000
Ch10 IS95 Low	158.40 to 159.70	158.24	0.18/3.30	145.14	183.22	111904/111904	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111910/111910	1.000
September 1 to 30, 2013							
Female							
Ch19 IS95 High	280.80 to 280.80	280.29	0.17/5.48	259.03	319.31	107392/107392	1.000
Ch18 IS95 Med	202.80 to 202.80	202.31	0.15/3.44	187.61	229.81	107392/107392	1.000
Ch21 IS95 Low	141.90 to 143.40	141.65	0.19/3.06	124.62	173.37	107392/107392	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107392/107392	1.000
Male							
Ch07 IS95 High	316.70 to 316.70	315.70	0.21/7.59	257.86	362.09	107392/107392	1.000
Ch08 IS95 Med	225.90 to 226.80	225.46	0.16/4.24	209.57	250.04	107392/107392	1.000
Ch10 IS95 Low	159.70 to 160.40	159.61	0.18/3.40	146.04	175.95	107392/107392	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107406/107406	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
October 1 to 31, 2013							
Female							
Ch19 IS95 High	280.80 to 283.80	281.41	0.18/5.86	206.58	313.65	113492/113494	1.000
Ch18 IS95 Med	202.80 to 204.80	203.01	0.20/4.82	126.61	231.08	113412/113496	0.999
Ch21 IS95 Low	143.40 to 144.80	143.35	0.19/3.19	102.19	178.48	113492/113494	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	113496/113496	1.000
Male							
Ch07 IS95 High	316.70 to 318.10	315.94	0.20/7.20	245.25	373.22	113494/113496	1.000
Ch08 IS95 Med	226.80 to 227.70	226.35	0.18/4.72	167.22	250.80	113494/113496	1.000
Ch10 IS95 Low	160.40 to 161.00	160.43	0.19/3.55	115.17	178.59	113494/113496	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	113614/113614	1.000
November 1 to 30, 2013							
Female							
Ch19 IS95 High	283.80 to 286.80	284.53	0.22/7.15	222.74	322.36	110644/110646	1.000
Ch18 IS95 Med	204.80 to 206.70	205.34	0.19/4.62	114.00	327.33	110638/110646	1.000
Ch21 IS95 Low	144.80 to 146.10	144.71	0.23/3.95	107.65	179.75	110644/110646	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110646/110646	1.000
Male							
Ch07 IS95 High	318.10 to 319.40	316.80	0.21/7.75	195.44	357.91	110658/110660	1.000
Ch08 IS95 Med	227.70 to 228.70	227.67	0.23/6.06	191.58	294.96	110652/110658	1.000
Ch10 IS95 Low	161.00 to 161.70	161.12	0.22/4.20	136.29	193.18	110658/110658	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	110664/110664	1.000
December 1 to 31, 2013							
Female							
Ch19 IS95 High	286.80 to 289.60	287.74	0.20/6.62	239.22	323.90	106114/106114	1.000
Ch18 IS95 Med	206.70 to 208.20	206.92	0.16/3.94	175.52	229.96	106198/106198	1.000
Ch21 IS95 Low	146.10 to 147.30	146.20	0.20/3.39	119.22	173.18	106114/106114	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	106198/106198	1.000
Male							
Ch07 IS95 High	319.40 to 319.40	317.36	0.21/7.68	129.45	351.31	106212/106216	1.000
Ch08 IS95 Med	228.70 to 228.70	227.86	0.24/6.43	149.29	296.06	106040/106212	0.998
Ch10 IS95 Low	161.70 to 161.70	161.31	0.20/3.83	133.47	176.37	106198/106198	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	106222/106222	1.000
January 1 to 31, 2014							
Female							
Ch19 IS95 High	289.60 to 292.20	290.54	0.17/5.74	255.37	335.23	111242/111242	1.000
Ch18 IS95 Med	208.20 to 209.60	208.51	0.14/3.48	188.48	232.86	111252/111252	1.000
Ch21 IS95 Low	147.30 to 148.20	147.24	0.17/2.87	126.66	175.22	111242/111242	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111252/111252	1.000
Male							
Ch07 IS95 High	319.40 to 320.80	318.05	0.20/7.53	284.68	354.25	111254/111254	1.000
Ch08 IS95 Med	228.70 to 230.60	229.35	0.17/4.50	198.04	248.14	111254/111254	1.000
Ch10 IS95 Low	161.70 to 162.40	161.88	0.18/3.41	143.19	179.92	111254/111254	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111258/111258	1.000
February 1 to 28, 2014							
Female							
Ch19 IS95 High	292.20 to 296.40	294.48	0.17/5.90	254.46	333.00	103948/103948	1.000
Ch18 IS95 Med	209.60 to 210.70	209.78	0.15/3.68	187.04	232.71	103948/103948	1.000
Ch21 IS95 Low	148.20 to 149.70	148.69	0.18/3.11	122.51	175.92	103948/103948	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	103948/103948	1.000
Male							
Ch07 IS95 High	320.80 to 322.10	319.38	0.19/7.15	285.45	352.56	103948/103948	1.000
Ch08 IS95 Med	230.60 to 231.50	230.65	0.16/4.37	202.15	252.10	103948/103948	1.000
Ch10 IS95 Low	162.40 to 163.70	163.15	0.19/3.52	142.50	177.01	103948/103948	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	103960/103960	1.000
March 1 to 31, 2014							
Female							
Ch19 IS95 High	296.40 to 298.00	296.72	0.16/5.55	275.93	351.43	111408/111408	1.000
Ch18 IS95 Med	210.70 to 211.70	210.79	0.13/3.27	197.92	238.93	111408/111408	1.000
Ch21 IS95 Low	149.70 to 150.20	149.49	0.16/2.76	134.93	173.56	111408/111408	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
Male							
Ch07 IS95 High	322.10 to 323.40	320.78	0.19/7.24	291.74	354.86	111408/111408	1.000
Ch08 IS95 Med	231.50 to 231.50	231.09	0.16/4.30	212.10	253.84	111408/111408	1.000
Ch10 IS95 Low	163.70 to 163.70	163.52	0.17/3.29	150.14	179.49	111408/111408	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111408/111408	1.000
April 1 to 30, 2014							
Female							
Ch19 IS95 High	298.00 to 300.40	299.05	0.16/5.69	269.69	337.86	107286/107286	1.000
Ch18 IS95 Med	211.70 to 213.10	212.13	0.14/3.33	120.89	231.36	107286/107288	1.000
Ch21 IS95 Low	150.20 to 150.20	149.60	0.16/2.80	130.96	175.51	107286/107286	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107288/107288	1.000
Male							
Ch07 IS95 High	323.40 to 323.40	321.14	0.19/7.22	157.72	354.94	107292/107294	1.000
Ch08 IS95 Med	231.50 to 232.50	231.60	0.16/4.24	203.00	254.30	107292/107292	1.000
Ch10 IS95 Low	163.70 to 163.70	163.39	0.17/3.27	126.33	187.37	107286/107288	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107298/107298	1.000
May 1 to 31, 2014							
Female							
Ch19 IS95 High	300.40 to 300.40	299.44	0.16/5.43	270.97	335.58	111204/111204	1.000
Ch18 IS95 Med	213.10 to 213.10	212.29	0.13/3.28	198.92	245.53	111208/111208	1.000
Ch21 IS95 Low	150.20 to 150.70	150.10	0.17/2.90	134.01	180.82	111204/111204	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111208/111208	1.000
Male							
Ch07 IS95 High	323.40 to 323.40	321.82	0.18/6.66	285.79	359.61	111208/111208	1.000
Ch08 IS95 Med	232.50 to 232.50	231.90	0.16/4.25	205.74	250.46	111208/111208	1.000
Ch10 IS95 Low	163.70 to 163.70	163.33	0.17/3.31	146.99	180.37	111208/111208	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111208/111208	1.000
June 1 to 30, 2014							
Female							
Ch19 IS95 High	300.40 to 301.30	299.48	0.21/7.42	219.87	391.50	107460/107534	0.999
Ch18 IS95 Med	213.10 to 214.00	213.08	0.15/3.59	26.52	239.11	107540/107542	1.000
Ch21 IS95 Low	150.70 to 151.00	150.40	0.16/2.76	136.04	174.27	107534/107534	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107542/107542	1.000
Male							
Ch07 IS95 High	322.10 to 323.40	320.57	0.18/6.62	290.40	364.33	107546/107546	1.000
Ch08 IS95 Med	232.50 to 233.40	232.22	0.16/4.18	213.90	253.72	107546/107546	1.000
Ch10 IS95 Low	163.00 to 163.70	163.46	0.17/3.26	149.73	178.52	107542/107542	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	107548/107548	1.000
July 1 to 31, 2014							
Female							
Ch19 IS95 High	302.10 to 302.10	301.20	0.16/5.68	281.66	342.63	111202/111202	1.000
Ch18 IS95 Med	214.00 to 214.50	213.60	0.12/3.09	201.33	245.08	111202/111202	1.000
Ch21 IS95 Low	151.00 to 151.00	150.44	0.16/2.86	136.02	217.00	111196/111202	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111202/111202	1.000
Male							
Ch07 IS95 High	322.10 to 322.10	320.71	0.17/6.37	294.89	357.89	111202/111202	1.000
Ch08 IS95 Med	231.50 to 232.50	231.74	0.15/4.11	216.21	256.04	111202/111202	1.000
Ch10 IS95 Low	163.00 to 163.70	163.06	0.16/3.07	150.77	178.56	111202/111202	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	111206/111206	1.000
August 1 to 31, 2014							
Female							
Ch19 IS95 High	302.10 to 302.10	301.12	0.17/6.05	255.77	346.10	108708/108708	1.000
Ch18 IS95 Med	214.50 to 214.50	213.73	0.14/3.37	188.69	244.76	108708/108708	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch21 IS95 Low	151.00 to 151.00	150.40	0.18/3.13	125.88	182.30	108708/108708	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	108708/108708	1.000
Male							
Ch07 IS95 High	320.80 to 322.10	319.89	0.18/6.73	282.50	361.05	108708/108708	1.000
Ch08 IS95 Med	229.60 to 231.50	230.28	0.17/4.59	198.60	258.77	108708/108708	1.000
Ch10 IS95 Low	161.70 to 163.00	162.21	0.17/3.17	138.29	181.98	108708/108708	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	108716/108716	1.000
September 1 to 30, 2014							
Female							
Ch19 IS95 High	302.10 to 302.10	301.19	0.17/6.11	248.08	395.37	104162/104176	1.000
Ch18 IS95 Med	214.00 to 214.50	213.67	0.14/3.50	183.75	264.84	104176/104176	1.000
Ch21 IS95 Low	150.70 to 151.00	150.24	0.17/2.89	115.49	202.67	104172/104176	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	104176/104176	1.000
Male							
Ch07 IS95 High	320.80 to 320.80	319.75	0.16/6.09	268.63	408.47	104174/104176	1.000
Ch08 IS95 Med	228.70 to 229.60	228.28	0.16/4.23	190.37	288.41	104174/104176	1.000
Ch10 IS95 Low	163.00 to 163.00	162.73	0.15/2.84	136.12	200.08	104176/104176	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	104176/104176	1.000
August 8, 2012 to September 30, 2014							
Female							
Ch19 IS95 High	192.60 to 302.10	275.55	0.17/5.49	167.01	395.37	2800288/2800388	1.000
Ch18 IS95 Med	135.90 to 214.50	197.17	0.16/3.62	26.52	327.33	2800246/2800498	1.000
Ch21 IS95 Low	096.30 to 151.00	138.76	0.18/2.87	82.90	217.00	2800366/2800388	1.000
Ch15 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	2800510/2800510	1.000
Male							
Ch07 IS95 High	192.20 to 323.40	304.76	0.19/6.72	129.45	408.47	2800624/2800648	1.000
Ch08 IS95 Med	136.20 to 233.40	218.09	0.18/4.65	106.47	296.06	2799872/2800638	1.000
Ch10 IS95 Low	096.30 to 163.70	154.21	0.18/3.24	78.99	200.08	2800458/2800524	1.000
Ch04 Sham Control	000.00 to 000.00	0.00	–/0.00	0.00	0.00	2800844/2800844	1.000

Ch = chamber (e.g., Ch19 = Chamber 19).

Table I-7. Summary of CDMA-modulated Cell Phone RFR Exposure Data in Rats – E-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
August 8 to 31, 2012							
Male							
Ch07 IS95 High	246.1 to 283.8	249.97	0.24/6.98	136.7	310.0	85778/85786	1.000
Ch08 IS95 Med	174.0 to 200.7	177.30	0.22/4.61	102.5	212.3	85778/85786	1.000
Ch10 IS95 Low	123.1 to 143.4	124.27	0.21/3.05	77.3	151.3	85778/85786	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	85786/85786	1.000
September 1 to 30, 2012							
Female							
Ch19 IS95 High	192.6 to 269.0	239.48	0.24/6.85	181.6	297.4	105910/105918	1.000
Ch18 IS95 Med	135.9 to 190.2	157.66	0.26/4.84	114.7	202.6	105916/105918	1.000
Ch21 IS95 Low	96.3 to 134.5	115.70	0.45/6.14	80.0	157.2	105916/105918	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105918/105918	1.000
Male							
Ch07 IS95 High	192.2 to 266.5	225.11	0.25/6.52	167.5	349.5	105916/105918	1.000
Ch08 IS95 Med	136.2 to 190.2	159.21	0.33/6.15	123.1	288.1	105874/105918	1.000
Ch10 IS95 Low	96.3 to 134.5	113.59	0.28/3.67	81.2	192.6	105910/105918	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105918/105918	1.000
October 1 to 31, 2012							
Female							
Ch19 IS95 High	193.8 to 228.7	216.72	0.28/7.06	179.0	264.8	110670/110670	1.000
Ch18 IS95 Med	139.3 to 164.6	140.84	0.24/3.88	121.3	173.0	110670/110670	1.000
Ch21 IS95 Low	96.9 to 116.4	93.80	0.20/2.16	74.5	118.2	110610/110670	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110678/110678	1.000
Male							
Ch07 IS95 High	201.3 to 269.0	227.53	0.30/8.03	173.4	294.1	110702/110702	1.000
Ch08 IS95 Med	145.9 to 192.2	148.83	0.23/3.99	112.0	203.3	110456/110702	0.998
Ch10 IS95 Low	103.1 to 135.9	118.65	0.31/4.25	88.8	149.3	110678/110678	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110702/110702	1.000
November 1 to 30, 2012							
Female							
Ch19 IS95 High	232.8 to 246.1	245.80	0.29/8.41	206.0	317.3	107678/107706	1.000
Ch18 IS95 Med	167.6 to 177.3	158.36	0.23/4.24	139.2	187.4	107706/107706	1.000
Ch21 IS95 Low	118.5 to 125.4	104.95	0.20/2.39	92.6	131.4	107610/107706	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107706/107706	1.000
Male							
Ch07 IS95 High	271.8 to 292.2	273.49	0.30/9.67	234.5	337.5	107706/107706	1.000
Ch08 IS95 Med	194.2 to 208.2	174.47	0.22/4.47	152.3	208.3	107632/107706	0.999
Ch10 IS95 Low	137.4 to 147.3	136.25	0.27/4.29	117.0	159.9	107706/107706	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107706/107706	1.000
December 1 to 31, 2012							
Female							
Ch19 IS95 High	246.1 to 254.9	262.13	0.28/8.64	229.3	312.2	111216/111216	1.000
Ch18 IS95 Med	177.3 to 182.7	166.16	0.24/4.65	145.9	193.7	111216/111216	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch21 IS95 Low	125.4 to 129.2	110.20	0.20/2.58	94.7	141.2	111082/111216	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111216/111216	1.000
Male							
Ch07 IS95 High	292.2 to 298.0	284.03	0.30/9.99	246.4	335.2	111216/111216	1.000
Ch08 IS95 Med	208.2 to 211.7	183.33	0.25/5.27	162.5	217.1	111120/111216	0.999
Ch10 IS95 Low	147.3 to 149.7	141.52	0.29/4.73	122.4	163.2	111216/111216	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111216/111216	1.000
January 1 to 31, 2013							
Female							
Ch19 IS95 High	254.9 to 261.4	265.88	0.28/8.61	235.6	306.1	111234/111234	1.000
Ch18 IS95 Med	182.7 to 186.7	167.70	0.28/5.41	108.4	259.5	111168/111234	0.999
Ch21 IS95 Low	129.2 to 130.7	111.49	0.20/2.54	96.5	132.2	111104/111234	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111238/111238	1.000
Male							
Ch07 IS95 High	298.0 to 302.1	286.80	0.30/9.97	250.6	333.8	111238/111238	1.000
Ch08 IS95 Med	211.7 to 214.0	190.71	0.25/5.56	167.5	226.3	111236/111238	1.000
Ch10 IS95 Low	149.7 to 151.4	140.71	0.28/4.62	122.8	164.2	111238/111238	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111238/111238	1.000
February 1 to 28, 2013							
Female							
Ch19 IS95 High	261.4 to 261.4	266.69	0.25/7.85	238.4	306.1	100164/100164	1.000
Ch18 IS95 Med	186.7 to 188.4	172.86	0.21/4.27	154.2	198.3	100164/100164	1.000
Ch21 IS95 Low	130.7 to 132.0	113.76	0.20/2.71	99.6	137.0	100046/100164	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	100164/100164	1.000
Male							
Ch07 IS95 High	302.1 to 303.4	290.09	0.31/10.66	253.4	342.9	100164/100164	1.000
Ch08 IS95 Med	214.0 to 215.5	198.37	0.27/6.30	173.0	229.6	100164/100164	1.000
Ch10 IS95 Low	151.4 to 152.0	139.08	0.26/4.29	124.1	165.2	100164/100164	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	100164/100164	1.000
March 1 to 31, 2013							
Female							
Ch19 IS95 High	261.4 to 264.0	268.11	0.26/8.10	228.7	311.7	111408/111408	1.000
Ch18 IS95 Med	188.4 to 190.2	173.32	0.22/4.37	154.8	200.4	111408/111408	1.000
Ch21 IS95 Low	132.0 to 133.2	114.08	0.20/2.65	100.5	139.9	111270/111408	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
Male							
Ch07 IS95 High	303.4 to 304.7	292.56	0.30/10.38	254.0	342.3	111408/111408	1.000
Ch08 IS95 Med	215.5 to 216.8	201.55	0.25/5.99	178.1	229.9	111408/111408	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch10 IS95 Low	152.0 to 153.3	141.64	0.28/4.60	123.3	171.0	111408/111408	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
April 1 to 30, 2013							
Female							
Ch19 IS95 High	264.0 to 266.5	273.12	0.27/8.76	236.1	324.4	107438/107438	1.000
Ch18 IS95 Med	190.2 to 192.2	176.82	0.21/4.26	161.1	204.2	107438/107438	1.000
Ch21 IS95 Low	133.2 to 134.5	114.99	0.21/2.82	98.1	141.4	107112/107438	0.997
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107438/107438	1.000
Male							
Ch07 IS95 High	304.7 to 306.6	292.26	0.31/10.78	239.3	382.8	107440/107442	1.000
Ch08 IS95 Med	216.8 to 218.3	202.70	0.26/6.24	181.2	235.9	107442/107442	1.000
Ch10 IS95 Low	153.3 to 154.4	141.39	0.26/4.34	124.8	165.1	107438/107438	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107444/107444	1.000
May 1 to 31, 2013							
Female							
Ch19 IS95 High	266.5 to 269.0	277.77	0.28/9.13	233.4	325.0	105524/105524	1.000
Ch18 IS95 Med	192.2 to 194.2	176.25	0.21/4.38	152.7	205.0	105522/105524	1.000
Ch21 IS95 Low	134.5 to 135.9	116.63	0.23/3.07	98.1	140.2	105172/105524	0.997
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105524/105524	1.000
Male							
Ch07 IS95 High	306.6 to 308.8	294.02	0.31/10.66	136.5	344.2	105576/105578	1.000
Ch08 IS95 Med	218.3 to 220.1	205.31	0.28/6.81	180.4	240.7	105576/105576	1.000
Ch10 IS95 Low	154.4 to 155.6	144.69	0.30/5.15	120.2	171.5	105522/105524	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	105580/105580	1.000
June 1 to 30, 2013							
Female							
Ch19 IS95 High	269.0 to 271.8	281.70	0.27/8.80	248.5	327.6	107074/107074	1.000
Ch18 IS95 Med	194.2 to 196.4	179.48	0.22/4.60	160.6	214.7	107074/107074	1.000
Ch21 IS95 Low	135.9 to 137.4	117.99	0.22/2.96	105.2	146.6	106772/107074	0.997
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107074/107074	1.000
Male							
Ch07 IS95 High	308.8 to 310.0	294.60	0.32/10.97	256.1	354.5	107084/107084	1.000
Ch08 IS95 Med	220.1 to 221.0	196.86	0.37/8.48	117.2	227.5	106566/107084	0.995
Ch10 IS95 Low	155.6 to 156.3	144.10	0.30/5.01	125.1	172.4	107074/107074	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107088/107088	1.000
July 1 to 31, 2013							
Female							
Ch19 IS95 High	271.8 to 277.7	287.00	0.27/8.92	256.8	342.3	111054/111054	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch18 IS95 Med	196.4 to 200.7	180.29	0.21/4.33	163.3	209.1	111054/111054	1.000
Ch21 IS95 Low	137.4 to 140.4	120.20	0.20/2.84	106.0	145.9	110840/111054	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111054/111054	1.000
Male							
Ch07 IS95 High	310.0 to 314.0	298.96	0.32/11.20	255.1	350.2	111056/111056	1.000
Ch08 IS95 Med	221.0 to 223.9	200.01	0.25/5.80	176.8	237.8	111052/111056	1.000
Ch10 IS95 Low	156.3 to 158.4	146.34	0.28/4.77	129.0	173.4	111054/111054	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111062/111062	1.000
August 1 to 31, 2013							
Female							
Ch19 IS95 High	277.7 to 280.8	285.33	0.28/9.36	249.9	335.8	111904/111904	1.000
Ch18 IS95 Med	200.7 to 202.8	184.94	0.21/4.62	166.9	215.2	111904/111904	1.000
Ch21 IS95 Low	140.4 to 141.9	122.00	0.21/2.95	106.6	148.1	111758/111904	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111904/111904	1.000
Male							
Ch07 IS95 High	314.0 to 316.7	298.56	0.31/10.91	257.5	354.7	111904/111904	1.000
Ch08 IS95 Med	223.9 to 225.9	202.11	0.25/5.95	177.1	254.5	111902/111904	1.000
Ch10 IS95 Low	158.4 to 159.7	145.77	0.30/5.20	125.4	175.7	111898/111904	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111910/111910	1.000
September 1 to 30, 2013							
Female							
Ch19 IS95 High	280.8 to 280.8	290.94	0.28/9.36	258.5	341.8	107392/107392	1.000
Ch18 IS95 Med	202.8 to 202.8	184.37	0.22/4.62	165.6	215.7	107392/107392	1.000
Ch21 IS95 Low	141.9 to 143.4	122.62	0.22/3.14	105.5	152.3	107002/107392	0.996
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107392/107392	1.000
Male							
Ch07 IS95 High	316.7 to 316.7	302.84	0.33/11.56	247.3	354.3	107378/107392	1.000
Ch08 IS95 Med	225.9 to 226.8	205.80	0.25/5.96	181.1	237.0	107392/107392	1.000
Ch10 IS95 Low	159.7 to 160.4	148.83	0.30/5.18	128.0	173.1	107392/107392	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107406/107406	1.000
October 1 to 31, 2013							
Female							
Ch19 IS95 High	280.8 to 283.8	291.26	0.28/9.50	218.6	333.1	113492/113494	1.000
Ch18 IS95 Med	202.8 to 204.8	178.28	0.33/6.85	102.4	215.2	113226/113496	0.998
Ch21 IS95 Low	143.4 to 144.8	124.81	0.23/3.29	89.9	151.7	113288/113494	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	113496/113496	1.000
Male							
Ch07 IS95 High	316.7 to 318.1	300.02	0.30/10.59	228.1	355.7	113492/113496	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch08 IS95 Med	226.8 to 227.7	207.25	0.27/6.42	155.1	243.1	113494/113496	1.000
Ch10 IS95 Low	160.4 to 161.0	148.27	0.29/5.10	100.6	175.3	113482/113496	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	113614/113614	1.000
November 1 to 30, 2013							
Female							
Ch19 IS95 High	283.8 to 286.8	296.51	0.31/10.60	230.6	338.6	110646/110646	1.000
Ch18 IS95 Med	204.8 to 206.7	176.06	0.25/5.07	102.4	304.9	109814/110646	0.992
Ch21 IS95 Low	144.8 to 146.1	126.33	0.25/3.76	92.3	158.7	110042/110646	0.995
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110646/110646	1.000
Male							
Ch07 IS95 High	318.1 to 319.4	299.24	0.31/10.89	186.9	352.4	110646/110660	1.000
Ch08 IS95 Med	227.7 to 228.7	204.39	0.28/6.62	171.0	265.5	110464/110658	0.998
Ch10 IS95 Low	161.0 to 161.7	148.39	0.31/5.42	124.1	187.1	110620/110658	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	110664/110664	1.000
December 1 to 31, 2013							
Female							
Ch19 IS95 High	286.8 to 289.6	297.13	0.29/10.16	241.1	344.8	106114/106114	1.000
Ch18 IS95 Med	206.7 to 208.2	173.06	0.22/4.50	140.4	199.2	104002/106198	0.979
Ch21 IS95 Low	146.1 to 147.3	126.41	0.22/3.28	101.1	150.9	105674/106114	0.996
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	106198/106198	1.000
Male							
Ch07 IS95 High	319.4 to 319.4	299.40	0.31/10.95	120.2	350.5	106202/106216	1.000
Ch08 IS95 Med	228.7 to 228.7	203.24	0.31/7.46	114.4	258.8	105908/106212	0.997
Ch10 IS95 Low	161.7 to 161.7	149.99	0.32/5.63	122.6	178.0	106162/106198	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	106222/106222	1.000
January 1 to 31, 2014							
Female							
Ch19 IS95 High	289.6 to 292.2	299.08	0.27/9.44	261.9	355.6	111242/111242	1.000
Ch18 IS95 Med	208.2 to 209.6	175.32	0.20/4.07	155.1	198.6	110204/111252	0.991
Ch21 IS95 Low	147.3 to 148.2	127.63	0.19/2.87	108.0	153.6	111094/111242	0.999
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111252/111252	1.000
Male							
Ch07 IS95 High	319.4 to 320.8	300.75	0.32/11.25	259.2	355.8	111254/111254	1.000
Ch08 IS95 Med	228.7 to 230.6	205.87	0.26/6.19	173.4	239.9	111232/111254	1.000
Ch10 IS95 Low	161.7 to 162.4	149.74	0.30/5.23	128.5	178.9	111254/111254	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111258/111258	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
February 1 to 28, 2014							
Female							
Ch19 IS95 High	292.2 to 296.4	306.93	0.27/9.79	257.6	355.4	103948/103948	1.000
Ch18 IS95 Med	209.6 to 210.7	180.40	0.22/4.53	157.1	208.9	103744/103948	0.998
Ch21 IS95 Low	148.2 to 149.7	129.10	0.21/3.15	106.5	160.4	103764/103948	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	103948/103948	1.000
Male							
Ch07 IS95 High	320.8 to 322.1	305.52	0.31/11.28	266.4	355.0	103948/103948	1.000
Ch08 IS95 Med	230.6 to 231.5	206.84	0.24/5.88	179.0	237.2	103940/103948	1.000
Ch10 IS95 Low	162.4 to 163.7	153.91	0.32/5.79	129.9	180.0	103946/103948	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	103960/103960	1.000
March 1 to 31, 2014							
Female							
Ch19 IS95 High	296.4 to 298.0	306.62	0.26/9.46	271.2	388.9	111406/111408	1.000
Ch18 IS95 Med	210.7 to 211.7	179.74	0.21/4.34	161.9	205.3	111206/111408	0.998
Ch21 IS95 Low	149.7 to 150.2	130.12	0.20/2.98	116.8	156.9	111388/111408	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
Male							
Ch07 IS95 High	322.1 to 323.4	304.23	0.32/11.46	264.8	365.8	111408/111408	1.000
Ch08 IS95 Med	231.5 to 231.5	206.39	0.24/5.85	184.2	235.2	111408/111408	1.000
Ch10 IS95 Low	163.7 to 163.7	153.63	0.30/5.35	133.9	178.2	111408/111408	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111408/111408	1.000
April 1 to 30, 2014							
Female							
Ch19 IS95 High	298.0 to 300.4	306.92	0.27/9.72	264.0	362.4	107286/107286	1.000
Ch18 IS95 Med	211.7 to 213.1	179.35	0.20/4.08	100.9	207.4	106834/107288	0.996
Ch21 IS95 Low	150.2 to 150.2	130.62	0.20/3.09	115.5	156.4	107260/107286	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107288/107288	1.000
Male							
Ch07 IS95 High	323.4 to 323.4	304.21	0.29/10.50	154.1	364.7	107292/107294	1.000
Ch08 IS95 Med	231.5 to 232.5	206.77	0.23/5.63	175.3	235.2	107290/107292	1.000
Ch10 IS95 Low	163.7 to 163.7	153.19	0.28/4.94	117.4	180.0	107284/107288	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107298/107298	1.000
May 1 to 31, 2014							
Female							
Ch19 IS95 High	300.4 to 300.4	310.23	0.26/9.46	278.7	361.9	111204/111204	1.000
Ch18 IS95 Med	213.1 to 213.1	181.02	0.20/4.21	160.2	213.0	110928/111208	0.997
Ch21 IS95 Low	150.2 to 150.7	130.02	0.20/3.01	116.2	160.0	111138/111204	0.999

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111208/111208	1.000
Male							
Ch07 IS95 High	323.4 to 323.4	301.79	0.28/9.78	260.5	350.8	111208/111208	1.000
Ch08 IS95 Med	232.5 to 232.5	207.87	0.24/5.84	183.8	239.2	111204/111208	1.000
Ch10 IS95 Low	163.7 to 163.7	156.28	0.33/6.00	133.8	188.0	111208/111208	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111208/111208	1.000
June 1 to 30, 2014							
Female							
Ch19 IS95 High	300.4 to 301.3	305.71	0.35/12.62	201.8	415.4	107452/107534	0.999
Ch18 IS95 Med	213.1 to 214.0	181.65	0.22/4.69	28.4	210.4	107186/107542	0.997
Ch21 IS95 Low	150.7 to 151.0	131.33	0.20/3.01	117.0	158.9	107524/107534	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107542/107542	1.000
Male							
Ch07 IS95 High	322.1 to 323.4	300.92	0.27/9.67	258.0	353.6	107546/107546	1.000
Ch08 IS95 Med	232.5 to 233.4	206.99	0.24/5.68	181.2	235.7	107534/107546	1.000
Ch10 IS95 Low	163.0 to 163.7	155.78	0.29/5.34	134.9	182.9	107542/107542	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	107548/107548	1.000
July 1 to 31, 2014							
Female							
Ch19 IS95 High	302.1 to 302.1	309.91	0.27/9.74	274.2	367.1	111202/111202	1.000
Ch18 IS95 Med	214.0 to 214.5	180.86	0.18/3.69	163.2	213.4	110936/111202	0.998
Ch21 IS95 Low	151.0 to 151.0	133.65	0.20/3.14	118.5	198.6	111190/111202	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111202/111202	1.000
Male							
Ch07 IS95 High	322.1 to 322.1	298.86	0.28/9.65	257.8	360.2	111202/111202	1.000
Ch08 IS95 Med	231.5 to 232.5	206.55	0.22/5.37	183.6	236.4	111200/111202	1.000
Ch10 IS95 Low	163.0 to 163.7	153.76	0.28/5.01	134.7	175.0	111202/111202	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	111206/111206	1.000
August 1 to 31, 2014							
Female							
Ch19 IS95 High	302.1 to 302.1	301.60	0.26/9.21	252.8	352.3	108708/108708	1.000
Ch18 IS95 Med	214.5 to 214.5	183.36	0.21/4.42	158.5	222.5	108518/108708	0.998
Ch21 IS95 Low	151.0 to 151.0	131.88	0.24/3.69	107.2	164.6	108494/108708	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	108708/108708	1.000
Male							
Ch07 IS95 High	320.8 to 322.1	297.12	0.28/9.67	245.4	349.2	108704/108708	1.000
Ch08 IS95 Med	229.6 to 231.5	207.72	0.26/6.27	171.0	246.6	108676/108708	1.000
Ch10 IS95 Low	161.7 to 163.0	153.23	0.27/4.91	127.2	175.6	108706/108708	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	108716/108716	1.000
September 1 to 30, 2014							
Female							
Ch19 IS95 High	302.1 to 302.1	300.56	0.26/9.05	247.7	395.6	104162/104176	1.000
Ch18 IS95 Med	214.0 to 214.5	187.43	0.23/4.98	166.7	242.6	104170/104176	1.000
Ch21 IS95 Low	150.7 to 151.0	138.88	0.32/5.13	115.5	199.1	104172/104176	1.000
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	104176/104176	1.000
Male							
Ch07 IS95 High	320.8 to 320.8	299.37	0.22/7.51	250.7	395.7	104174/104176	1.000
Ch08 IS95 Med	228.7 to 229.6	212.35	0.31/7.69	182.0	276.7	104176/104176	1.000
Ch10 IS95 Low	163.0 to 163.0	155.34	0.25/4.55	129.6	193.5	104176/104176	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	104176/104176	1.000
August 8, 2012 to September 30, 2014							
Female							
Ch19 IS95 High	192.6 to 302.1	282.98	0.30/9.79	164.5	415.4	2800246/2800388	1.000
Ch18 IS95 Med	135.9 to 214.5	175.09	0.37/7.72	28.4	304.9	2794118/2800498	0.998
Ch21 IS95 Low	96.3 to 151.0	121.89	0.36/5.19	74.5	199.1	2796040/2800388	0.998
Ch15 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	2800510/2800510	1.000
Male							
Ch07 IS95 High	192.2 to 323.4	289.61	0.31/10.58	120.2	395.7	2800580/2800648	1.000
Ch08 IS95 Med	136.2 to 233.4	197.37	0.33/7.73	102.5	288.1	2799062/2800638	0.999
Ch10 IS95 Low	96.3 to 163.7	144.71	0.32/5.37	77.3	193.5	2800404/2800524	1.000
Ch04 Sham Control	0.0 to 0.0	0.00	–/0.00	0.0	0.0	2800844/2800844	1.000

Ch = chamber (e.g., Ch19 = Chamber 19).

Table I-8. Summary of CDMA-modulated Cell Phone RFR Exposure Data in Rats – H-Field

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
August 8 to 31, 2012							
Female							
Ch19 IS95 High	0.65 to 0.75	0.699	0.15/0.012	0.45	0.83	85780/85786	1.000
Ch18 IS95 Med	0.46 to 0.53	0.510	0.17/0.010	0.35	0.62	85780/85786	1.000
Ch21 IS95 Low	0.33 to 0.38	0.342	0.16/0.006	0.22	0.42	85780/85786	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	85786/85786	1.000
September 1 to 30, 2012							
Female							
Ch19 IS95 High	0.51 to 0.71	0.627	0.21/0.016	0.46	0.81	105918/105918	1.000
Ch18 IS95 Med	0.36 to 0.51	0.469	0.31/0.017	0.34	0.58	105798/105918	0.999
Ch21 IS95 Low	0.26 to 0.36	0.324	0.49/0.019	0.24	0.43	105844/105918	0.999

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	105918/105918	1.000
Male							
Ch07 IS95 High	0.51 to 0.71	0.650	0.25/0.019	0.49	1.07	105906/105918	1.000
Ch08 IS95 Med	0.36 to 0.51	0.456	0.31/0.016	0.34	0.75	105820/105918	0.999
Ch10 IS95 Low	0.26 to 0.36	0.319	0.27/0.010	0.22	0.53	105758/105918	0.998
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	105918/105918	1.000
October 1 to 31, 2012							
Female							
Ch19 IS95 High	0.51 to 0.61	0.555	0.25/0.016	0.45	0.67	110670/110670	1.000
Ch18 IS95 Med	0.37 to 0.44	0.435	0.25/0.013	0.35	0.53	110668/110670	1.000
Ch21 IS95 Low	0.26 to 0.31	0.322	0.25/0.009	0.26	0.40	110606/110670	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	110678/110678	1.000
Male							
Ch07 IS95 High	0.53 to 0.71	0.663	0.23/0.018	0.50	0.82	110702/110702	1.000
Ch08 IS95 Med	0.39 to 0.51	0.516	0.27/0.017	0.38	0.65	110676/110702	1.000
Ch10 IS95 Low	0.27 to 0.36	0.329	0.28/0.011	0.24	0.42	110678/110678	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	110702/110702	1.000
November 1 to 30, 2012							
Female							
Ch19 IS95 High	0.62 to 0.65	0.625	0.26/0.019	0.54	0.78	107706/107706	1.000
Ch18 IS95 Med	0.45 to 0.47	0.498	0.23/0.013	0.44	0.58	107706/107706	1.000
Ch21 IS95 Low	0.31 to 0.33	0.369	0.24/0.011	0.32	0.45	107640/107706	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107706/107706	1.000
Male							
Ch07 IS95 High	0.72 to 0.78	0.772	0.20/0.018	0.69	0.87	107706/107706	1.000
Ch08 IS95 Med	0.52 to 0.55	0.607	0.27/0.019	0.51	0.71	107688/107706	1.000
Ch10 IS95 Low	0.36 to 0.39	0.396	0.25/0.012	0.35	0.46	107706/107706	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107706/107706	1.000
December 1 to 31, 2012							
Female							
Ch19 IS95 High	0.65 to 0.68	0.644	0.24/0.018	0.56	0.74	111216/111216	1.000
Ch18 IS95 Med	0.47 to 0.49	0.519	0.24/0.015	0.45	0.61	111214/111216	1.000
Ch21 IS95 Low	0.33 to 0.34	0.385	0.24/0.011	0.33	0.47	111128/111216	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111216/111216	1.000
Male							
Ch07 IS95 High	0.78 to 0.79	0.817	0.21/0.020	0.74	0.95	111216/111216	1.000
Ch08 IS95 Med	0.55 to 0.56	0.630	0.28/0.020	0.56	0.72	111210/111216	1.000
Ch10 IS95 Low	0.39 to 0.40	0.415	0.26/0.013	0.37	0.48	111216/111216	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111216/111216	1.000
January 1 to 31, 2013							
Female							
Ch19 IS95 High	0.68 to 0.69	0.665	0.22/0.017	0.59	0.77	111234/111234	1.000
Ch18 IS95 Med	0.49 to 0.50	0.536	0.31/0.019	0.38	0.81	111018/111234	0.998
Ch21 IS95 Low	0.34 to 0.35	0.392	0.25/0.011	0.35	0.46	111170/111234	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111238/111238	1.000
Male							
Ch07 IS95 High	0.79 to 0.80	0.830	0.21/0.021	0.76	0.92	111238/111238	1.000
Ch08 IS95 Med	0.56 to 0.57	0.622	0.27/0.020	0.57	0.70	111238/111238	1.000
Ch10 IS95 Low	0.40 to 0.40	0.425	0.25/0.013	0.38	0.48	111238/111238	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111238/111238	1.000
February 1 to 28, 2013							
Female							
Ch19 IS95 High	0.69 to 0.69	0.678	0.21/0.017	0.60	0.77	100164/100164	1.000
Ch18 IS95 Med	0.50 to 0.50	0.536	0.20/0.012	0.48	0.60	100164/100164	1.000
Ch21 IS95 Low	0.35 to 0.35	0.394	0.24/0.011	0.34	0.47	100088/100164	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	100164/100164	1.000
Male							
Ch07 IS95 High	0.80 to 0.81	0.835	0.23/0.023	0.75	0.92	100164/100164	1.000
Ch08 IS95 Med	0.57 to 0.57	0.611	0.26/0.018	0.55	0.71	100164/100164	1.000
Ch10 IS95 Low	0.40 to 0.40	0.435	0.25/0.013	0.39	0.50	100164/100164	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	100164/100164	1.000
March 1 to 31, 2013							
Female							
Ch19 IS95 High	0.69 to 0.70	0.683	0.21/0.017	0.61	0.76	111408/111408	1.000
Ch18 IS95 Med	0.50 to 0.51	0.544	0.22/0.014	0.50	0.64	111406/111408	1.000
Ch21 IS95 Low	0.35 to 0.35	0.399	0.24/0.011	0.35	0.49	111336/111408	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111408/111408	1.000
Male							
Ch07 IS95 High	0.81 to 0.81	0.835	0.21/0.021	0.76	0.92	111408/111408	1.000
Ch08 IS95 Med	0.57 to 0.58	0.610	0.22/0.016	0.56	0.67	111408/111408	1.000
Ch10 IS95 Low	0.40 to 0.41	0.433	0.25/0.013	0.39	0.50	111408/111408	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111408/111408	1.000
April 1 to 30, 2013							
Female							
Ch19 IS95 High	0.70 to 0.71	0.684	0.24/0.019	0.61	0.78	107438/107438	1.000
Ch18 IS95 Med	0.51 to 0.51	0.546	0.20/0.013	0.49	0.61	107438/107438	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch21 IS95 Low	0.35 to 0.36	0.404	0.25/0.012	0.36	0.49	107356/107438	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107438/107438	1.000
Male							
Ch07 IS95 High	0.81 to 0.81	0.847	0.25/0.024	0.73	1.04	107426/107442	1.000
Ch08 IS95 Med	0.58 to 0.58	0.616	0.23/0.016	0.56	0.70	107442/107442	1.000
Ch10 IS95 Low	0.41 to 0.41	0.441	0.25/0.013	0.39	0.51	107438/107438	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107444/107444	1.000
May 1 to 31, 2013							
Female							
Ch19 IS95 High	0.71 to 0.71	0.686	0.23/0.019	0.62	0.79	105524/105524	1.000
Ch18 IS95 Med	0.51 to 0.52	0.559	0.22/0.014	0.50	0.65	105520/105524	1.000
Ch21 IS95 Low	0.36 to 0.36	0.407	0.26/0.013	0.35	0.49	105446/105524	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	105524/105524	1.000
Male							
Ch07 IS95 High	0.81 to 0.82	0.853	0.23/0.022	0.41	0.95	105576/105578	1.000
Ch08 IS95 Med	0.58 to 0.58	0.619	0.24/0.018	0.55	0.69	105576/105576	1.000
Ch10 IS95 Low	0.41 to 0.41	0.439	0.27/0.014	0.38	0.51	105524/105524	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	105580/105580	1.000
June 1 to 30, 2013							
Female							
Ch19 IS95 High	0.71 to 0.72	0.691	0.22/0.018	0.63	0.80	107074/107074	1.000
Ch18 IS95 Med	0.52 to 0.52	0.563	0.23/0.015	0.50	0.64	107074/107074	1.000
Ch21 IS95 Low	0.36 to 0.36	0.412	0.27/0.013	0.36	0.50	106962/107074	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107074/107074	1.000
Male							
Ch07 IS95 High	0.82 to 0.82	0.858	0.22/0.022	0.78	0.95	107084/107084	1.000
Ch08 IS95 Med	0.58 to 0.59	0.643	0.34/0.025	0.44	0.74	107080/107084	1.000
Ch10 IS95 Low	0.41 to 0.42	0.445	0.29/0.015	0.40	0.51	107074/107074	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107088/107088	1.000
July 1 to 31, 2013							
Female							
Ch19 IS95 High	0.72 to 0.74	0.695	0.22/0.017	0.63	0.78	111054/111054	1.000
Ch18 IS95 Med	0.52 to 0.53	0.573	0.20/0.013	0.52	0.66	111054/111054	1.000
Ch21 IS95 Low	0.36 to 0.37	0.415	0.25/0.012	0.36	0.50	110982/111054	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111054/111054	1.000
Male							
Ch07 IS95 High	0.82 to 0.83	0.860	0.23/0.023	0.78	0.97	111056/111056	1.000
Ch08 IS95 Med	0.59 to 0.59	0.649	0.23/0.018	0.59	0.73	111056/111056	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch10 IS95 Low	0.42 to 0.42	0.446	0.25/0.013	0.40	0.51	111054/111054	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111062/111062	1.000
August 1 to 31, 2013							
Female							
Ch19 IS95 High	0.74 to 0.75	0.717	0.23/0.019	0.63	0.82	111904/111904	1.000
Ch18 IS95 Med	0.53 to 0.54	0.574	0.20/0.014	0.52	0.66	111904/111904	1.000
Ch21 IS95 Low	0.37 to 0.38	0.420	0.25/0.012	0.37	0.49	111844/111904	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111904/111904	1.000
Male							
Ch07 IS95 High	0.83 to 0.84	0.871	0.24/0.024	0.80	0.98	111904/111904	1.000
Ch08 IS95 Med	0.59 to 0.60	0.649	0.24/0.019	0.53	0.82	111894/111904	1.000
Ch10 IS95 Low	0.42 to 0.42	0.453	0.28/0.015	0.40	0.53	111904/111904	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111910/111910	1.000
September 1 to 30, 2013							
Female							
Ch19 IS95 High	0.75 to 0.75	0.715	0.22/0.018	0.64	0.82	107392/107392	1.000
Ch18 IS95 Med	0.54 to 0.54	0.584	0.21/0.015	0.53	0.67	107392/107392	1.000
Ch21 IS95 Low	0.38 to 0.38	0.426	0.25/0.013	0.37	0.52	107314/107392	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107392/107392	1.000
Male							
Ch07 IS95 High	0.84 to 0.84	0.871	0.24/0.024	0.70	1.01	107392/107392	1.000
Ch08 IS95 Med	0.60 to 0.60	0.650	0.23/0.017	0.59	0.72	107392/107392	1.000
Ch10 IS95 Low	0.42 to 0.43	0.452	0.27/0.014	0.39	0.51	107392/107392	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107406/107406	1.000
October 1 to 31, 2013							
Female							
Ch19 IS95 High	0.75 to 0.75	0.720	0.22/0.019	0.52	0.82	113492/113494	1.000
Ch18 IS95 Med	0.54 to 0.54	0.604	0.33/0.024	0.40	0.69	113448/113496	1.000
Ch21 IS95 Low	0.38 to 0.38	0.429	0.28/0.014	0.30	0.54	113444/113494	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	113496/113496	1.000
Male							
Ch07 IS95 High	0.84 to 0.84	0.880	0.23/0.024	0.70	1.04	113496/113496	1.000
Ch08 IS95 Med	0.60 to 0.60	0.651	0.25/0.019	0.48	0.73	113494/113496	1.000
Ch10 IS95 Low	0.43 to 0.43	0.458	0.26/0.014	0.34	0.52	113496/113496	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	113614/113614	1.000
November 1 to 30, 2013							
Female							
Ch19 IS95 High	0.75 to 0.76	0.723	0.25/0.021	0.57	0.84	110644/110646	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch18 IS95 Med	0.54 to 0.55	0.622	0.25/0.018	0.33	0.93	110634/110646	1.000
Ch21 IS95 Low	0.38 to 0.39	0.433	0.31/0.016	0.33	0.53	110610/110646	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	110646/110646	1.000
Male							
Ch07 IS95 High	0.84 to 0.85	0.887	0.25/0.026	0.54	0.99	110658/110660	1.000
Ch08 IS95 Med	0.60 to 0.61	0.666	0.30/0.024	0.55	0.87	110572/110658	0.999
Ch10 IS95 Low	0.43 to 0.43	0.461	0.29/0.016	0.39	0.53	110658/110658	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	110664/110664	1.000
December 1 to 31, 2013							
Female							
Ch19 IS95 High	0.76 to 0.77	0.738	0.23/0.020	0.62	0.84	106114/106114	1.000
Ch18 IS95 Med	0.55 to 0.55	0.639	0.24/0.018	0.55	0.72	106170/106198	1.000
Ch21 IS95 Low	0.39 to 0.39	0.440	0.27/0.014	0.36	0.52	106088/106114	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	106198/106198	1.000
Male							
Ch07 IS95 High	0.85 to 0.85	0.889	0.25/0.026	0.34	0.99	106212/106216	1.000
Ch08 IS95 Med	0.61 to 0.61	0.670	0.29/0.023	0.48	0.88	106204/106212	1.000
Ch10 IS95 Low	0.43 to 0.43	0.458	0.29/0.016	0.37	0.52	106198/106198	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	106222/106222	1.000
January 1 to 31, 2014							
Female							
Ch19 IS95 High	0.77 to 0.78	0.748	0.22/0.019	0.65	0.84	111242/111242	1.000
Ch18 IS95 Med	0.55 to 0.56	0.641	0.21/0.016	0.58	0.71	111246/111252	1.000
Ch21 IS95 Low	0.39 to 0.39	0.443	0.24/0.012	0.37	0.52	111212/111242	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111252/111252	1.000
Male							
Ch07 IS95 High	0.85 to 0.85	0.890	0.25/0.026	0.77	1.00	111254/111254	1.000
Ch08 IS95 Med	0.61 to 0.61	0.671	0.24/0.019	0.59	0.74	111254/111254	1.000
Ch10 IS95 Low	0.43 to 0.43	0.462	0.26/0.014	0.40	0.54	111254/111254	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111258/111258	1.000
February 1 to 28, 2014							
Female							
Ch19 IS95 High	0.78 to 0.79	0.748	0.21/0.018	0.63	0.84	103948/103948	1.000
Ch18 IS95 Med	0.56 to 0.56	0.634	0.22/0.016	0.55	0.71	103946/103948	1.000
Ch21 IS95 Low	0.39 to 0.40	0.446	0.25/0.013	0.37	0.54	103908/103948	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	103948/103948	1.000
Male							
Ch07 IS95 High	0.85 to 0.85	0.884	0.25/0.026	0.79	0.98	103948/103948	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch08 IS95 Med	0.61 to 0.61	0.675	0.22/0.018	0.58	0.76	103948/103948	1.000
Ch10 IS95 Low	0.43 to 0.43	0.457	0.27/0.014	0.40	0.52	103948/103948	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	103960/103960	1.000
March 1 to 31, 2014							
Female							
Ch19 IS95 High	0.79 to 0.79	0.761	0.22/0.020	0.68	0.87	111408/111408	1.000
Ch18 IS95 Med	0.56 to 0.56	0.642	0.22/0.016	0.59	0.72	111404/111408	1.000
Ch21 IS95 Low	0.40 to 0.40	0.448	0.23/0.012	0.40	0.52	111388/111408	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111408/111408	1.000
Male							
Ch07 IS95 High	0.85 to 0.86	0.895	0.25/0.026	0.80	1.02	111408/111408	1.000
Ch08 IS95 Med	0.61 to 0.61	0.678	0.23/0.018	0.62	0.75	111408/111408	1.000
Ch10 IS95 Low	0.43 to 0.43	0.460	0.25/0.013	0.41	0.52	111408/111408	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111408/111408	1.000
April 1 to 30, 2014							
Female							
Ch19 IS95 High	0.79 to 0.80	0.772	0.22/0.020	0.69	0.89	107286/107286	1.000
Ch18 IS95 Med	0.56 to 0.57	0.650	0.20/0.015	0.37	0.72	107284/107288	1.000
Ch21 IS95 Low	0.40 to 0.40	0.447	0.24/0.012	0.39	0.53	107262/107286	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107288/107288	1.000
Male							
Ch07 IS95 High	0.86 to 0.86	0.897	0.22/0.023	0.43	0.99	107292/107294	1.000
Ch08 IS95 Med	0.61 to 0.62	0.680	0.22/0.017	0.61	0.74	107292/107292	1.000
Ch10 IS95 Low	0.43 to 0.43	0.460	0.24/0.013	0.36	0.53	107288/107288	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107298/107298	1.000
May 1 to 31, 2014							
Female							
Ch19 IS95 High	0.80 to 0.80	0.766	0.20/0.018	0.69	0.86	111204/111204	1.000
Ch18 IS95 Med	0.57 to 0.57	0.646	0.21/0.016	0.59	0.74	111204/111208	1.000
Ch21 IS95 Low	0.40 to 0.40	0.451	0.25/0.013	0.40	0.55	111114/111204	0.999
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111208/111208	1.000
Male							
Ch07 IS95 High	0.86 to 0.86	0.907	0.24/0.025	0.81	1.01	111208/111208	1.000
Ch08 IS95 Med	0.62 to 0.62	0.679	0.23/0.018	0.60	0.74	111208/111208	1.000
Ch10 IS95 Low	0.43 to 0.43	0.452	0.28/0.015	0.40	0.52	111208/111208	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111208/111208	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
June 1 to 30, 2014							
Female							
Ch19 IS95 High	0.80 to 0.80	0.778	0.24/0.022	0.57	1.00	107446/107534	0.999
Ch18 IS95 Med	0.57 to 0.57	0.649	0.21/0.016	0.60	0.72	107540/107542	1.000
Ch21 IS95 Low	0.40 to 0.40	0.450	0.23/0.012	0.40	0.52	107524/107534	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107542/107542	1.000
Male							
Ch07 IS95 High	0.85 to 0.86	0.902	0.22/0.023	0.82	1.01	107546/107546	1.000
Ch08 IS95 Med	0.62 to 0.62	0.683	0.24/0.019	0.62	0.75	107546/107546	1.000
Ch10 IS95 Low	0.43 to 0.43	0.454	0.25/0.013	0.41	0.51	107542/107542	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	107548/107548	1.000
July 1 to 31, 2014							
Female							
Ch19 IS95 High	0.80 to 0.80	0.776	0.22/0.019	0.71	0.88	111202/111202	1.000
Ch18 IS95 Med	0.57 to 0.57	0.653	0.20/0.015	0.61	0.73	111188/111202	1.000
Ch21 IS95 Low	0.40 to 0.40	0.444	0.23/0.012	0.40	0.62	111186/111202	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111202/111202	1.000
Male							
Ch07 IS95 High	0.85 to 0.85	0.909	0.23/0.024	0.84	1.03	111202/111202	1.000
Ch08 IS95 Med	0.61 to 0.62	0.681	0.22/0.017	0.61	0.75	111202/111202	1.000
Ch10 IS95 Low	0.43 to 0.43	0.457	0.24/0.013	0.42	0.51	111202/111202	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	111206/111206	1.000
August 1 to 31, 2014							
Female							
Ch19 IS95 High	0.80 to 0.80	0.797	0.22/0.020	0.67	0.91	108708/108708	1.000
Ch18 IS95 Med	0.57 to 0.57	0.647	0.22/0.016	0.58	0.73	108702/108708	1.000
Ch21 IS95 Low	0.40 to 0.40	0.448	0.27/0.014	0.38	0.54	108676/108708	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	108708/108708	1.000
Male							
Ch07 IS95 High	0.85 to 0.85	0.909	0.24/0.025	0.80	1.01	108708/108708	1.000
Ch08 IS95 Med	0.61 to 0.61	0.671	0.26/0.020	0.57	0.77	108708/108708	1.000
Ch10 IS95 Low	0.43 to 0.43	0.454	0.25/0.013	0.39	0.53	108708/108708	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	108716/108716	1.000
September 1 to 30, 2014							
Female							
Ch19 IS95 High	0.80 to 0.80	0.801	0.23/0.022	0.65	1.06	104152/104176	1.000
Ch18 IS95 Med	0.57 to 0.57	0.636	0.25/0.018	0.52	0.77	104166/104176	1.000
Ch21 IS95 Low	0.40 to 0.40	0.429	0.33/0.017	0.31	0.55	104170/104176	1.000

Chamber	Target Range [V/m]	Mean [V/m]	Stdev [dB]/[V/m]	Min [V/m]	Max [V/m]	In Range/Total	Ratio
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	104176/104176	1.000
Male							
Ch07 IS95 High	0.85 to 0.85	0.902	0.20/0.021	0.76	1.12	104170/104176	1.000
Ch08 IS95 Med	0.61 to 0.61	0.648	0.32/0.024	0.53	0.80	104174/104176	1.000
Ch10 IS95 Low	0.43 to 0.43	0.451	0.24/0.013	0.38	0.55	104174/104176	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	104176/104176	1.000
August 8, 2012 to September 30, 2014							
Female							
Ch19 IS95 High	0.51 to 0.80	0.711	0.25/0.021	0.45	1.06	2800266/2800388	1.000
Ch18 IS95 Med	0.36 to 0.57	0.582	0.38/0.026	0.33	0.93	2800006/2800498	1.000
Ch21 IS95 Low	0.26 to 0.40	0.413	0.38/0.019	0.22	0.62	2799016/2800388	1.000
Ch15 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	2800510/2800510	1.000
Male							
Ch07 IS95 High	0.51 to 0.86	0.849	0.24/0.024	0.34	1.12	2800596/2800648	1.000
Ch08 IS95 Med	0.36 to 0.62	0.633	0.32/0.024	0.29	0.88	2800368/2800638	1.000
Ch10 IS95 Low	0.26 to 0.43	0.434	0.29/0.015	0.21	0.55	2800350/2800524	1.000
Ch04 Sham Control	0.00 to 0.00	0.000	–/0.000	0.00	0.00	2800844/2800844	1.000

Ch = chamber (e.g., Ch19 = Chamber 19).

Appendix J. Ingredients, Nutrient Composition, and Contaminant Levels in NTP-2000 Rat and Mouse Ration

Tables

Table J-1. Ingredients of NTP-2000 Rat and Mouse Ration J-2
Table J-2. Vitamins and Minerals in NTP-2000 Rat and Mouse Ration J-2
Table J-3. Nutrient Composition of NTP-2000 Rat and Mouse Ration J-3
Table J-4. Contaminant Levels in NTP-2000 Rat and Mouse Ration J-4

Table J-1. Ingredients of NTP-2000 Rat and Mouse Ration

Ingredients	Percent by Weight
Ground hard winter wheat	22.26
Ground #2 yellow shelled corn	22.18
Wheat middlings	15.0
Oat hulls	8.5
Alfalfa meal (dehydrated, 17% protein)	7.5
Purified cellulose	5.5
Soybean meal (49% protein)	5.0
Fish meal (60% protein)	4.0
Corn oil (without preservatives)	3.0
Soy oil (without preservatives)	3.0
Dried brewer's yeast	1.0
Calcium carbonate (USP)	0.9
Vitamin premix[a]	0.5
Mineral premix[b]	0.5
Calcium phosphate, dibasic (USP)	0.4
Sodium chloride	0.3
Choline chloride (70% choline)	0.26
Methionine	0.2

[a]Wheat middlings as carrier.
[b]Calcium carbonate as carrier.

Table J-2. Vitamins and Minerals in NTP-2000 Rat and Mouse Ration[a]

	Amount	Source
Vitamins		
A	4,000 IU	Stabilized vitamin A palmitate or acetate
D	1,000 IU	D-activated animal sterol
K	1.0 mg	Menadione sodium bisulfite complex
α-Tocopheryl acetate	100 IU	–
Niacin	23 mg	–
Folic acid	1.1 mg	–
d-Pantothenic acid	10 mg	d-Calcium pantothenate
Riboflavin	3.3 mg	–
Thiamine	4 mg	Thiamine mononitrate
B12	52 µg	–
Pyridoxine	6.3 mg	Pyridoxine hydrochloride

	Amount	Source
Biotin	0.2 mg	d-Biotin
Minerals		
Magnesium	514 mg	Magnesium oxide
Iron	35 mg	Iron sulfate
Zinc	12 mg	Zinc oxide
Manganese	10 mg	Manganese oxide
Copper	2.0 mg	Copper sulfate
Iodine	0.2 mg	Calcium iodate
Chromium	0.2 mg	Chromium acetate

[a]Per kg of finished product.

Table J-3. Nutrient Composition of NTP-2000 Rat and Mouse Ration

Nutrient	Mean ± Standard Deviation	Range	Number of Samples
Protein (% by weight)	14.4 ± 0.38	13.9–15.1	17
Crude fat (% by weight)	8.4 ± 0.37	7.7–9.2	17
Crude fiber (% by weight)	9.4 ± 0.41	8.6–9.9	17
Ash (% by weight)	4.9 ± 0.13	4.7–5.1	17
Amino Acids (% of total diet)			
Arginine	0.794 ± 0.070	0.67–0.97	26
Cystine	0.220 ± 0.022	0.15–0.25	26
Glycine	0.700 ± 0.038	0.62–0.80	26
Histidine	0.344 ± 0.074	0.27–0.68	26
Isoleucine	0.546 ± 0.041	0.43–0.66	26
Leucine	1.092 ± 0.063	0.96–1.24	26
Lysine	0.700 ± 0.110	0.31–0.86	26
Methionine	0.408 ± 0.043	0.26–0.49	26
Phenylalanine	0.621 ± 0.048	0.47–0.72	26
Threonine	0.508 ± 0.040	0.43–0.61	26
Tryptophan	0.153 ± 0.027	0.11–0.20	26
Tyrosine	0.413 ± 0.063	0.28–0.54	26
Valine	0.663 ± 0.040	0.55–0.73	26
Essential Fatty Acids (% of total diet)			
Linoleic	3.95 ± 0.242	3.49–4.55	26
Linolenic	0.31 ± 0.030	0.21–0.35	26
Vitamins			
Vitamin A (IU/kg)	3,899 ± 77	2,820–5,450	17

Nutrient	Mean ± Standard Deviation	Range	Number of Samples
Vitamin D (IU/kg)	1,000[a]	–	–
α-Tocopherol (ppm)	79.7 ± 20.42	27.0–124.0	26
Thiamine (ppm)[b]	11.8 ± 17.85	6.6–81.0	17
Riboflavin (ppm)	8.1 ± 2.91	4.20–17.50	26
Niacin (ppm)	78.9 ± 8.52	66.4–98.2	26
Pantothenic acid (ppm)	26.7 ± 11.63	17.4–81.0	26
Pyridoxine (ppm)[b]	9.7 ± 2.09	6.44–14.3	26
Folic acid (ppm)	1.59 ± 0.45	1.15–3.27	26
Biotin (ppm)	0.32 ± 0.10	0.20–0.704	26
Vitamin B12 (ppb)	51.8 ± 36.6	18.3–174.0	26
Choline (ppm)b	2,665 ± 631	1,160–3,790	26
Minerals			
Calcium (%)	0.903 ± 0.070	0.697–1.01	17
Phosphorus (%)	0.553 ± 0.026	0.510–0.596	17
Potassium (%)	0.669 ± 0.030	0.626–0.733	26
Chloride (%)	0.386 ± 0.037	0.300–0.474	26
Sodium (%)	0.193 ± 0.024	0.160–0.283	26
Magnesium (%)	0.216 ± 0.057	0.185–0.490	26
Sulfur (%)	0.170 ± 0.029	0.116–0.209	14
Iron (ppm)	190.5 ± 38.0	135–311	26
Manganese (ppm)	50.7 ± 9.72	21.0–73.1	26
Zinc (ppm)	58.2 ± 26.89	43.3–184.0	26
Copper (ppm)	7.44 ± 2.60	3.21–16.3	26
Iodine (ppm)	0.514 ± 0.195	0.158–0.972	26
Chromium (ppm)	0.674 ± 0.265	0.330–1.380	25
Cobalt (ppm)	0.235 ± 0.157	0.094–0.864	24

[a]From formulation.
[b]As hydrochloride (thiamine and pyridoxine) or chloride (choline).

Table J-4. Contaminant Levels in NTP-2000 Rat and Mouse Ration[a]

	Mean ± Standard Deviation[b]	Range	Number of Samples
Contaminants			
Arsenic (ppm)	0.20 ± 0.039	0.14–0.28	17
Cadmium (ppm)	0.05 ± 0.004	0.04–0.06	17
Lead (ppm)	0.21 ± 0.027	0.07–1.19	17
Mercury (ppm)	<0.02	–	17
Selenium (ppm)	0.17 ± 0.024	0.10–0.20	17

	Mean ± Standard Deviation[b]	Range	Number of Samples
Aflatoxins (ppb)	<5.00	–	17
Nitrate nitrogen (ppm)c	18.76 ± 9.49	10.0–45.9	17
Nitrite nitrogen (ppm)c	0.61	–	17
BHA (ppm)d	<1.0	–	17
BHT (ppm)d	<1.0	–	17
Aerobic plate count (CFU/g)	<10.0	–	17
Coliform (MPN/g)	3.0	–	17
Escherichia coli (MPN/g)	<10	–	17
Salmonella (MPN/g)	Negative	–	17
Total nitrosoamines (ppb)e	9.2 ± 5.55	0.0–19.9	17
N-Nitrosodimethylamine (ppb)e	1.3 ± 1.04	0.0–3.0	17
N-Nitrosopyrrolidine (ppb)e	8.0 ± 5.02	0.0–18.6	17
Pesticides (ppm)			
α-BHC	<0.01	–	17
β-BHC	<0.02	–	17
γ-BHC	<0.01	–	17
δ-BHC	<0.01	–	17
Heptachlor	<0.01	–	17
Aldrin	<0.01	–	17
Heptachlor epoxide	<0.01	–	17
DDE	<0.01	–	17
DDD	<0.01	–	17
DDT	<0.01	–	17
HCB	<0.01	–	17
Mirex	<0.01	–	17
Methoxychlor	<0.05	–	17
Dieldrin	<0.01	–	17
Endrin	<0.01	–	17
Telodrin	<0.01	–	17
Chlordane	<0.05	–	17
Toxaphene	<0.10	–	17
Estimated PCBs	<0.20	–	17
Ronnel	<0.01	–	17
Ethion	<0.02	–	17
Trithion	<0.05	–	17
Diazinon	<0.10	–	17

	Mean ± Standard Deviation[b]	Range	Number of Samples
Methyl chlorpyrifos	0.16 ± 0.179	0.02–0.686	17
Methyl parathion	<0.02	–	17
Ethyl parathion	<0.02	–	17
Malathion	0.117 ± 0.140	0.02–0.585	17
Endosulfan I	<0.01	–	17
Endosulfan II	<0.01	–	17
Endosulfan sulfate	<0.03	–	17

[a]All samples were irradiated. CFU = colony-forming units; MPN = most probable number; BHC = hexachlorocyclohexane or benzene hexachloride.
[b]For values less than the limit of detection, the detection limit is given as the mean.
[c]Sources of contamination: alfalfa, grains, and fish meal.
[d]Sources of contamination: soy oil and fish meal.
[e]All values were corrected for percent recovery.

Appendix K. Sentinel Animal Program

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Table K-1. Laboratory Methods and Agents Tested for in the Sentinel Animal Program K-2

K.1. Methods

Rodents used in the National Toxicology Program are produced in optimally clean facilities to eliminate potential pathogens that may affect study results. The Sentinel Animal Program is part of the periodic monitoring of animal health that occurs during the evaluation of test agents. Under this program, the disease state of the rodents is monitored via sera or feces from extra (sentinel) or dosed animals in the study rooms. The sentinel animals and the study animals are subject to identical environmental conditions. Furthermore, the sentinel animals come from the same production source and weanling groups as the animals used for the studies of test agents.

Blood samples were collected and allowed to clot and the serum was separated. All samples were processed appropriately with serology testing performed by IDEXX BioResearch [formerly Research Animal Diagnostic Laboratory (RADIL), University of Missouri], Columbia, MO, for determination of the presence of pathogens. The laboratory methods and agents for which testing was performed are tabulated below; the times at which samples were collected during the studies are also listed.

Blood was collected from five rats per sex per time point except for the following:

- 2-year study, arrival collection: 10 females
- 2-year study, 4-week collection: 10 females
- 2-year study, 12-month collection: six females
- 2-year study, end of study collection: 10 males and 10 females

Table K-1. Laboratory Methods and Agents Tested for in the Sentinel Animal Program

Method and Test	Time of Collection
Twenty-eight-day Study	
Multiplex Fluorescent Immunoassay	
H-1 (Toolan's H-1 virus)	Study termination
KRV (Kilham's rat virus)	Study termination
Mycoplasma pulmonis	Study termination
PVM (pneumonia virus of mice)	Study termination
RCV/SDA (rat coronavirus/sialodacryoadenitis virus)	Study termination
RMV (rat minute virus)	Study termination
RPV (rat parvovirus)	Study termination
RTV (rat theilovirus)	Study termination
Sendai	Study termination
TMEV (Theiler's murine encephalomyelitis virus)	Study termination
Two-year Study	
Multiplex Fluorescent Immunoassay	
H-1	Arrival[a], 4 weeks[b], 9 weeks[c], 6, 12, and 18 months, study termination

Method and Test	Time of Collection
KRV	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
M. pulmonis	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
PVM	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
RCV/SDA	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
RMV	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
RPV	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
RTV	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
Sendai	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination
TMEV	Arrival, 4 weeks, 9 weeks, 6, 12, and 18 months, study termination

[a]Age-matched nonpregnant females.
[b]Time-mated females that did not have a litter.
[c]Offspring, 3 weeks post weaning.

K.2. Results

All test results were negative.

Appendix L. Peer Reviews of the Draft NTP Technical Reports on Cell Phone Radiofrequency Radiation

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L.1. Introduction

The peer review of the Draft NTP Technical Reports on Cell Phone Radiofrequency Radiation was convened March 26 to 28, 2018 in Rodbell Auditorium, Rall Building, National Institute of Environmental Health Sciences (NIEHS), Research Triangle Park, North Carolina. Dr. David Eaton served as chair. Other peer-review panel members in attendance were Drs. Rick Adler, Lydia Andrews-Jones, Frank Barnes, J. Mark Cline, George Corcoran, Susan Felter, Jack Harkema, Wolfgang Kaufmann, Asimina Kiourti, James Lin, Tyler Malys, Matthias Rinke, and Laurence Whiteley, and Ms. Kamala Pant. Dr. Donald Stump attended as the NTP Board of Scientific Counselors liaison. Interested members of the public attended the meeting in person or watched the proceedings via webcast.

Dr. Eaton welcomed everyone to the meeting and asked all in-person attendees to introduce themselves. Dr. John Bucher welcomed participants, thanked the panel, and provided an orientation to the 3-day meeting. Designated Federal Official Dr. Mary Wolfe read the conflict of interest statement and asked panel members to sign updated conflict of interest forms. Dr. Eaton presented the meeting format, with Day 1 devoted to the technical aspects of the radiofrequency radiation (RFR) exposure facility, Day 2 addressing the mouse studies, and Day 3 covering the rat studies. Slide presentations for the meeting are available on the NTP website (https://ntp.niehs.nih.gov/go/Presentations_RFR).

L.2. Attendees

Peer-Review Panel Chair

David Eaton, University of Washington

Peer-Review Panel 1

Provided consultation on the reverberation chamber exposure system

Frank Barnes, University of Colorado (retired)

Asimina Kiourti, The Ohio State University (present for Days 1 and 2)

James Lin, University of Illinois at Chicago

Peer-Review Panel 2

Provided input on study findings and voted on NTP's draft conclusions

Rick Adler, GlaxoSmithKline

Lydia Andrews-Jones, Allergan, Inc.

J. Mark Cline, Wake Forest School of Medicine

George Corcoran, Wayne State University

Susan Felter, Procter & Gamble

Jack Harkema, Michigan State University

Wolfgang Kaufmann, Merck (retired)

Tyler Malys, Data Management Services

Kamala Pant, BioReliance

Matthias Rinke, Bayer Pharma AG (retired)

Laurence Whiteley, Pfizer

Technical Experts

Myles Capstick, IT'IS Foundation

Niels Kuster, IT'IS Foundation

John Ladbury, National Institute of Standards and Technology

L.3. Panel 1: Peer Review of Exposure System for NTP Studies on Cell Phone RFR

L.3.1. Charge

Dr. Chad Blystone presented the Day 1 charge to the panel: to assess the reverberation chamber technology for evaluating the effects of cell phone RFR exposure in rats and mice.

L.3.2. Nomination, NTP's Considerations for Toxicological Evaluation of Radiofrequency Radiation Exposure in Rodents, and Background on Exposure System Selection

Dr. Michael Wyde described the NTP nomination of cell phone RFR exposure by the U.S. Food and Drug Administration (FDA) in 1999. The nomination was based on widespread and expanding human exposure, with little known about potential long-term health effects and insufficient data to assess risk to human health. The FDA and the Federal Communications Commission (FCC) share regulatory responsibility for RFR.

Dr. Wyde provided information about the background of the program, including establishment of research collaborations with RFR experts at the National Institute of Standards and Technology (NIST) and the IT'IS Foundation in Zurich, Switzerland. The IIT Research Institute (IITRI) in Chicago was chosen as the study laboratory. He discussed previous RFR toxicology studies and the selection of the exposure system for the NTP studies: frequencies of 900 MHz (rat) and 1,900 MHz (mouse) with both Global System for Mobile communications (GSM) and Code Division Multiple Access (CDMA) modulations, reflecting the standards in use when the study began. He described the reverberation chamber exposure system designed for the initiative.

Twenty-one reverberation chambers were constructed in Switzerland: seven each for mice, male rats, and female rats; male and female rats were separated due to weight differences between the

sexes. For mice, male rats, and female rats, each group had separate low, medium, and high dosage chambers for the GSM and CDMA modulations, plus one common control chamber. Dosage for RFR is measured as specific absorption rate (SAR).

The toxicology and carcinogenicity studies, consisting of three phases, were conducted on B6C3F1/N mice and Hsd:Harlan Sprague Dawley® SD® rats.

- 5-day thermal pilot studies at SARs of 4 to 12 Watts/kilogram (W/kg) in young and aged mice and rats and pregnant rats (10 studies) – presented on Day 1
- 28-day prechronic toxicology studies – presented on Days 2 (mice) and 3 (rats)
- 2-year toxicology and carcinogenicity studies – presented on Days 2 (mice) and 3 (rats)

In all studies, daily exposure to RFR in the reverberation chambers totaled 9 hours, 10 minutes per day — 18 hours, 20 minutes per day in 10-minute on-off cycles. The system generating the signals ran continuously, alternating exposure to the GSM and CDMA groups.

L.3.2.1. Questions for Clarification

Dr. Harkema asked whether the study design had been flexible, given the project would be lengthy and inevitably the technology would change. Dr. Wyde said the program was locked into the technologies in use at the time, because switching during the course of the studies would have been very expensive.

Dr. Cline asked about the provenance of the cell phone usage information presented in Dr. Wyde's slides. Dr. Wyde said that most of the information had come from surveys. Dr. Cline also asked if the animals could perceive whether the machine was on or off and what kind of emissions were perceptible with the exposure. Dr. Wyde deferred this question for discussion during the toxicology portion of the studies.

Dr. Eaton asked about the involvement of light-cycle circadian rhythms in the exposure schedules, noting that mice and rats are nocturnal animals. Dr. Wyde described the two husbandry periods: one in the early morning and one in the afternoon. The exposures continued throughout the night, and circadian rhythms were not taken into account.

Dr. Lin asked about the presence of mechanical noise, particularly as related to stirrers or paddles. He asked if the stirrers were turned on in the control chambers. Dr. Wyde confirmed the stirrers were turned on in the control chambers. Dr. Lin asked about the sequence of the exposures. Dr. Wyde explained that the system that generated the signals alternated between GSM and CDMA, but all animals were exposed to only one of the modulations.

Dr. Barnes asked about the statistical variation of rodent exposure between the chambers. Dr. Wyde deferred the answer to Dr. Myles Capstick's talk.

L.3.3. Reverberation Chamber System for RFR Exposures

Dr. Capstick from the IT'IS Foundation briefed the panel on the physical and environmental design of the reverberation chambers. Requirements included:

- Ability to expose large numbers of rodents

- Ability to expose to a high SAR up to 20 hours per day
- Animals to be unconstrained and housed in standard laboratory cages
- Food and water to be available on demand
- Excellent field and SAR homogeneity
- Detailed dosimetry (numerical and experimental)
- Ability to discern a possible dose response
- Third-party verification of the correct operation of the system

Several elements were involved in the rationale for the selection of the exposure, including frequency, modulation, and extremely low frequency envelope. Dr. Capstick described the GSM and CDMA modulation methods. The reverberation chambers were described, including:

- Two mode stirrers per chamber to achieve high field homogeneity and isotropy (including stirrer speeds)
- Standard gain antennas
- Air flow system
- Chamber design
- Lighting (per specific NTP requirements)
- Chamber field uniformity
- Exposure field uncertainty
- Noise
- Air handling
- Drinking water provision/Automatic watering system
- Stirrers and sensors
- Control equipment and amplifiers
- Data acquisition

Details on aspects of the reverberation chamber listed above are included in Dr. Capstick's online presentation[n]. The constructed chambers were shipped from Switzerland to Chicago, where they were installed in a specially designed facility.

L.3.3.1. Questions for Clarification

Dr. Whiteley asked whether the animals were rotated in the caging. Dr. Capstick said that, as the animals moved around the cages, any inhomogeneity was evened out. The cages were rotated twice per week. Dr. Whiteley asked whether the 10 minutes on, 10 minutes off approach was used for a biological reason. Dr. Capstick said previous studies had shown the intermittency of exposure was an important factor biologically. Dr. Kuster elaborated on the prior studies.

[n]The slides and video of Dr. Capstick's presentation on the reverberation chamber system for RFR exposures are available at https://ntp.niehs.nih.gov/go/Presentations_RFR (slides) and https://doi.org/10.22427/NTP-VIDEO-47 (video). Web links updated September 24, 2018.

Dr. Lin asked for more information regarding the exposure alternation. Dr. Capstick explained that, for 10 minutes, the energy was sent to the GSM chambers, and in the following 10 minutes, it was sent to the CDMA chambers. The chambers and their exposures were separate.

Dr. Lin noted that the historical data had been gathered in conditions using fluorescent lighting, as opposed to the incandescent lighting chosen for the NTP experiments. He considered the different lighting sources weakened the comparison of this study with historical studies. Dr. Bucher responded that the issue highlights a perplexing aspect of the study when trying to bring a historical perspective to interpreting the tumor data. He noted several of the differences between the current study and previous studies, including lighting, food, housing, and exposure methods.

Dr. Harkema asked about the phantoms and activity of the animals affecting the dose they received. Dr. Capstick deferred the answer to the dosimetry talk. Dr. Harkema also commented about the lighting, noting that lighting studies on plants by other researchers are ongoing.

Dr. Felter asked about the basis for choosing the different radiofrequencies for mice versus rats. She asked how frequency selection applied to animals of different sizes. Dr. Capstick deferred the answer to the dosimetry talk.

Dr. Kiourti asked about the statistical variation of animal sizes and weights. Dr. Capstick deferred the answer to the dosimetry talk.

Dr. Cline asked Dr. Capstick to elaborate on the ambient noise within the rats' hearing range. Dr. Capstick said that the GSM noise was measured, and no components were above 14 kHz. He said that high-frequency noise emanating from the air conditioning equipment was not measured. Efforts were made to keep the stirrers well lubricated to minimize potential noise.

Dr. Lin asked about the GSM noise, and how it was transmitted into the chambers. Dr. Capstick replied the GSM noise was generated inside the chamber, but its origin was unclear, and efforts to dampen it were unsuccessful. Dr. Lin wondered if the noise was instead introduced from the electronics and power-transfer systems.

L.3.4. Dosimetric Considerations for Rodents Exposed in Reverberation Chambers

Dr. Capstick briefed the panel on dosimetry used in the cell phone RFR studies.

Dosimetry in the fields of health physics and radiation protection is the measurement, calculation, and assessment of the internal exposure to the body, expressed in Watts per kilogram (W/kg). Directly measuring SAR in a subject, human or animal, is not possible, so SAR is calculated using numerical simulations and is validated in homogenous experimental phantoms. High-resolution, anatomical models were used to determine numerical dosimetry, with tissue parameters based on published databases. Ultimately, the appropriate frequencies were determined to be 1,900 MHz for mice and 900 MHz for rats to obtain a more uniform SAR distribution. Dosimetry in the reverberation chambers was calculated based on generation of a homogeneous, isotropic field, using Rayleigh-distributed, temporal variations. Exposure-environment measures used representations employing the random plane-wave method and the 12 plane-wave method.

An automated watering system was designed to ensure that no energy was absorbed by water, which would cause a dose-dependent elevation in drinking water temperature. Also, the system was designed to avoid increased SAR or RF burns to the animals, which could deter them from drinking.

The isotropic field employed ensured minimum variation in whole-body SAR with posture. Variation in organ-specific SAR was also taken into account. Dr. Capstick also presented details regarding uncertainty and variability estimates. Full details on the dosimetric considerations are included in Dr. Capstick's online presentation[o].

L.3.4.1. Questions for Clarification

Dr. Eaton expressed continued confusion about the units of SARs. Dr. Capstick explained that SAR is measured in watts per kilogram (W/kg) and that the limit for human exposure is SAR averaged over a 1 gram (g) or 10 g cube. He described how SAR was calculated in mice and rats over smaller cubes scaled to the relative adult weights. He explained that the measures in decibels (dB) used is a logarithmic ratio that can be related to either the whole-body average or the peak SAR. He explained how SAR sensitivity, the SAR per unit of electric field strength, is calculated.

Dr. Eaton said that although not the focus of the current studies, the data would be used for risk assessment at some point. He asked if anyone had derived modeled dosimetry in humans based on behaviors. Dr. Capstick said much work was ongoing in that area, particularly on exposures in children and device placement on the body.

Dr. Felter asked for clarification about how mass affects the measurement of SAR and if surface area has an effect. Dr. Capstick explained the concept of whole-body average SAR as an average over the mass. So, the larger the animal, for a given whole-body SAR, the more power is absorbed. He described the difference between organ-based SAR (oSAR) and whole-body SAR. His answer to the question about surface area explained that the ratio of surface area to total mass affects an animal's thermal regulation — a larger ratio means the animal can cool itself more quickly.

Dr. Kuster remarked that the study was run under the assumption that the fields locally induced in the tissue are the biologically relevant parameters, not the total absorbed power or whole-body averaged exposure. He noted that SAR and the square of the E-field are directly related, whereas the square of the local H-field (magnetic field) is sufficiently related for uniform exposures. As little is known about the radiofrequency sensitivity of specific tissues, the exposure was optimized for maximally uniform local E-field and H-field exposures.

Dr. Lin asked for more detail on organ-based SAR and whole-body-based SAR as it related to the figures Dr. Capstick presented on individual organ SAR differentials from whole body.

Dr. Melnick, retired NIEHS/NTP scientist and public attendee, was recognized by the chair for a question. He was concerned about the exposure of some of the sub-tissues in the heart. Dr.

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[o]The slides and video of Dr. Capstick's presentation on the dosimetric considerations are available at https://ntp.niehs.nih.gov/go/Presentations_RFR (slides) and https://doi.org/10.22427/NTP-VIDEO-48 (video). Web links updated September 24, 2018.

Kuster explained that the anatomical models provided a good proxy of exposure for different body regions and tissues, but no effort was made in this study to examine sub-tissues.

Dr. Felter asked about the pups, which were housed with the mother until weaning. She indicated her understanding was, when pups are clumped, their SAR can be increased, but when pups are apart, their exposure is similar to that of the dam. She asked why their estimated exposures would not be higher, given their much smaller body weight. Dr. Capstick explained that in terms of body weight and length, the pups are on an upward curve and the dams are on a downward curve, ending up at approximately the same SAR sensitivity.

L.3.5. Reverberation Chamber System Validation and Verification

John Ladbury from NIST briefed the panel on validation and verification of the reverberation chamber system. He provided background information on NIST and described the ideal characteristics of a reverberation chamber. The validation and verification plan emphasized uniformity of temperature in the phantoms, probe field, and antenna power. Validations were performed in 2007, 2012, and 2015. The standard deviation for the loaded chamber field uniformity was 1.3 dB. Calibration was performed with radiofrequency field probes. Signal quality was within standard parameters for communications standards. Full details on the reverberation chamber system validation and verification are included in Mr. Ladbury's online presentation[p].

L.3.5.1. Questions for Clarification

After remarking about the robustness of reverberation chambers, Dr. Lin asked why the reverberation chambers had not been built in Chicago. Dr. Bucher explained that the system was assembled through contractual arrangements with various organizations, including IT'IS. Mr. Ladbury noted, although commercial reverberation chambers are available, they are designed primarily for electronics testing, and at the time, none were available for biological testing.

Dr. Cline asked if he was correct that no measurements were taken in the control chambers. Mr. Ladbury confirmed that no measurements were made in the control chambers. Dr. Capstick noted field probes were placed inside the control chambers and noise levels of the measurement system were recorded throughout the study.

L.3.6. Thermal Pilot Studies of Cell Phone Radiofrequency Radiation

Dr. Wyde presented information about the 5-day thermal pilot studies at SARs of 4 to 12 W/kg in mice, young and aged rats, and pregnant rats — 10 studies. The studies were designed to evaluate a wide range of SARs to determine the threshold for potential thermal effects of cell phone RFR, the impact of animal size and pregnancy on body temperature, and the potential effects of RFR exposure on pregnancy in rats. Body temperatures were collected via implanted microchips at multiple time points over 5 days.

[p]The slides and video of Mr. Ladbury's presentation on the validation and verification of the reverberation chamber system are available at https://ntp.niehs.nih.gov/go/Presentations_RFR (slides) and https://doi.org/10.22427/NTP-VIDEO-49 (video). Web links updated September 24, 2018.

In the mouse studies:

- No thermal effects were observed at SARs up to 12 W/kg regardless of age, sex, or modulation.
- 5, 10, and 15 W/kg were selected for 28-day studies.

In the rat studies:

- Lethal effects and excessive increases in body temperatures were observed at 10 and 12 W/kg.
- Increased early resorptions and decreased body-weight gain in pregnant dams were observed at 12 W/kg GSM.
- Based on those data, SARs of ≥10 W/kg were not recommended for further study in rats.
- 3, 6, and 9 W/kg were selected for 28-day studies.

L.3.6.1. Questions for Clarification

Dr. Adler asked whether body temperatures were measured at night, when rodents are eating, metabolically more active, and likely to have diurnal variation in body temperature, or only during the light cycle. Dr. Wyde said they were measured only during the day, and all measurements were made within 2–3 minutes of system shutdown to minimize the effect of heat loss. The temperature decay rate of an animal with elevated temperature was not independently measured, although some preliminary studies with the thermal sensors were done. Dr. Adler asked if any other physical parameters were measured, such as respiratory rate. Dr. Wyde said the goal was to examine only gross effects in body temperature, body weight, and survival, with no provision for histopathology. Some additional measures were performed in the 28-day studies. Dr. Adler pointed out that rodents acclimate quickly to environmental changes so that differences occurring at 5 days might not be detected at 28 days. The pharmaceutical industry prefers these measurements be done in 5-day studies because they can understand what additional systems are perturbed by the external influence before the animals reach steady state.

Dr. Felter asked why the 10 minutes on, 10 minutes off standard was chosen, and whether any experiments had been conducted with longer exposures. Dr. Wyde said that no other exposure lengths had been explored. Ten minutes was considered sufficient to allow for thermal regulation. The intermittent exposure was considered important to determining response to the RFR exposure, while the 10-minute exposure was somewhat arbitrary.

Dr. Kiourti asked about the implanted temperature sensors and how they communicated with the reader. Dr. Capstick confirmed that the system was radio-frequency identification.

Dr. Lin asked what other temperatures would be monitored if the experiment were to be repeated. Dr. Wyde said that NTP is considering some follow-up studies using data loggers to collect information in real time during the exposures.

Dr. Barnes asked if distortion of the fields with the sensor under the skin were possible. Dr. Kuster said that had been evaluated, and no distortion or interference with the measurements was apparent.

Dr. Rinke asked if the same rodent strains were used in all studies. Dr. Wyde said yes.

Dr. Gamboa da Costa from FDA asked whether some important information regarding the temperature of the main organs might have been missed. Dr. Wyde replied that was possible. Dr. Lin pointed out that the hottest spot was at the tail, so anatomy should be considered when determining where to implant the temperature sensor. Dr. Capstick noted that in his previous presentation showing the tail as a hot spot, it was the SAR distribution that had been modeled, which is not necessarily directly related to temperature distribution in the animal.

Dr. Barnes asked if use of an infrared camera had been considered. Dr. Kuster said that some investigators had tried to use thermal cameras in dosimetry, but they lacked the needed sensitivity.

L.3.7. Oral Public Comments on Technical Aspects of the NTP Exposure System

Dr. Eaton identified the written public comments received and presented a list of those public commenters. He described the format for presenting the oral public comments; five public commenters made oral comments on the exposure system.

Theodora Scarato, a private citizen, addressed the unique vulnerability of children to RFR, and the ever-increasing combined RFR exposures to the public. Cell phone use is now widespread, and they and other wireless devices are often used near the body. Pregnant women and children are exposed at much higher levels. Children, with thinner skulls and smaller heads, are much more vulnerable to RFR energy deposition. Published research modeling children's exposure shows that children's heads and brains are proportionally more exposed compared to adults. The use of multiple devices can increase SAR, as does the presence of metal inside or outside the body. The public is unaware that phones and wireless devices emit radiation, or that health concerns are associated with the exposures.

Dr. Olga Naidenko presented comments on behalf of the Environmental Working Group (EWG). She expressed EWG's support for NTP and NIEHS for having embarked on the absolutely essential cell phone RFR study and its appreciation that the first part of the study had been completed. EWG believes the exposures are relevant to people and to the exposures people are facing today. The study was conducted in 2G technology, whereas today 3G and 4G are in use, with 5G being rolled out. EWG's position is that the science currently in hand must prevail. EWG believes the next generation of exposure studies should increase emphasis on biological factors. The recent National Institute on Drug Abuse's study is a good example of research designed to elucidate short-term, immediate, and subtle effects such as changes in metabolism and in calcium-channel transmission and impacts on blood-glucose metabolism.

Dr. Devra Davis presented comments on behalf of the Environmental Health Trust (EHT). The exposure system is an important, positive study that was well executed under difficult conditions. She noted, however, that the exposure system used does not reflect current exposures. Historical controls are not relevant in the study; the only relevant controls are those from the study, and using historical controls from other NTP studies for comparison is a mistake. With respect to SAR values, basing guideline limits on average tissue volume data is inaccurate, as body parts are not cubes. She pointed out several issues for disagreement with NTP's study: The NTP study does not account for the multiple exposures experienced every day and cannot clarify what is happening in the occupational workforce, where RFR levels are much higher. The NTP study

will not be relevant to 5G. She believes the whole-body approach taken in the NTP study is appropriate. French studies of exposures related to phones placed beside the body have shown much higher levels than those permitted by the FCC.

Dr. Kuster asked Dr. Davis why she believes the NTP study did not cover multiple exposures, noting that the local exposure levels used were higher than actual exposures from multiple exposure sources, even higher than occupational exposures. Dr. Davis said that current smartphones can have as many as four different antennas operating simultaneously, and that the synergy that occurs when electric and magnetic fields are combined with radiofrequencies or chemicals cannot be evaluated in a study like the NTP study. The real world is complicated, and studies such as the NTP study cannot capture that complexity. She stated the use of the technology has exploded and the capacity within experimental models to fully approximate human exposures is not available, noting little information is available about the impact on human health today and in the future. Children are routinely exposed to RFR devices in close proximity.

Kevin Mottus spoke for the California Brain Tumor Association, which supports individuals who have developed brain tumors from cell phone radiation. NTP is to be thanked for embarking on the study and following it through so conscientiously. The study reflects what is being seen in the real world, particularly DNA damage related to the carcinogenic effect. The association works not only with brain tumor sufferers, but also with people who have become sick from RFR and microwave exposures. The NTP study and the Ramazzini study offer biological confirmation of the cellular effects observed in human studies for years. Wireless should be reclassified as a Class 1 human carcinogen. The NTP study shows a clear increase in brain tumors in the areas that get the most cell phone use — the frontal lobe, cerebellum, and temporal lobe. Brain cancer is now the number one cancer in children 15 to 19 years old and is one of the top three cancers up to age 39 years, reflecting an epidemic. Mr. Mottus was critical of FDA's critiques of the NTP study. Addition of 5G high-frequency transmission on top of low-frequency 3G and 4G will result in more disease. The use of multiple devices and frequencies will result in a microwaving of the U.S. population. He stated the FCC is hiding health effects of exposures and exempting new technologies from environmental review. He believes FCC is an industry-compromised organization and that NTP should take a stand against such compromise and insulate itself from industry influence.

Dr. Paul Heroux from McGill University was the final public commenter. He believes the NTP reverberation chamber delivered the test animals a stable challenge over a specific, integrated time frame. The variation of ± 2.5 dB quoted in the report, although excellent performance, could have been reduced by using larger chambers, so that the objects would occupy less of the total chamber volume. The study shows its age by its overemphasis on heat. The finding of lower survival in the non-exposed animals is not simply an artifact and has been borne out in other large animal studies. Another interesting aspect is that the survival advantage effects are stronger in males than in females. The NTP studies do not mention control of the background extremely low frequency environment. The effects of GSM and CDMA differ, so the details of the exposure are significant and important.

L.3.8. Peer Review Comments on the Reverberation Chamber Exposure System

Dr. Barnes, the first peer reviewer, felt the study was very well done in terms of accomplishing what it set out to do. With SAR as the critical parameter to define, NTP did a very good job of determining the exposure distributions and confirming the average values were as stated. Those elements were well tested and monitored throughout the studies. If the studies were designed today, however, Dr. Barnes said a variety of additional experiments could be conducted and additional parameters could be controlled. For example, translations from physics to chemistry and chemistry to biology could be built into future studies. Examining problems of feed-forward and feedback loops in more detail should be incorporated. Overall, NTP is to be complimented on a very thorough study. Dr. Bucher asked Dr. Barnes to elaborate on the concept of feed-forward, as NTP is interested in improving its studies. Dr. Barnes said feed-forward is related to elaborate communication systems inside the body, such as acupuncture points. An exciting development is the opportunity to convert electric and magnetic signals to biological signals in the chemical realm that the body already knows how to use.

Dr. Lin, the second peer reviewer, applauded NTP and NIEHS for having conducted the cell phone RFR studies because for the U.S. government to conduct such research and not leave it entirely to industry is important. He noted we are exposed to more and more RFR every day. The NTP study was the largest of its kind, was expensive, and took a long time to complete. The study showed that prolonged exposure to RFR levels, roughly three times current RFR exposure guidelines, could lead to tumor development, particularly schwannomas in the heart tissue of rats, and to some degree gliomas in the brain. He said the reverberation chamber (RC) apparently was selected *a priori* for the project and whether it is the optimal technology for the project or alternative, competing technologies were considered is unclear.

Descriptions in the report of what was implemented are clear and measurement techniques are accurate, within limitations. Although RCs are generally acknowledged to provide substantially uniform, average-field distributions in the absence of a test object, the bodies of rats and mice would be major radiofrequency energy absorbers, resulting in a very different interaction mechanism for fields inside the RCs. Free-roaming animals inside the cages would make the exposure field substantially less uniform compared to an empty RC. Although much effort was expended to achieve RF-field homogeneity and so-called “isotropy,” whether the RC approach had any advantage over simpler approaches is unclear.

He voiced concerns that mixing dB and linear scales is confusing and felt that describing uniformity using average-field distribution would have been more appropriate. He pointed out that field distortions introduced by the watering system do not appear to have been quantified.

Dr. Lin believes the use of liquid-filled, round, plastic bottles for the measures of uniformities in the RCs does not provide realistic simulations of animals’ body shapes, resulting in inaccurate measures of SAR variations. He speculated that differences in resonant absorption might account for the different observed biological responses in the rats and mice, and wondered what influence, if any, the differential whole-body SAR (wbSAR), peak spatial SAR (psSAR) or organ-specific SAR (oSAR) could have had on observed cancer incidence.

He believes the methodologies, paradigms, and protocols used in the studies were reasonable, but whether the studies are intended for cell phone or base-station RF exposures, and whether they represent near-field or far-field exposure scenarios, is unclear. The use of temporal and spatial

averaging ignores anatomy-related responses of the animal as functions of time or age to RF SAR and SAR distribution. He noted the apparent lack of provision for physiological monitoring or animal behavioral observation during the 2-year studies. He raised concerns about the sonic noise in the chambers. Ear or tympanic temperature should have been measured periodically throughout the study to monitor core temperature. Seeing the SAR-dependent reports of schwannomas in rats is perplexing. The experiments specified whole-body exposure, and wbSAR was the key metric for exposure, but a correlation study of psSAR or oSAR with total observed primary tumors should be included in the report.

Dr. Bucher noted that one objective of the peer review is to identify how the report could be improved in communicating several of the issues Dr. Lin had raised. He said that some of the information Dr. Lin suggests has been in the day's presentations, although not currently in the report, and welcomed suggestions for how best to encapsulate some of that information, particularly with respect to psSARs.

Dr. Kiourti, the third peer reviewer, congratulated NTP on a very thorough study. She had no major comments regarding technical aspects of the study. She asked how NTP could catch up with the technology, in general. Although the study delivers 100% of what had been promised, 2G is not even used today. She noted that the report should clarify that "exposures cycled between modulations every 10 minutes" means the cycling was between on and off for a given modulation, not cycling between the different modulations. She asked for more details about where the RF sensors were placed and why those locations were selected. She asked how the specific environmental conditions had been chosen and whether any differences would have affected the study's results. Although the animals were freely moving, they were still caged for 2 years, and she wondered whether that could have compounded stress. She asked for more details on the design of the antennas used, cage rotation, and how the NIST and IT'IS phantom studies compared.

L.3.9. Panel Discussion and Recommendations for Reporting of Chamber Design and Performance and Dosimetry Considerations

Dr. Eaton introduced the panel discussion section of the session. He said that, as a biologist, he appreciated the presentations detailing how the exposures were conducted. With the technologies having changed considerably since the studies were conducted, he asked whether the biological effects are likely to be better or worse now. Dr. Barnes replied that how the power is distributed as a function of frequency differs between the older technologies used in the study and the upcoming 5G. How to go from the physics to the chemistry to the biology can change, but there could be common responses, and elucidating the exact mechanisms affecting biology is very challenging.

Dr. Kuster addressed the question regarding bottles versus more anatomical phantoms. He stated that the numerical study provides the dosimetry and the purpose of the experimental study with the bottle phantoms was to validate the numerical dosimetry. First, the presence of any coupling between phantoms had been carefully evaluated, and based on that information, the cages were separated to exclude coupling. Thus, how the energy was scattered did not matter; the only concern was how the energy was absorbed. The bottle phantoms were optimized to absorb the same amount of energy as the rats absorbed. Dr. Kuster said that the dosimetry information has only recently been published. Dr. Lin noted that the animals' posture made a difference in

dosimetry, and therefore a round bottle was not an animal-shaped body. Dr. Kuster explained the bottles were used to validate the numerical dosimetry and the uniformity of exposure throughout the chamber, and were not affected by the presence of the animals. The differences caused by the postures of the anatomical phantoms are addressed in the numerical dosimetry. Dr. Lin noted additional field measurements after installation of the watering system were not indicated. Dr. Kuster replied that the measurements were made at the end of the process, after everything had been installed. Dr. Lin said that the field inside the animal would depend on posture and geometry. Dr. Kuster agreed that that needed to be better explained in the report.

Dr. Cline mentioned that “dosimetry” is used incorrectly in the discussion. Dosimetry is the measurement of the dose, not a mathematical model of what the dose might be. Dr. Lin disagreed with that statement, noting the differences between ionizing and nonionizing radiation. Drs. Lin and Cline exchanged several comments on the point.

Dr. Lin added that, despite that 5G technology is being rolled out, 3G is still most relevant, as it remains what most people have in their pockets. He also discussed what the fundamental purpose and impacts of this study should be and the validity of this study.

Dr. Harkema asked if the design of the study (in 2007) was influenced or hindered in some way by constraining the timeline to a 2-year bioassay. Dr. Eaton added that NTP had been rather clairvoyant in the design of the study, as starting studies in utero was unheard of at the time, and now the importance of early-life exposures is recognized. Dr. Bucher pointed out that NTP tried to set the number of animals used to achieve maximum efficiency, with minimal animal use versus costs involved in increasing statistical power by adding population. He described some of the challenges associated with that approach.

Dr. Eaton recognized two public attendees: Dr. Davis from Environmental Health Trust and Dr. Melnick, retired NIEHS/NTP scientist. Dr. Davis commented on the issue of the relatively lower power of 5G, and whether it would result in fewer biological effects. She cited a study that reported the opposite effect — the weaker power but higher frequency was more biologically potent. Dr. Melnick noted that the objective of the NTP study, like all toxicology studies, was to test the null hypothesis. People were saying, “this is nonionizing radiation, there’s no possibility of adverse biological effect,” and therefore the study was designed to challenge that hypothesis. The assumption that no biological effect occurs holds for consideration of 5G technologies. With the current study having disproved the null hypothesis, testing the newer technology would be wise to determine if any health effects on the general population occur.

Dr. Eaton returned the discussion to consideration of the draft NTP reports. His sense was that the panel offered strong praise for the NTP program and for its designers and consultants for having constructed a very challenging exposure situation. He perceived no “fatal flaws” in terms of the exposures.

The panelists discussed uncertainties in the dose metric used in the studies. Dr. Felter alluded to the concept that the male rats experienced more effects because they are larger and pointed out that that should have resulted in lower exposure due to a larger body surface area. Dr. Barnes elaborated on the SAR dose metric and added that some additional properties were taken into account during dose measurement. Dr. Kuster noted the goal in the studies was to achieve uniform exposures of all tissues, to the extent possible. Dr. Felter said that the complicated nature of the dosing and dose metrics should be described in more detail in the reports.

Dr. Lin speculated about the best path forward in future studies. He noted that investing in a repeatable experiment might be appropriate before shifting the investigation to new technologies such as 5G.

The panelists and other experts discussed the issue of thermal versus non-thermal effects. Dr. Gamboa da Costa from the FDA urged caution about ascribing effects observed in the studies as non-thermal because monitoring temperature with fine granularity is difficult. Other panelists agreed that information to specifically rule out a thermal or non-thermal effect was insufficient. Dr. Barnes commented that a non-thermal effect is fairly ill defined.

Mr. Ladbury commented on the difficulty of moving the inquiry from reactive to predictive in terms of assessing the impact of newer technologies. He felt that the field would remain reactive to changing technologies for many years to come.

Dr. Whiteley stated that before studying 5G, gaining a better understanding of the dose-response biology of effects observed in the current studies, in the specific cell types that were affected, would be advisable.

Although panelists referred to the Ramazzini Institute study, Dr. Bucher cautioned that this meeting's intent is to peer review the NTP studies, so comparison to another study is probably not appropriate at the time. Again, an emphasis on clearly explaining dosimetry was brought up because the exposure system for the Ramazzini study was different. Concern was expressed that the broader field would make incorrect comparisons to this study if exposure were not clearly defined.

Day 1 of the proceedings was adjourned at 5:05 p.m.

Day 2: March 27, 2018

Dr. Eaton welcomed everyone to Day 2 of the meeting and asked all attendees to introduce themselves. Designated Federal Official Dr. Mary Wolfe read the conflict of interest statement. Dr. Eaton presented the meeting format for Days 2 and 3.

L.4. Panel 2: Peer Review of Draft NTP Technical Reports on Cell Phone RFR

L.4.1. NTP's Toxicology and Carcinogenesis Studies: Experimental Design, Statistical Analyses, Genetic Toxicology Testing, and Hazard Determinations

Dr. Blystone provided an overview of the methodologies and approaches used in standard NTP chronic studies and in the cell phone RFR studies, including design considerations, and the animal models and numbers used: Hsd:Sprague Dawley® SD® rats and B6C3F1/N mice, 90 animals per sex per group. For these RFR studies, the exposure language differed from that for typical toxicology studies. He described several elements of the statistical analyses used in the studies, including the historical controls. He informed the panel about the NTP Levels of Evidence of Carcinogenic Activity, which form the basis for conclusions.

L.4.1.1. Questions for Clarification

Dr. Harkema asked if historical control data are available from the contractor, IITRI. Dr. Blystone said no. Dr. Harkema asked about the low and medium doses and what "additional lower doses were spaced accordingly" meant — according to what, he inquired. Dr. Blystone explained the variety of factors taken into account, including capturing a wide dose range, appropriately evaluating hazard identification, and considering route of exposure. Dr. Harkema asked how the low and medium doses were scientifically determined. Dr. Wyde explained that, due to system feasibility, only three exposure groups were an option. Extending the exposure range to 6 W/kg enabled NTP to challenge animals on a thermal basis, and extending the range to 1.5 W/kg brought the exposures to a relevant level near the FCC regulatory limit. Dr. Bucher followed by explaining that dropping the exposure range to even lower levels would have diminished the likelihood of detecting effects.

Dr. Harkema asked for clarification on the difference between "some" and "equivocal" in the levels of evidence. Dr. Blystone addressed the issue, noting that the "bright line" between the two was whether an observed effect was considered associated with the exposure. Responding to a question from Dr. Eaton, he added that historical control data and discussions among staff are used when making those decisions, but the determination ultimately relies on discerning positive versus negative effects.

Beginning with a question from Dr. Felter, a discussion ensued regarding the historical controls, particularly for the rats, which consisted of four studies, and including the concurrent controls in the overall historical control incidences. Dr. Felter felt that the historical controls should have been kept separate from the concurrent controls. Dr. Blystone stated that including the concurrent controls heavily weighted the historical control data for these studies, but both options were examined. Dr. Keith Shockley from NIEHS noted that the concurrent controls were used as part of the statistical testing, but the historical controls were not. Dr. Bucher said that in the next version of the reports, an appendix will delineate the studies included in the historical controls. The panel also posed questions regarding the use of a common control for the studies, which Dr. Wyde explained was due to space constraints and the cost of additional chambers. Dr. Bucher followed by saying that, if the studies were done again, a second control group would probably be included.

Dr. Harkema asked for more detail on the historical controls used in the mouse studies. Dr. Blystone said 11 studies, including the concurrent controls, were included. Dr. Lin said the historical controls were not relevant to the current studies due to differences in exposure, such as different lighting and different study designs. Dr. Harkema stated that, although the historical controls might not have been the most appropriate, they are still informative. Dr. Barnes pointed out that the assumption is that we are looking at a linear system with regard to dose response, but, in some sense, the historical controls are not free of exposure to RFR. He cautioned against treating historical controls as unexposed, as RFR probably was not measured and, therefore, they might not be true controls for comparison in these studies. Dr. Felter also cautioned against disregarding the historical control data, as they provide a wealth of information on variability in tumor response.

Dr. Rinke asked if the rodents used were from the same breeder or supplier as the historical controls were from. Dr. Bucher said that the rats were, and he believed that the mice were from the same breeder, although he was not certain.

Dr. Kaufmann asked what elements of neurobehavioral observations had been considered in the design of the studies. Dr. Blystone explained that, due to the constraints posed by the closed exposure chambers, detailed clinical observations were not possible. The nervous system was pathologically examined, with increased sectioning of the brain from three to seven slices.

L.4.2. Genetic Toxicology Studies in Mice and Rats Exposed to Radiofrequency Radiation

Ms. Kristine Witt briefed the panel on the genetic toxicology studies, describing the rationale for selecting the assays, the assay protocols (erythrocyte micronucleus assay, comet assay), the data analysis used, and how the data were interpreted. Subsets of mice and rats were assessed for genetic damage after 14 weeks (mice) or 19 weeks (rats) of exposure.

L.4.2.1. Questions for Clarification

Dr. Cline asked if comet assays had been performed on brain tissue. Ms. Witt replied that they had. Dr. Cline asked how the brain cell types were selected. Ms. Witt said that they selected the hippocampus, cerebellum, and frontal cortex, with no microscopic selection of particular cell types. Dr. Cline noted that several types of micronucleus tests are available and recommended that the assay be called the erythrocyte micronucleus assay in the report to avoid potential confusion.

Dr. Harkema asked how the brain sites for the comet assay were determined and how they were handled statistically. Ms. Witt said that each tissue type was considered independently and they were not combined for statistical analysis. She explained that the frontal cortex was selected because of the possibility of brain tumors, and the hippocampus and cerebellum were selected because they comprise large portions of the brain and cover a wide space for analysis. Dr. Harkema followed by asking if the comet assay was the most appropriate for comparisons to histopathology. Dr. Malarkey described the standard neurohistopathological evaluations and stated that there are no findings that correlate with the genetic toxicological findings. Dr. Bucher clarified that the animals taken for the comet assay were different from the animals used for interim histopathology.

Dr. Adler asked if the comet assay has a positive/negative threshold and whether a positive control was run. Ms. Witt said NTP animal studies do not have a positive control, but positive control slides with human cells exposed to a known genotoxic agent are run as an internal technical control. She added that the software does not delineate between positive and negative. No historical controls for the comet assay for these studies were included.

Dr. Lin asked whether other parts of the animals were assayed in addition to the neurological tissues. Ms. Witt said the liver and peripheral blood leukocytes also were assayed.

Relating to the positive predictive value of the erythrocyte micronucleus test, Dr. Eaton asked about the occurrence of false positives. Ms. Witt said that the last time a systematic review of the test was compared with a bioassay was 2000, which showed a 95% to 98% rate of positive predictivity. She noted the test has low sensitivity, but a positive response is meaningful, in both rats and mice. Ms. Witt is unaware of any chemical that induced micronuclei in vitro and does not induce carcinogenicity in vivo in 2-year studies.

Dr. Felter asked how the results from the comet assays were reported, in terms of positive, equivocal, and negative findings. She wondered what would be done if the data supported a trend in the opposite direction. She cited the example of a statistically significant decrease in the comet tail in the females in both modulations in the frontal cortex. Ms. Witt said that 2-sided trend tests are not conducted, so whether it was a statistically significant decrease could not be stated, although it might appear to be.

L.4.3. Pathology Peer Review Process for Two-year Studies of Cell Phone Radiofrequency Radiation

Dr. Amy Brix briefed the panel on the pathology peer review process used in the 2-year studies. She described the role of the study pathologist and outlined the steps in the pathology peer review process, including the Pathology Data Review (PDR), the audit of pathology specimens, the pathology quality assessment slide review, the Pathology Working Group (PWG), and the final steps to complete the process.

L.4.3.1. Questions for Clarification

Dr. Harkema noted that no place conducts the pathology process better than NTP, which sets the gold standard. He asked Dr. Brix to summarize the review process used with the lymphomas. She said that the study pathologist initially noted them in the report. During the PDR, items were flagged for review including the statistical tables, incidence tables, and anything unusually high or low in the controls. All lymphomas diagnosed in the mice were reviewed, and all tissues with neoplasms were automatically reviewed. That information was then given to the PWG. She said the conclusion did not change throughout the study. Dr. Harkema said it seems unusual to have two pathologists, one looking at males, one looking at females. Dr. Brix agreed, although it was necessary because of the size of this project. Dr. Harkema also asked where the study pathologist was located for these studies. Dr. Brix replied that IITRI used a subcontractor as a pathologist and did not have one on site.

Dr. Lin asked about blinding at the study pathologist and pathology review levels. Dr. Brix said that slides are not blinded at the study laboratory, because it is not considered as sensitive a read if something is seen when blinded. Slides are also not blinded during the quality assurance (QA) review. At the PWG level, blinding is used and PWG participants are unaware of the study or QA pathologist calls. Dr. Lin noted that at the study pathology level, the pathologist would have known whether a tissue came from exposed or control animals. Dr. Brix confirmed that impression and added that NTP follows the industry standard for such studies, and non-blinding is the most scientifically appropriate method. She said that the pathologists are evaluating a biologically complex system, and they must be able to compare treatment-related findings to control incidences to distinguish which findings actually differ. Dr. Lin and Dr. Brix exchanged several comments on the issue. Dr. Bucher noted that the argument is not unique to NTP, having persisted among pathologists for a long time. Dr. Harkema said that even pathologists do not take the issue lightly and the entire process has been rigorously reviewed. He believes that in this case, the peer review, which is the most unbiased, is at the correct level in the process. If any study pathologist bias were to occur, it would be caught at the peer review level.

Dr. Cline asked how the rest of the head, aside from the brain, was assessed. In particular, he wanted to know if the vestibular system and auditory nerve were included. Dr. Malarkey said they were not assessed in the mouse, but some exploration in the rat was conducted.

L.4.4. Oral Public Comments on Technical Aspects of NTP Studies in Rats and Mice

Dr. Eaton acknowledged the written public comments received and presented a list of those public commenters. He described the format for the oral public comments to be delivered. In the second session, nine oral public commenters on the NTP studies in rats and mice were accommodated.

Ms. Scarato drew a distinction between FCC human exposure limits and safety guidelines. Proper safety testing has never been completed on chronic, low-level exposure. The NTP findings of increased cancerous and pre-cancerous lesions confirm that the FCC limits are non-protective. The technical reports should include the regulatory limits of other countries and summarize that the FCC limits are far higher. Co-exposures should also be taken into account, with studies showing synergies included in the reports. The reports also should refer to studies addressing changes in the permeability of the blood-brain barrier related to cell phone use, decreases in brain cells resulting from prenatal exposures, and behavioral issues related to prenatal exposures. The reports should include information on worldwide governmental actions to reduce RF exposure. Maryland and California have acted to reduce RF exposures. The mouse technical report omitted NTP data presented in 2016 regarding DNA damage analysis and this data should be added back in. Similarly, reference to the conclusion by the World Health Organization's International Agency for Research on Cancer was included in the 2016 report but not in the 2018 draft technical report, and it should be added. Discussion of the Ramazzini studies should be added to the technical reports, as should the concordance of the observations of schwannoma in rats and lymphoma in mice, considering that we live in a world of multiple exposures. The U.S. government should act to limit public exposures.

Dr. Naidenko from the Environmental Working Group stated that NTP is in a unique position to study the biological effects of cell phone RFR exposures. She alluded to a company, Novocure, which has FDA approval to use electromagnetic fields (EMF) for treatment of glioblastoma. She described how EMF can impact biological systems, showcasing the extremely complex biology involved, including the effects investigated by NTP and considered by the peer review panel.

Dr. Davis spoke on behalf of colleagues at Hebrew University. She noted that this 3-day review was unprecedented. She appreciated the explication of the blinded pathology review. The NTP study is not a lifetime study, ending at 2 years, and 60% of all cancers in humans occur after age 60. The rodent studies end at the equivalent of age 60. She recommended using the NTP study's controls and not historical controls. She noted that the baseline rate of cardiac schwannoma was quite low, even in historical controls. She recommended reexamining the data on reproductive endpoints and birth weight impacts. She added several other detailed recommendations. She further discussed the Ramazzini study and presented relevant conclusions from the study. She presented data from several other recent studies, suggesting reproductive endpoint effects of RFR exposures and increasing rates of brain tumors in the United States.

Dr. Kuster said that Dr. Davis' comparison of exposures was "apples and bananas," and that the NTP study is conservative with respect to simulating the exposure, independent of usage. She agreed, but pointed out that phone testing methods vary, with many agencies testing them in a holster away from the body, which would reduce exposures and is an out-of-date method. The French, she observed, test phones in close proximity to the body.

The next oral public commenter was Dr. Marc Arazi from the Phonegate Alert Association in France, an organization devoted to sharing technical and scientific information on cell phone radiation and formulating safety recommendations. He commended NTP for using high SAR levels in its studies and found the results in several organs particularly important to understanding the risks associated with RFR on the whole body, not just the brain. He described a 2016 report on tests by the French government called Exposure to Radiofrequency and Child Health. Data showed that many of the most popular phones in the European market exceed regulatory RFR limits. Another new report showed that RFR sensitivity is a real and widespread illness. He emphasized that focusing on realistic use of cell phones by users and implementing simple measures to protect the billions of users in the world are necessary.

Dr. Annie Sasco is a former Unit Chief at the World Health Organization's International Agency for Research on Cancer and retired director of research at INSERM (Institut national de la santé et de la recherche médicale, the French National Institute of Health and Medical Research). At this review, she spoke on her own behalf. She described her background and education as a cancer epidemiologist. She said that over her 35-year career, the situation with regard to cancer had not really improved. She noted that today hardly anyone on the planet has not been exposed to EMF radiation, making the demonstration that EMF exposure is a carcinogen difficult. Focusing research on those most heavily exposed and exposed for long duration will be important. Most case-control studies of that nature have found increased risk. With the challenges to epidemiology in the area, experimental studies such as those undertaken by NTP will be important going forward. She suggested using a larger unexposed group to avoid the need to use historical controls. With the need to rely on experimental studies in the future, the research needs to accelerate to keep up with introduction of new technologies. She commended the NTP studies, which she described as large, well conducted, and methodologically sound, providing more evidence of RFR carcinogenicity. She said the situation has evolved from precaution to prevention, as evidence has accumulated.

Dr. Lin reiterated his assertion that there are no unexposed animals.

Kevin Mottus from the California Brain Tumor Association wished to highlight the comments of Dr. Lennart Hardell, an oncologist and leading authority on wireless radiation and cancer. His comments pertained to the NTP studies and others. He cited clear evidence of several cancers including glioma and some evidence of other cancers, and the International Agency for Research on Cancer recommendation that RFR be classified as a Group One carcinogen to humans. Mr. Mottus then added his own comments. He said the mechanism behind RFR and cancer is now known, and that evidence is mounting of brain cancers in the frontal lobe, the cerebellum, and the temporal lobe — the brain regions that receive the most cell phone radiation. He believes FDA should take quick action to rein in FCC, which is dominated by industry, especially with the rollout of 5G and its thousands of transmitters. He said everyone should be alarmed, because the situation is not a public health crisis in the making, but is going on currently, and could become horrific in the near future.

Dr. Young Hwan Ahn from the EMF Research Committee of the Korean Institute of Electromagnetic Engineering and Science spoke by phone from Korea. He briefly introduced himself and described his background as a neurosurgeon. He described classification of tumors of the nervous system, such as glioblastomas and schwannomas, which comprise about 8% of brain tumors. Cardiac schwannomas are extremely rare. Despite that fact, the NTP study reports

have drawn special attention to tumors of the nervous system. If life-span RF exposure can cause increased incidence of tumors of the nervous system, regardless of statistical significance, attention must be paid to the carcinogenic potential of RFR in humans. He stated the NTP study was well organized, with the survival of the sham-exposed group the most significant drawback.

Dr. Heroux from McGill University suggested that page 13 of the rat document be reworked to group the results according to tissue types, which would highlight that brain and nervous tissues showed carcinogenic action at various stages and in various locations in the body. He felt that health effects in rodents would emerge later in life, past the 2-year bioassay point. If bandwidth is increased, the chances of interferences and consequences to biological systems also increase. He concurred with Dr. Lin's point that there are no controls, in that the rats thought of as controls have in fact been exposed to extremely low frequency radiation. Genetic drift caused by exposure is also a problem, with potentially serious consequences that are not discussed in the literature.

Dr. Ronald Melnick, a retired NIEHS/NTP toxicologist and one of the original scientists associated with the NTP cell phone RFR studies, spoke on the utility of the NTP data on cell phone RFR for assessing human health risks. He provided background information about the history of the project, which began with the original nomination in 1999. The initial objectives were to test the null hypothesis — that cell phone RFR at non-thermal exposure intensities is incapable of inducing adverse health effects — and to provide dose-response data that could be used to assess potential human health risks for any detected adverse effects. The results described in the technical reports "show quite clearly" that the null hypothesis has been disproven, with many adverse effects identified. Dr. Melnick delineated the adverse effects observed and described their levels of evidence of carcinogenicity. He pointed out that even a small increase in cancer risk could have a serious public health impact due to the widespread use of cell phones.

Dr. Lin asked Dr. Melnick to discuss how the decision was made to use one common control in the studies. Dr. Melnick said that comparing exposed groups to sham controls was ideal for space constraints and feasibility. In hindsight, he said, including additional control groups might have been better. With the provision of 90 animals per group, NTP felt sufficient power was achieved with the common controls. He acknowledged the historical controls were difficult to work with due to differences in housing and the exposure system; however, they were used to demonstrate how rare a particular event was in the NTP database, not for direct comparisons.

Day 2 of the proceedings was adjourned at 4:31 p.m.

Day 3: March 28, 2018

Dr. Eaton welcomed everyone to Day 3 of the meeting and asked all attendees to introduce themselves. Designated Federal Official Dr. Mary Wolfe read the conflict of interest statement.

L.4.5. Peer Review of NTP Studies in Rats of Cell Phone RFR

L.4.6. Charge to the Panel

Dr. Blystone presented the charge to Panel 2, addressing the draft NTP Technical Report TR-595, Toxicology and Carcinogenicity Studies in Hsd:Sprague Dawley® SD® Rats Exposed to

Whole-Body Radiofrequency Radiation at a Frequency (900 MHz) and Modulations (GSM and CDMA) Used by Cell Phones. The panel was charged to:

- Review and evaluate the scientific and technical elements of the study and its presentation
- Determine whether the study's experimental design, conduct, and findings support the NTP's conclusions regarding the carcinogenic activity and toxicity of the test agent

L.4.7. Results of the NTP Studies of Cell Phone Radiofrequency Radiation in Hsd:Sprague Dawley® Rats

Dr. Wyde briefed the panel on the partial findings, the results of the 28-day prechronic toxicology studies, and the 2-year toxicology and carcinogenicity studies in rats, which included 14-week interim evaluations of histopathology, genetic toxicity, and hematology.

In December 2015, the final report was received from the study lab, in which concern was raised regarding the findings in the brain and the heart. That led to a complete review of the brain and heart lesions, and preparation of the partial findings report, which was released in May 2016, following external peer review.

The draft report's preliminary conclusions (subject to peer-review modification) were as follows:

In male rats exposed to GSM-modulated cell phone RFR at 900 MHz, there was *some evidence of carcinogenic activity*, based on incidences of malignant schwannoma in the heart. Lesions that *may have been related to cell phone RFR exposure (equivocal evidence)* included:

- Incidences of adenoma or carcinoma (combined) in the prostate gland
- Incidences of malignant glioma and benign or malignant granular cell tumors in the brain
- Incidences of adenoma of the pars distalis in the pituitary gland
- Incidences of pheochromocytoma (benign, malignant, or complex combined) in the
- adrenal medulla
- Incidences of pancreatic islet cell adenoma or carcinoma (combined)

Non-neoplastic lesions occurred in the heart, brain, and prostate gland.

In female rats exposed to GSM-modulated cell phone RFR at 900 MHz, there was *no evidence of carcinogenic activity.* Nonneoplastic lesions occurred in the heart, thyroid gland, and adrenal gland.

In male rats exposed to CDMA-modulated cell phone RFR at 900 MHz, there was *some evidence of carcinogenic activity*, based on incidences of malignant schwannoma in the heart. Lesions that *may have been related to cell phone RFR exposure (equivocal evidence)* included:

- Incidences of malignant glioma in the brain
- Incidences of adenoma of the pars distalis in the pituitary gland
- Incidences of adenoma or carcinoma (combined) in the liver

Non-neoplastic lesions occurred in the heart, brain, and prostate gland.

In female rats exposed to CDMA-modulated RFR at 900 MHz, there was *equivocal evidence of carcinogenic activity*, based on incidences of malignant glioma in the brain and incidences of pheochromocytoma (benign, malignant, or complex combined) in the adrenal medulla. Nonneoplastic lesions occurred in the brain.

L.4.7.1. Questions for Clarification

Dr. Andrews-Jones asked in which sub-anatomic area of the brain the lesions occurred. Dr. Wyde said he was not sure they were limited to one anatomic region, and Dr. Cesta confirmed they were scattered.

Dr. Felter asked about the impact of the CDMA modulation on the pups in terms of survival and decreased body weight, and whether that would meet the bar for exceeding a maximum tolerated dose in an NTP bioassay. Dr. Wyde said that, although the body weight decreases in the 28-day study in the 9 W/kg group were quite significant, they were not as severe in the 2-year study in the 6 W/kg group, and neither group exceeded a maximum tolerated dose. Dr. Blystone said the decreases were considered delays, and the pups caught up eventually, constituting a transitory effect. Dr. Felter asked about the decreased pup survival at postnatal day 4 in the high-dose group. Dr. Blystone said that NTP believes the decrease in survival was a real effect, but was not significant enough to exceed the maximum tolerated dose.

Dr. Felter asked about the GSM females, with the three different tumor types in the heart. If they occurred in separate animals, would they not be combined to give an incidence? Dr. Cesta said that for the analysis, the myocardial and endocardial lesions were combined, but nonneoplastic lesions were not combined with neoplastic lesions, so the hyperplasias were not combined with the schwannomas. Dr. Malarkey noted that the paragangliomas would not have been combined with the schwannomas due to the different cell origins. Dr. Felter asked for confirmation that in the GSM females, the incidence in the mid-dose group for related heart tumors was 3 in 90. Dr. Cesta confirmed that.

Dr. Kaufmann asked about litter size and its effect on growth rate, and how the pups were distributed to the dose groups. Dr. Wyde said that three pups from each litter were included in the 2-year study, but as all but the control pups came from exposure chambers, they were already exposed. Dr. Blystone noted pup distribution was randomized.

Dr. Andrews-Jones asked whether dam lactation had been considered as a possible source of the decreased pup weights. Dr. Blystone said that had not been evaluated.

Dr. Cline asked for confirmation that the ears were not trimmed in this study. Dr. Wyde said they were not. Dr. Cline asked if gynecomastia was present in any male animals, to which Dr. Wyde replied, no. Dr. Cline asked about adrenal weights in the animals with pituitary gland tumors, and whether the tumors were derived from lactotroph or corticotroph tumors. Dr. Wyde said that had not been explored. Dr. Cline asked about the association between lymphoid organ atrophy and chronic progressive nephropathy, and whether there was a rationale for the association. Dr. Cesta explained the atrophy was due to decreased blood flow because of exacerbated polyarteritis nodosa.

Regarding the body weight issue, Dr. Lin asked for confirmation that food and water consumption were not monitored, which Dr. Wyde said was true.

Dr. Felter asked about the mammary gland tumors observed in the 14-week interim evaluation—whether that finding was unusual, or such tumors are sometimes seen that early. Dr. Cesta said that some mammary gland tumors can occur earlier, but the adenocarcinoma was surprising.

Dr. Whiteley asked Dr. Cesta about assessment of reproductive cyclicity with vaginal swabs and whether, during the histopathology evaluation, ovarian and vaginal morphologies were evaluated to assess cyclicity. Dr. Cesta said that cyclicity data cannot be determined from the histopathology evaluation, but the ovaries and vagina can be evaluated to determine if there is discordance or evidence that they are not cycling.

Dr. Whiteley asked why no functional observational battery assessments were done, particularly because concerns for central nervous system toxicity were present. Dr. Wyde said they were not part of the study design, and animals could not be observed during the exposures.

Dr. Andrews-Jones asked Dr. Cesta what the typical severity expectation would be for chronic progressive nephropathy in this age of rats at 2 years, noting the 4-point severity scale. Dr. Cesta said many grade 4s had been observed, especially in the controls, which was unusual.

L.4.8. Pathology Peer-Review Process and Selected Lesions for the Two-year Study of Cell Phone Radiofrequency Radiation in Rats

Dr. Cesta briefed the panel on the pathology peer-review process for the rat study, and described and depicted heart, brain, and kidney lesions observed in the 2-year study. He delineated the standard NTP pathology peer-review process. With the release of the partial report findings, some adjustments were required. Potential treatment-related proliferative heart and brain lesions were initially selected for early reporting in the partial report. Four Pathology Working Groups (PWGs) were convened — the two initial PWGs and two additional PWGs composed of specialists in neuro- and cardiovascular pathology.

Dr. Cesta provided more details about the heart, brain, and kidney (chronic progressive nephropathy) lesions observed, including diagnostic criteria and visual depictions.

L.4.8.1. Questions for Clarification

Dr. Kaufmann questioned the diagnostic criteria for the brain lesions. He made a distinction between a tumor-like hyperplasia and a true hyperplasia. The similarity to a glioma is an important finding and should be in the report, he noted. He asked about the basis of the diagnostic term, glial cell hyperplasia. Dr. Cesta said he was unsure of the term’s origin, but that the PWG agreed with the study pathologist’s diagnosis. Dr. Kaufmann asked if any special staining had been considered to differentiate between neoplastic and nonneoplastic changes. Dr. Cesta said staining had been discussed, with plans to investigate the issue further. Dr. Malarkey said that NTP agrees with Dr. Kaufmann’s concerns about differentiation of the tumors, and studies are underway to look at immunohistochemical staining of the lesions.

Dr. Lin asked if one or more of the sections taken from the brain were through the auditory nerve or the cranial nerve, and whether any special attention had been paid to any sections where the auditory nerve was visible. Dr. Cesta replied that none of the sections had gone through the

auditory nerve. He said no lesions in the brain having morphology of a schwannoma had been observed. Dr. Lin said, without a section through the auditory nerve, observing any pathologies in that region would have been difficult. He added that the team, having a priori knowledge that acoustic neuroma or vestibular schwannoma were part of the findings in humans, should have investigated the issue. Dr. Sills, NTP Chief of Pathology, provided further clarification and explained that a comprehensive evaluation of the nervous system (i.e., central nervous system, peripheral nervous system, all the nerves within the animal) is performed with all studies. He said that as such, if involvement of the nerves of the brain, including cranial nerves or nerves that deal with the auditory system, were indicated, it would have been detected at that point. Dr. Sills stated that Dr. Cesta did perform a comprehensive reevaluation of the whole neck area because of the issue of acoustic neuromas.

Dr. Adler was concerned about the incidence of right ventricular cardiomyopathy. He asked, in NTP's experience, if the left ventricle is more often the site of spontaneous cardiomyopathy. Dr. Cesta said spontaneous cardiomyopathy was certainly more prevalent and more obvious in the left ventricle. Dr. Adler asked if concluding that RFR was related to the lesions on the right side would be fair. Dr. Cesta replied that if the criteria were applied to nonneoplastic lesions, it would be equivocal; it was a minor component of the overall cardiomyopathy. Dr. Adler noted that he would like additional references to examples in which spatial associations were not found. Dr. Adler asked whether there was an effect compared to the historical data. Dr. Cesta replied that NTP does not have historical control data on this issue. Dr. Malarkey added that more data should be available for Hsd:Sprague Dawley® SD® rats soon. Dr. Adler asked if any correlation was observed between the malignant schwannomas and the ventricular cardiomyopathy. Dr. Cesta said not that NTP could discern.

Dr. Rinke supported Dr. Adler's point about the right ventricular cardiomyopathies and asked about their distribution, along with the distribution of the endocardial schwannomas or hyperplasias, which are not frequently observed in the right ventricle. He asked Dr. Cesta if immunohistochemistry was performed on the schwannomas. Dr. Cesta said plans are to explore that further, but NTP has done nothing to this point.

Dr. Whiteley asked if anything was unique about the schwannomas—any features more prominent in those observed in the animals relative to textbook descriptions. Dr. Cesta said that he noticed nothing unusual about the schwannomas; they were relatively standard.

Dr. Andrews-Jones asked if a spatial irregularity in the right ventricular schwannoma had been shown. Dr. Cesta said some, but not all, lesions tended to occur more toward the apex. Dr. Andrews-Jones asked Dr. Sills to confirm that glioblastoma multiforme is a term used only in humans. He replied that in this study, the lesions were called malignant gliomas based on peer-review agreement. He noted that more information is emerging about the molecular nature of the tumors in humans and rodents.

Dr. Harkema asked for more information about the variability of the right ventricular lesions, and whether they were more epicardial. Dr. Cesta said they tended to be focused in the subepicardial region, while more severe cases extended somewhat into the myocardium. Such lesions are quite rare, Dr. Cesta confirmed.

Dr. Cline asked whether a hyperthermia effect, independent of the radiation effect, had been considered relative to the CPN. Dr. Cesta replied that had not been examined. He noted that no

thermal effect was observed on the testes, which are sensitive to heat and were examined for that reason. Dr. Cline asked if heat shock proteins were evaluated. Dr. Cesta said no and agreed that that would be an interesting area to investigate. Dr. Lin noted that a 1°C variation in body temperature is within a normal physiological range, should not per se be a matter of concern, and would not trigger heat shock proteins.

Dr. Barnes noted some data in the literature indicate that a very small increase in temperature could trigger heat shock proteins. Dr. Harkema said that different organs respond differently to heat increases, such as the skin and testes. Dr. Lin agreed, but clarified that temperature variations are not always deleterious. Dr. Kuster added that only the whole-body temperature change was determined, not tissue-specific temperatures, and cautioned against putting too much weight on the temperature data. Dr. Gamboa da Costa from FDA clarified that only subcutaneous temperatures were measured, not internal temperatures, and also cautioned against making claims about tissue-specific temperature effects.

The chair permitted ad hoc comments from some public attendees. Mr. Mottus from the California Brain Tumor Association questioned why no EMF experts were included on the peer-review panel. As a result, he asked that the panel recuse itself and reconvene with EMF scientists. Dr. Eaton responded that the studies are pathology studies and the panel has some of the best animal pathologists in the country.

Dr. Melnick, retired NIEHS/NTP scientist and public attendee, noted that the hyperplasia severity varied among the rats, and asked Dr. Cesta to show a severity grade 4 instead of a 2, as he had, so that the lesion would be evident. He also observed that, although the right ventricular cardiomyopathy had been characterized as not being very severe, it had been listed as the cause of death on some of the individual animal pathology reports. Having detected a schwannoma at necropsy, he asked why additional sections were not cut to see if any schwannomas in the heart had been missed. Dr. Cesta replied that he could not show a grade 4 hyperplasia because he did not have one available. He said some debate had occurred among the pathologists about the right ventricular cardiomyopathy as the cause of death, so confidence in that very subjective assessment was not high. He noted that additional sections of the heart are not typically taken to avoid bias and maintain consistency.

Regarding the 28-day studies, Dr. Davis from Environmental Health Trust asked Dr. Cesta if perinatal effects might have been consistent. She pointed specifically to Table H-2 in the report, which summarizes the epididymal spermatozoa measurements. She said there appeared to have been a significant pathological impact and statistically significant declines and wondered why a Williams' trend test had not been conducted. She noted the lack of linearity in the response. Dr. Shockley said the tests had been conducted, and the trend was not significant. The 6 W/kg group showed a large uncertainty and did not pass the statistical filter. Dr. Davis said that, in many cases, statistical significance might not be the same as public health importance.

L.4.9. Presentation of Peer Review Comments

Dr. Felter, the first peer reviewer, felt that it would be important for the report to contain an in-depth discussion of dose metrics that a layperson could understand, particularly SAR sensitivity. She was concerned that discussion of SAR sensitivity in the report as it relates to humans could be misinterpreted, especially the comparison of the low dose in rats, 1.5 W/kg, to the FCC limit of 1.6 W/kg. She suggested more discussion of the increased pup mortality and survival at high

doses. Regarding interpretation of the tumor findings, the evidence should be considered in totality. Regardless of the lack of statistical significance in the females, the findings in the heart and brain of that group should be discussed due to biological evidence of an effect. She commended NTP for taking on such a challenging project, and suggested including all information that might be illustrative, or if not considered helpful, expressing why. One example is time to first tumor information, especially for the key target organs, such as the malignant schwannomas in the heart. She suggested adding more context about the inherent qualities of cell phone RFR and the conditions under which they occur, as they should not simply be assumed.

Dr. Lin commented on the issue of the term “whole body SAR” and pointed out that the exposure would be the same regardless of the organ involved. Dr. Felter responded the presentation was that the exposure of all target organs would not be the same, with several elements such as tissue absorption affecting SAR sensitivity. Dr. Lin said that Dr. Felter’s point about time to tumor is relevant. Dr. Kuster noted that the dosimetry was based on numerical animal models, and that all tissues were exposed to approximately the same levels for the same amount of time. He said the translation to thermal dose had not been fully addressed in these studies, so it could not be excluded that some tissues might have experienced mild hyperthermia at the highest dose. Dr. Adler noted that the study did not evaluate tissue-specific SAR.

Dr. Whiteley asked about the physics of how surrounding organs, such as the lungs or ribcage (in the case of the heart) affect radiation exposure. Dr. Barnes replied that the surrounding organs do affect absorption and distribution. Dr. Eaton agreed with Dr. Lin’s assertion that the situation is similar to pharmacokinetic or pharmacodynamic modeling. Dr. Adler noted that the heart is surrounded by an electrolyte fluid-filled pericardial sac and uniquely sits in an air cavity, essentially its own reverberation chamber.

Dr. Bucher noted that SAR distribution is an important topic and including the information in the report as accurately as possible is important. He noted that the goal of the report is to describe what was done in the studies as clearly as possible and asked that the focus return to evaluating this study in and of itself with regard to the exposures and what they mean. He said that adding hyperlinks in the report to the video recordings of the Day 1 presentations was a consideration.

Dr. Kuster said that the view of the heart as sitting in its own reverberation chamber was not accurate. He said a dosimetric evaluation of the heart had been done, and although it did not provide extensive detail, it is a very good proxy of the heart exposure.

Responding to Dr. Felter’s review comments, Dr. Wyde acknowledged the difficulty faced by the risk assessment community when addressing localized exposures. The goal of NTP’s studies is hazard identification, so the goal in this study was to expose each organ to as much RFR as possible. Efforts were made to expose all organs to the same level of RFR, but how much that can be controlled is limited. It fell within a two-fold range. Dr. Wyde reiterated a lay summary would be prepared, and he endorsed the ideas of a glossary to help the public understand some of the EMF terminology and access to the Day 1 presentations. He acknowledged the presentations had aided understanding and pledged good effort to include much of the added information into the revised reports.

Regarding Dr. Felter’s comments on pup mortality and body weight, he said the body weight changes were transient and NTP was unsure whether this was a dam effect or a pup effect. Dr. Felter asked that the report be more explicit in its discussion of this point, and Dr. Wyde agreed.

Regarding the decreased survival in the highest CDMA dose group, Dr. Wyde said additional description would be added to the report. He addressed Dr. Felter's comment regarding similarities between the modulations. All information was taken into account, for both the rats and mice, in looking for similarities. Combining the GSM and CDMA modulations was the topic of much discussion. Doing so is challenging from a statistical standpoint, and NTP is looking at ways to do so, as it addresses the issue of whether modulation or frequency was important. He said further evaluation of the differences or similarities would likely be addressed in follow-up manuscripts. Regarding the time-to-tumor issue, he urged caution, but noted that more information would be added to the report, where appropriate. He committed to adding some of the more recent references and, in the introduction, clarifying how this study fits in with others that have been conducted.

Dr. Cline, the second peer reviewer, called for greater clarity on the reverberation chambers, particularly regarding the control chambers and how they were confirmed to be zero or near-zero exposure, to be explicit that they were shielded, and that no antenna was present. He suggested some assessment of the sympathetic and hypothalamic-pituitary-adrenal axis with respect to stress assessments in the animals. He noted that the adrenal gland weights were not presented in the body of the report and suggested they be moved, with discussion that the pheochromocytomas could have resulted from a stress response. Multiple indicators of stress are included in the report and seeing if they occurred within the same animals would be interesting. He called for more discussion of the mammary gland assessments and differentiation of pituitary gland tumors. He requested clarification on the nature of the noise within the testing apparatus and how audible it was to the rats, including whether it was in the rat vocalization range, where it could be a source of stress. He agreed that the female reproductive system should be histologically examined; specifically, the 14-week assessment should be reevaluated. He asked for more mechanistic discussion of why the chronic progressive nephropathy was lower in the RFR-treated groups and asked why NTP attributed chronic progressive nephropathy in the control animals to decreased feed consumption when it was not measured. He recommended that the auditory vestibular tissues be examined histologically. He disagreed with using historical and concurrent controls at the same time.

Responding to Dr. Cline's comments, Dr. Wyde said more detail would be added to the description of the control chambers and exposures in the report. He said that stress lesions were not examined in relation to specific animals; however, Dr. Cline's remarks about stress assessment would be kept in mind in the design of future studies. Moving the discussion of adrenal gland weights forward would be considered. Regarding the mammary gland and pituitary gland tumor assessments, Dr. Wyde said that histopathology suggestions would be taken into consideration for future studies. Dr. Wyde acknowledged that the noise was in the range of rat vocalization, but the correlation between noise and stress is only speculation at this point. He said that the noise was accounted for by piping the same sound into the low-dose chambers so that everything was normalized across the study. He noted that feed consumption had not been monitored, so it should not have been related to the chronic progressive nephropathy rate. He said that assessment of the auditory vestibular tissues could still be done. He noted that the treatment of the controls would be discussed in more detail to help clarify the approach taken.

Dr. Eaton asked Dr. Wyde to clarify his statement that the high-dose noise had been piped into the other chambers, and whether that included the controls, which Dr. Wyde confirmed. Dr. Lin

asked how the piping had been done. Dr. Capstick explained the process, which involved placing speakers in the air vents.

Regarding the incidence of chronic progressive nephropathy, Dr. Adler asked about evaluation of the feed exposed to RFR, and whether thermal degradation or micronutrient effects were observed. Dr. Capstick said they measured the power absorbed by feed when the bowls were full, and the level was quite small, so no increase in feed temperature was expected. Dr. Adler asked if any follow-up micronutrient evaluation of the feed had been done, to which Dr. Wyde replied no. Dr. Adler said that it might have a bearing, and that NTP should consider following up on that issue. Dr. Bucher said the diets are autoclaved and so are nutrient-enriched to take this into consideration. He felt it was unlikely that any heating from RFR would be anywhere near the autoclave temperature. Dr. Wyde added that because the radiation was nonionizing, energy to strip any of the electrons was insufficient. Dr. Adler recommended transparency in the report on the issue. NTP laboratory animal veterinarian Dr. Angela King-Herbert corrected Dr. Bucher and stated that the diets are actually gamma-irradiated and not autoclaved, and nutrients are checked before and after irradiation.

Dr. Cesta commented further on the vestibular system and the intracranial nerve. He said a thorough gross review of those tissues had been completed, with no lesions identified. As to the pituitary gland tumors, individual animals were not examined to determine if they also had mammary gland tumors, although that could still be done, along with immunohistochemistry of the pituitary gland tumors to determine their types. Dr. Eaton asked if hormone analysis could be done on serum from the 28-day studies, perhaps addressing Dr. Cline’s concerns about stress-induced changes between controls and exposed animals. Dr. Cline said he assumed that the serum was no longer available, which Dr. Wyde confirmed.

Dr. Rinke noted that the Zymbal’s glands, which are close to the auditory cavity, were examined so the absence of lesions would support that none occurred in that region. Dr. Cline noted that the section of interest might have been examined, and if that were the case, it could be stated in the report. Dr. Malarkey described how the vestibular system had been sectioned, and noted that if there were any gross masses, they would be detected at that point. Dr. Whiteley asked how frequently the vestibular system and inner ear were hit, to be able to evaluate those structures. Dr. Malarkey said this is not seen very often. Dr. Sills said NTP would go back, look carefully at the issue, and add to the report or do additional studies as necessary. Dr. Harkema stated that the third head section having gone through the vestibular region was doubtful. Dr. Cesta noted that during the Audit of Pathology Specimens, the brains would be re-reviewed grossly.

Dr. Malys, the third peer reviewer, noted the elevated tumor occurrences in the mid-dose range and the apparent nonlinear dose-response trend. He recommended aggregating tumor occurrence information across dose range. He said he would be interested in a table for the rats that addresses the question of whether evidence supports a nonlinear, dose-dependent response, simply by exploring the existing data. He said he was both impressed and shocked to see how different the grades were for different types of tumors. He felt additional discussion of severity grading should be included in the report. He reiterated his point about the importance of a well fleshed-out system of controls, along with an emphasis on reproducibility of results. He asked NTP to better describe the relationship of the 2G and 3G technology used in the studies to the current 5G technology to make the data and exposures more relevant to casual cell phone users.

Dr. Andrews-Jones felt looking at the data more comprehensively was an excellent idea, particularly whole biological systems. For example, many of the equivocal tumors were in the endocrine or neuroendocrine systems.

Dr. Harkema asked Dr. Malys about the implications of having six times more exposed animals than controls. Dr. Adler noted that that would increase the opportunities for random events, unrelated to treatment, to occur in the treated groups. Dr. Malys agreed. He emphasized that future studies should have more robust control systems to support reproducibility. He said the positive results were difficult to interpret in this study due to decreased survival in controls. Dr. Barnes stated that the effects noted in this study should be considered a real outcome.

Dr. Shockley agreed that summarizing results that suggest nonlinear responses would be useful. Although a linear-based trend test is used, with lower power to detect nonlinear trends, the pairwise test is also used, which can help detect nonlinear responses. He noted that NTP uses a weight-of-evidence approach and not just statistics, and that the current NTP statistics methods are well supported. Dr. Malys clarified that, when he spoke of aggregating tumor information from different tumor types, he was not talking about combining to perform a statistical test, but solely about the concept of exploring the data to examine the possibility that a trend might exist. This analytical distinction is subtle, but important. Although combining results can be quite challenging statistically, simply exploring the data to gain wider insights can be useful.

Dr. Lin noted that because all tissues and organs were exposed to similar doses, he asked what would be the difficulty in comparing the occurrence of total tumors (e.g., adenoma, schwannoma, carcinoma, glioma) in all tissues and organs between the RF-exposed group and the concurrent control group? Dr. Shockley restated Dr. Lin's question as proposing that all tumors across all groups be combined as a total analysis of tumor burden. He noted that that is actually done in the report, although that information is not used to make the final decisions. Dr. Bucher noted the vast majority of total tumor burden comprises spontaneous tumors, which can lead to missing more subtle effects. Consideration of total tumor burden has been used historically in cancer studies but set aside as less useful.

Dr. Whiteley, the fourth peer reviewer, commended NTP for conducting the study, including its heroic efforts in engineering. He felt a written statement regarding tumor latency and whether it adds any value to the assessment of carcinogenicity for a given tumor should be included. He said distinguishing the different glial cell types would be important. He agreed adding a table of identified target effects, listing male and female and modulation, would be helpful. He said visualizing commonalities in the report is difficult, and that such a table would help. He addressed the report's discussion, noting several recently published and existing studies with a concordance of effects point in the same direction. He felt that that should be brought out in the report, because it adds significance to the findings in the NTP study. He recommended a discussion about the possibility of nonlinearity and biphasic effects and how they might impact the interpretations of significance. He also recommended adding a clear statement in the report's abstract about the rationale for dose selection.

Dr. Wyde said that a discussion of time to tumor would be added. The issue of glial cell types would be evaluated further. He agreed that adding summary tables from the modalities to identify similarities and differences would be good. He said that more discussion and comparison of information from other published studies and reported tumor types, which had been present in

the preliminary report, would be added to the 2-year study report. He said nonlinearity would be taken into consideration on a case-by-case basis, as many different factors are considered when making level-of-evidence calls. He agreed to add the dose selection rationale to the report's abstract.

Dr. Adler, the fifth peer reviewer, commended NTP for its very high degree of integrity and transparency. He felt that the studies were well designed, well conducted, and robustly analyzed and reviewed. He felt that the report should make clear that the study is solely a hazard identification study, not a risk assessment study, and that FDA is the agency responsible for risk assessment decisions. He agreed that the dose selection rationale and justification should be added, including an explanation of the dose spacing and why the doses changed between the 28-day and 2-year studies. He also recommended further discussion on the use of historical controls, including on the validity of control animal survival and chronic progressive nephropathy. A discussion of fluorescent versus incandescent lighting also should be included. He mentioned that in his company, the term "uncertain relationship to treatment" is used instead of "equivocal," and hoped that regulatory agencies would interpret that term in similar fashion, erring on the side of public health. He agreed with Dr. Corcoran's recommendation of a weighted decision rubric for making level-of-evidence calls, as there is a fine line between *equivocal* and *some* in many of these cases.

Dr. Adler requested better clarification between the non-SAR-dependent responses and nonlinearity of the dose response. He noted the maximum tolerated dose is based on thermal evaluations and clearly was not measured at night. He noted that thermal effects were measured during the day, although rodents are much more active at night, and asked that this be reconsidered in future studies. He said that information on specific biomarkers would be useful, particularly regarding time-to-tumor development. He agreed with Dr. Melnick that the studies should clearly name the target organs, such as the heart in rats. Regarding the right ventricular cardiomyopathy, he felt the occurrences in the studies were unusual and should be brought out. He asked that the report discuss the perceived lack of associations between the right ventricular cardiomyopathy and the occurrences of schwannomas and Schwann cell hyperplasias. He also asked for more discussion on the rat hippocampus being positive in the comet assay in the CDMA-exposed group without an association with brain tumors, spatially. He noted that in the rat study, the heart is clearly sending a signal, and represents a ripe area for future exploration of the adverse outcome pathways to neoplasia. It was also noted that 2-year rodent bioassay and lifetime epidemiology studies for RFR exposure represented an undue time period to further identify a hazard to public health.

Dr. Lin noted that, for a true control, all aspects of the environment should be the same except for the agent involved. He questioned whether that was the case in the rat studies, particularly with respect to the noise exposure for the controls. The lighting also differed from those in the historical controls. Thus, the environmental conditions were not the same.

Dr. Wyde responded to Dr. Adler's comments. He appreciated the distinction Dr. Adler made that these were hazard identification studies and confirmed that FDA would be responsible for any risk assessment. He committed to describing the dose spacing more accurately in the report and would consider expanding the discussion of the historical controls. He felt that a decision-making rubric would not be fully appropriate in this case, as the studies are conducted on a case-by-case basis without a one-size-fits-all approach. Further discussion of some of the decisions

made, however, would be appropriate. He acknowledged Dr. Adler's point about temperature taken during the day and not at night, and noted that in future studies, a different technology would be used for data collection of temperature, allowing for nighttime measurements. He agreed about the need for biomarkers. In terms of target organs, in the rat, the only one was the heart. He acknowledged that reorganizing and presenting the equivocal data in a more digestible, user-friendly form would help clarify several issues, including target organs. Dr. Wyde expressed confidence that NTP and other researchers would pursue the mechanism behind the effects observed in the heart. Dr. Cesta said that more information would be added to the report about the spatial associations with the right ventricular cardiomyopathy and the proliferative Schwann cell lesions, along with information about other spatial associations or lack thereof that the panel had mentioned.

Dr. Felter asked that the report make clear that the temperatures were taken subcutaneously.

Dr. Adler added that a discussion of the change of doses from the 28-day to the 2-year studies should be included in the report.

Dr. Andrews-Jones felt that Dr. Adler's point about hazard identification was important and should be included in the report's abstract.

Dr. Gamboa da Costa from FDA recommended caution when using the word "dose" for these studies, as W/kg is not dose — it is intensity of radiation. A better way to express the dose would be to integrate time of exposure and express it as Joules/kg.

Dr. Kaufmann, the sixth peer reviewer, agreed with the other reviewers' statements concerning the design and conduct of the studies. He asked that the rationale for the use of different frequencies for the rats and mice be added to the report. He said he would like to see more immunohistochemistry on the brain tumors and hyperplasias to aid in making decisions about the relevance of the brain effects. He agreed with the proposal to add a tumor totality table to the reports. He noted that distinguishing between a conclusion of *some* or *equivocal evidence* is very difficult, especially given the low survival in the controls.

Dr. Bucher noted that NTP does perform survival adjustments during the statistical evaluations, so survival differences are taken into account. Dr. Wyde said that the schwannomas occurred early, so the low survival in the controls is not believed to have influenced statistical interpretation. The gliomas occurred later. Dr. Eaton added that that is where it would be useful to discuss time to tumor.

Ms. Pant, the seventh peer reviewer, addressed the genetic toxicological aspects of the rat study. She noted the hippocampus was positive, whereas the frontal cortex showed a trend, which was not positive due to variability. She asked if the data could have been transformed for analysis or analyzed using nonparametric statistical methods. Because of so much variability in the percentage of damaged cells in the liver of control animals, and the percent tail DNA was very high, she asked if historical data were available. She asked if the slides were coded during the blinding process and speculated that shipping of the samples might have been a factor in the variability among the controls.

Ms. Witt agreed with Ms. Pant's comment on the frontal cortex in the CDMA-exposed male rats, which was close to statistical significance. She noted the variation among animals made

capturing the response statistically more difficult. She said that both parametric and nonparametric approaches were used. With regard to the liver, she agreed with Ms. Pant on the high values of percent tail DNA. The scorers were very conservative in using the scoring software, and visually eliminated cells that appeared to be hedgehogs. She described more details of the scoring process; the scoring was redone with the same slides and the software was confirmed as accurately determining tail DNA. She noted Ms. Pant's point about the potential impact of shipping, with a high level of DNA damage related to freezing of the cells, and added that in the past, the liver was not determined to be a cell type significantly affected by freezing.

Regarding the comet assay, Dr. Cline said there were different OECD methods of disaggregating the cells and having a description in the report of which was used in these studies would be helpful. He said that, of 10 cell types in the brain, there is a range of survival during the disaggregation process and variability in cell degradation rates. He asked Ms. Witt if she had an opinion on which brain cell types would be most likely to survive and make it to the slide. She replied that an explanation of the method of disaggregation is provided in Appendix E of the report. She said that due to the method of preparation, she could not answer Dr. Cline's inquiry about the surviving brain cell types. She was not aware of a method to distinguish between degradation based on cell type.

Ms. Pant said that once single-cell suspensions are made, the suspension is a mixture of cells, and there is no way to determine cell types. Ms. Pant said that problems in evaluation develop with too many cells on a slide.

L.4.10. Panel Discussion and Recommendations

Dr. Eaton introduced the session, noting that at the end of the discussion, Panel 2 members would vote on the conclusions for the draft rat NTP Technical Report TR 595. Each preliminary conclusion was considered individually, with commentary from NTP staff.

L.4.10.1. GSM-Exposed Males

The deliberations began with male Hsd:Sprague Dawley® SD® rats exposed to GSM-modulated cell phone RFR at 900 MHz. The first finding was "*some evidence of carcinogenic activity*" based on incidences of malignant schwannoma in the heart." Dr. Wyde explained the rationale behind the conclusion: increased incidences and a positive trend across the exposure groups with an incidence of zero in the controls. Dr. Harkema asked why the conclusion was not raised to *clear evidence*. Dr. Wyde said, although the level of the response exceeded the historical control range, it was not statistically significant and, therefore, was not raised to a higher level.

Dr. Felter noted a commonality of response across the other three test groups (females, both modulations), with malignant schwannomas in some of the treated animals and none in the controls. Dr. Adler asked about the line between *some evidence* and *clear evidence* and whether NTP would have called it clear evidence if the high-dose incidence had been statistically significant. Dr. Wyde said no, the level of response was not at the *clear evidence* level. Dr. Blystone clarified that NTP does try to look across reports to be consistent in level-of-evidence calls. NTP staff thought that the malignant schwannomas looked treatment-related but believed survival of the controls played a role in the observed effect.

Dr. Eaton called for a motion on the conclusion. Dr. Felter moved to upgrade the conclusion from *some evidence* to *clear evidence*. Dr. Adler seconded the motion. The panel voted 8 yes, 3

no, so the motion carried. The “no” votes were Drs. Harkema, Malys, and Cline. Dr. Malys said the numbers presented were not striking enough to justify the upgrade, along with the issues with the control group. Dr. Harkema and Dr. Cline said their reasons for voting no were similar to Dr. Malys’.

The panel next considered incidences that were deemed “may have been related to cell phone RFR exposure (*equivocal evidence of carcinogenic activity*).” The first conclusion in the category was “Incidences of adenoma or carcinoma (combined) in the prostate gland.” Dr. Wyde explained the rationale for the conclusion: although the incidences did exceed the historical control range, it was not by much. Also, the only increased incidence was at the mid-dose, 3 W/kg, and that increase was not statistically significant. Dr. Lin questioned the relevance of including the current controls in the historical controls. Dr. Bucher said a 5-year window is associated with historical controls. In this case, a small number of historical control studies fell in that window, so the current study heavily weighted the historical controls, but they still added validity to the report. The control animals from the current study were the most appropriate to consider. Dr. Lin felt that the issue deserved further consideration. Dr. Harkema moved to accept the conclusion as written; Dr. Andrews-Jones seconded. The panel voted 11 yes, 0 no, so the motion carried.

Next in the equivocal evidence category was “Incidences of malignant glioma and benign or malignant granular cell tumors in the brain.” Dr. Wyde explained the rationale behind the conclusion: Incidences occurred in all exposure groups, were flat across SARs, and fell within the historical control range. Replying to a query from Dr. Harkema, Dr. Wyde noted no statistical significance either by trend or pairwise comparison in this case. Dr. Eaton clarified instances can occur where a linear response is saturated at high doses, and, therefore, responses at lower doses cannot be ignored.

Dr. Felter moved to upgrade the conclusion from *equivocal evidence* to *some evidence*. Dr. Andrews-Jones seconded, but said she thought the discussion was solely about the gliomas. Dr. Felter noted that if the panel opted to change the conclusion to refer only to the gliomas, she would still move to upgrade based on the commonality of response observed across the different modalities and sexes, the lack of tumors in the controls, and considering the incidence of hyperplasia. The panel did elect to separate the conclusions. Voting on the upgrade moved by Dr. Felter just on the malignant gliomas, the panel voted 7 yes, 4 no, with Drs. Malys, Corcoran, Harkema, and Adler voting no. Dr. Malys explained his no vote stemmed from lack of statistical significance, lack of exceeding historical controls, and the appropriateness of how to put the information in the context of other findings. Explaining his no vote, Dr. Corcoran added the significant degradation of the control group due to premature death limited the strength of the controls as a comparison. Dr. Harkema agreed with Dr. Malys and Dr. Corcoran. Dr. Adler also agreed and felt that it was changing the rubric involved. He felt that the discussion of the report should look at what occurred across the two modalities to see patterns, as well as look for patterns across other studies. He also thought that patterns across studies should be considered for risk assessment, not hazard identification.

After splitting the brain conclusions, the panel next considered “Incidences of benign or malignant granular cell tumors in the brain” as a separate conclusion. Dr. Andrews-Jones moved to accept the conclusion as written (equivocal); Dr. Adler seconded. The panel voted 11 yes, 0 no, so the motion carried.

The next conclusion under the equivocal evidence category was “Incidences of adenoma of the pars distalis in the pituitary gland.” Dr. Wyde explained the rationale for the conclusion: although the incidences in exposed groups exceeded the historical control range, the incidences were similar across the exposed groups and not statistically significant by trend test or pairwise comparison. Dr. Felter moved to accept the conclusion as written; Dr. Rinke seconded. The vote was 10 yes, 1 no. Dr. Andrews-Jones said her “no” vote stemmed from her feeling that the issue needed to be examined more carefully, including effects in different cell types. She felt that there were many effects in the endocrine system in general, and that the call should be upgraded to *some evidence*.

Next, the panel considered “Incidences of pheochromocytoma (benign, malignant, or complex combined) in the adrenal medulla.” Dr. Wyde explained the rationale for the equivocal call: a statistically significant increase in the low- and mid-dose groups, no statistical significance in the high-dose group, and the incidences in the low- and mid-dose groups were at or only slightly above the historical control range. Dr. Adler asked what it would take to upgrade the call to *some evidence*. Dr. Bucher explained that this particular tumor is quite variable in the NTP experience, so more evidence of a dose response with higher incidences would be needed to upgrade. Dr. Corcoran asked about the propensity for a benign lesion to progress. Dr. Malarkey said little progression to malignancy was seen in the study. Dr. Corcoran asked about progression in broader experience with this type of tumor. Dr. Malarkey said the tumor would be low to medium potential to progress. Dr. Rinke asked about the nature of the malignancy. Dr. Cesta said it was based on invasion into the surrounding tissues. Dr. Harkema asked whether much weight had been given to the fact that there was no dose response. Dr. Bucher said the decision was based mainly on the fact that they are variable tumors with predominance toward the benign forms. Dr. Cline moved to upgrade the call to *some evidence*. Dr. Adler seconded. Dr. Cline explained his motion in that he viewed all pheochromocytomas as potentially malignant, whether they progressed or not, and that he did not think dose response was a big factor here. The panel voted 6 yes, 4 no, 1 abstain, so the motion carried. Drs. Malys, Harkema, Rinke, and Kaufmann were the “no” votes and Dr. Corcoran abstained. Dr. Malys explained that he had voted no due to the lack of establishment of a reason behind the nonlinear dose-response curve, the previously cited control issues, and being very close to the upper end of the historical control range. Dr. Corcoran said he had abstained because he felt it was too close to call. Dr. Rinke explained his no vote because the tumor was quite common, no metastasizing malignancies were seen, and decreased survival occurred in the control group, so he did not believe the effect was a true effect. Dr. Harkema agreed with Dr. Rinke. Dr. Kaufmann agreed with Drs. Rinke and Harkema in that the tumor was a common tumor, the survival rate in controls was low, and that he did not believe this was a true effect.

The next conclusion was “Incidences of pancreatic islet cell adenoma or carcinoma (combined).” Dr. Wyde explained the conclusion, which was that the incidences were close to the historical control range and there was limited statistical significance. Dr. Andrews-Jones moved to accept the conclusion as written; Dr. Kaufmann seconded. The vote was 11 yes, 0 no, so the motion carried.

Dr. Cline moved to include adrenal cortical adenomas as *equivocal evidence*. He explained that there was a theme of potentially stress-related mechanisms and that the incidences of adenoma were striking compared to concurrent controls. Dr. Eaton clarified that the normal procedure

would be to combine adenomas and carcinomas when in the same tissue. Following discussion by the panel, there was no second, so the motion did not carry.

L.4.10.2. GSM-Exposed Females

The panel moved on to female Sprague Dawley rats exposed to GSM modulation. The conclusion was *no evidence of carcinogenic activity*. Looking at the data related to the heart, Dr. Felter felt the data should be considered in relation to all heart findings across the studies. Dr. Eaton asked if she cared to make a motion. Dr. Felter moved to upgrade the incidences of malignant schwannoma in the heart to *equivocal evidence*; Dr. Andrews-Jones seconded. Dr. Harkema asked why this was not considered a significant effect. Dr. Wyde said that there was no statistical significance and no associated Schwann cell hyperplasia. Dr. Shockley said the incidences were not high enough to detect effects. Dr. Corcoran said that he was uncomfortable with this call, as low incidences similar to these occur in numerous findings across the report. He was concerned that too much thought was being put into specific findings relative to following hard evidence. The panel discussed the issue, and then voted 9 yes, 2 no; the motion carried. Dr. Corcoran and Dr. Harkema were the “no” votes. Dr. Corcoran voted no because he did not believe there was enough of a signal to elevate the call to equivocal. Dr. Harkema said he was equivocal about the upgrade itself.

L.4.10.3. GSM-Nonneoplastic Lesions

The panel next addressed the conclusions regarding nonneoplastic lesions in the GSM-treated males and females. The vote was divided by sex. For the males, Dr. Felter moved to accept the conclusion as written; Dr. Andrews-Jones seconded, and the panel voted 11 yes, 0 no. The motion carried. For the females, Dr. Andrews-Jones moved to accept the conclusion as written; Dr. Adler seconded, and the panel voted 11 yes, 0 no, and the motion carried.

L.4.10.4. CDMA-Exposed Males

The panel moved on to consider the conclusions for the male rats exposed to CDMA modulation cell phone RFR at 900 MHz.

The first draft conclusion was *some evidence of carcinogenic activity* due to incidences of malignant schwannoma in the heart. Dr. Wyde explained the rationale as being very similar to what had been seen in the GSM modulation: a statistically significant increase in the high-dose group and a statistically significant positive trend. Dr. Adler asked what influenced the decision to call for *some evidence* instead of *clear evidence*. Dr. Blystone responded that the rationale was the same as with the GSM modulation. Dr. Whiteley said a dose-related increase in latency was apparent and asked if that was considered. Dr. Wyde said yes. Dr. Andrews-Jones moved to upgrade the conclusion to *clear evidence of carcinogenic activity*. Dr. Felter seconded. The panel voted 8 yes, 3 no. The “no” votes were Drs. Malys, Cline, and Harkema. Dr. Malys said that the reasons for his no vote were the same as he had expressed for the GSM modulation. He noted that making some of these judgements would have been easier if the work had been done to integrate the information from the rats and mice and different modulations. Dr. Cline said he had voted no due to his concern about the low incidences. He said that he was comfortable with *some evidence* but not *clear evidence*. Dr. Harkema explained his no vote as stemming from the control issues.

The next conclusions were under "May have been related to cell phone RFR exposure (*equivocal evidence of carcinogenic activity*)." The first was for incidences of malignant glioma in the brain. Dr. Wyde explained the rationale for the call: the only incidences were in the high-dose group and the incidences were within the historical control range, but hyperplasia was observed. Dr. Lin asked if the historical control data for the brain were continually updated. Dr. Blystone said only three historical control studies could be used for the brain due to differences in histopathology sectioning (seven sections). Dr. Andrews-Jones moved to upgrade the call to *some evidence of carcinogenic activity* based on looking at the response observed across all groups (both modalities, both sexes). Dr. Rinke seconded the motion. The panel voted 6 yes, 4 no, 1 abstain, so the motion carried. The "no" votes were Drs. Malys, Corcoran, Harkema, and Cline. Dr. Adler abstained. Dr. Malys explained his no vote as due to lack of exceeding historical controls and the reasons he had discussed previously. Dr. Corcoran explained his no vote by reiterating the four items that had been discussed on a recurring basis for the negative votes for upgraded classification. Dr. Harkema, the third no vote, agreed, as did Dr. Cline, the fourth no vote. Dr. Adler, who abstained, said he could not decide on a yes or no vote based on the current information.

Continuing with the CDMA males, the next conclusion under *equivocal evidence* was "Incidences of adenoma of the pars distalis in the pituitary gland." Dr. Wyde explained the call, and that the rationale was that the only response occurred at the mid-dose. Dr. Felter moved to accept the conclusion as written; Dr. Adler seconded. The vote was 11 yes, 0 no, so the motion carried.

The final equivocal call for the CDMA males was "Incidences of adenoma or carcinoma (combined) of the liver." Dr. Wyde explained the rationale for the call, which was that the incidence at 3 W/kg exceeded the historical control but was not statistically significant, and there was no dose response. Dr. Corcoran moved to accept the conclusion as written; Dr. Andrews-Jones seconded. The vote was 11 yes, 0 no, so the motion carried.

L.4.10.5. CDMA-Exposed Females

For female Sprague Dawley rats exposed to CDMA modulated cell phone RFR, the initial conclusions were for *equivocal evidence of carcinogenic activity*. Starting with "incidences of malignant glioma in the brain," Dr. Wyde explained the call. Dr. Wyde said that the rationale was that the only incidences were in the lowest exposure group, the incidences were not statistically significant nor was there a positive trend, and the incidences were very close to the historical control range. Dr. Andrews-Jones moved to upgrade the call to *some evidence* for the same reasons cited for the gliomas in the other groups. Dr. Felter seconded, citing the grade of 2.0 for the hyperplasia in the glial cells, which was high compared to what was seen elsewhere. The vote was 4 yes, 6 no, 1 abstain, so the motion did not carry. The "no" votes were Drs. Malys, Corcoran, Harkema, Whiteley, Cline, and Adler. Dr. Rinke abstained. Drs. Malys, Corcoran, Harkema, Whiteley, and Cline explained their no votes as stemming from lack of statistical significance. Dr. Adler explained his no vote was because he felt that the data needed to be looked at in totality, and the patterns needed to be considered. Dr. Rinke explained his abstention based on indecision about whether raising the call was appropriate. Dr. Corcoran moved to accept the original conclusion as written; Dr. Harkema seconded. The vote was 8 yes, 3 no. The "no" votes were Drs. Felter, Pant, and Andrews-Jones. Dr. Felter voted no due to biological plausibility combined with the overall weight of evidence from all of the data and the degree of

severity of the hyperplasia. Dr. Pant, the second no vote, agreed, as did Dr. Andrews-Jones, the third no vote.

Dr. Andrews-Jones moved to add "incidences of malignant schwannoma in the heart" to the *some evidence* category. Dr. Felter seconded. The panel voted 4 yes, 6 no, 1 abstain, so the motion failed. Drs. Andrews-Jones, Felter, Whiteley, and Pant voted in favor of the motion; Dr. Rinke abstained. Dr. Rinke said he would agree with an equivocal call as there was no positive trend. Dr. Rinke moved to add the heart schwannoma to the *equivocal evidence* category. Dr. Corcoran seconded. Dr. Harkema asked why this did not rise to *equivocal evidence* in the first place. Dr. Wyde said it was because the incidences were low and there was no statistical significance. The vote was 9 yes, 2 no. The "no" votes were Dr. Malys and Dr. Harkema. Dr. Malys explained his no vote as being due to the relatively low incidence and a lower severity score in the higher dosage group. Dr. Harkema explained his no vote as stemming from trouble with the controls.

Still within the equivocal category, the panel moved on to "incidences of pheochromocytoma (benign, malignant, or complex combined) in the adrenal medulla." Dr. Wyde explained the rationale behind the conclusion: the highest incidence was in the low-dose group and the incidence was within or right at the high end of the historical control range. Dr. Corcoran moved to accept the conclusion as written; Dr. Adler seconded. The vote was 10 yes, 0 no, 1 abstain. Dr. Felter, who abstained, said she was considering what had been seen in some of the adrenal glands from the other studies, and was not convinced it did not rise to the level of *some evidence*.

L.4.10.6. CDMA-Nonneoplastic Lesions

As with the GSM modulation, the panel elected to split the calls on nonneoplastic lesions between the males and females.

Dr. Andrews-Jones moved to accept as written the conclusion for males; Dr. Corcoran seconded. The vote was 11 yes, 0 no, so the motion carried.

Dr. Felter moved to accept as written the conclusion for females; Dr. Andrews-Jones seconded. The vote was 11 yes, 0 no, so the motion carried.

L.5. Final Conclusions

The final list of conclusions recommended by the panel for the RFR studies in rats follows:

L.5.1. Technical Report TR 595: Cell Phone Radiofrequency Radiation Studies in Rats

L.5.1.1. GSM Modulation

Male Hsd:Sprague Dawley® SD® rats, exposed to GSM-modulated cell phone RFR at 900 MHz

- Clear evidence of carcinogenic activity
  - Incidences of malignant schwannoma in the heart
- Were considered to be related to cell phone RFR exposure (some evidence)
  - Incidences of malignant glioma in the brain

  - Incidences of pheochromocytoma (benign, malignant, or complex combined) in the adrenal medulla
- May have been related to cell phone RFR exposure (equivocal evidence)
  - Incidences of adenoma or carcinoma (combined) in the prostate gland
  - Incidences of benign or malignant granular cell tumors in the brain
  - Incidences of adenoma in the pars distalis of the pituitary gland
  - Incidences of pancreatic islet cell adenoma or carcinoma (combined)

Female Hsd:Sprague Dawley® SD® rats, exposed to GSM-modulated cell phone RFR at 900 MHz

- Equivocal evidence of carcinogenic activity
  - Incidences of malignant schwannoma in the heart

Increases in nonneoplastic lesions in the heart, brain, and prostate gland of male rats occurred with exposures to GSM cell phone RFR at 900 MHz.

Increases in nonneoplastic lesions in the heart, thyroid gland, and adrenal gland of female rats occurred with exposures to GSM cell phone RFR at 900 MHz.

L.5.1.2. CDMA Modulation

Male Hsd:Sprague Dawley® SD® rats, exposed to CDMA-modulated cell phone RFR at 900 MHz

- Clear evidence of carcinogenic activity
  - Incidences of malignant schwannoma in the heart
- Were considered to be related to cell phone RFR exposure (some evidence)
  - Incidences of malignant glioma in the brain
- May have been related to cell phone RFR exposure (equivocal evidence)
  - Incidences of adenoma in the pars distalis of the pituitary gland
  - Incidences of adenoma or carcinoma (combined) in the liver

Female Hsd:Sprague Dawley® SD® rats, exposed to CDMA-modulated cell phone RFR at 900 MHz

- Equivocal evidence of carcinogenic activity
  - Incidences of malignant glioma in the brain
  - Incidences of malignant schwannoma in the heart
  - Incidences of pheochromocytoma (benign, malignant, or complex combined) in the adrenal medulla.

Increases in nonneoplastic lesions of the heart, brain, and prostate gland in male rats occurred with exposures to CDMA cell phone RFR at 900 MHz.

Increases in nonneoplastic lesions of the brain in female rats occurred with exposures to CDMA cell phone RFR at 900 MHz.

Dr. Eaton opened the floor to any final comments from the panel members.

Dr. Andrews-Jones recommended that NTP follow up on Dr. Malys' remarks about nonlinearity and on the endocrine system as a whole. Dr. Malys added to his comments about data integration and recommended, as NTP moves forward with the results of these studies, that approach should be kept in mind, as the pressure for data integration will only increase.

Dr. Adler asked if the panel would receive a copy of the revised report. Dr. Bucher explained how the process would proceed, culminating in a final version accepted by the NTP director and made public at that point. Dr. Wolfe added that there would be a peer-review meeting report to be shared with the panelists to ensure that their comments were captured accurately.

L.5.2. Adjournment

Dr. Eaton thanked the panel for its work on a very complicated, challenging, and important study. He adjourned the meeting at 3:38 pm, March 28, 2018.

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