SUMMARY OF DATA FOR CHEMICAL SELECTION

DIAZOAMINOBENZENE

136-35-6

NOMINATION HISTORY

Nomination Source:	NIEHS. In-house Chemical Screening/Selection Group; previously nominated by NIOSH for *Salmonella* Testing.
Date of Nomination:	December, 1987
Recommendations:	Toxicological testing
Rationale / Comments:	_Potential for worker exposures based on wide use in scientific laboratories. _Positive in *Salmonella*. _Lack of adequate chronic testing; existing carcinogenicity studies (oral) were equivocal.

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CHEMICAL IDENTIFICATION

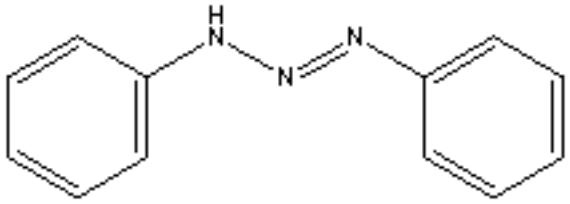
CAS Registry Number: 136-35-6

Chemical Abstracts Name: 1-Triazene, 1,3-diphenyl- (9CI)

Synonyms and Trade Names: Anilinoazobenzene; benzeneazoanilide; benzeneazoaniline; *p*-diazoaminobenzene; 1,3-diphenyltriazene; 1,3-diphenyl-1-triazene; N-(phenylazo)aniline; Porofor DB; DPT; DAAB

Structure, Molecular Formula and Molecular Weight:

(see p.2 of Executive Summary in Central Files)



$C_{12}H_{11}N_3$ Mol. wt.: 197.11

Chemical and Physical Properties

Description: Golden-yellow, small crystals or powder (Aldrich Chemical Co., Inc., 1995a; Budavari, 1989)

Boiling Point: 146˚C (Aldrich Chemical Co., Inc., 1994) [Explosion hazard when heated; see below.]

Melting Point: 98˚C (Lide, 1993)

Solubility: Insoluble in water (Budavari, 1989); soluble in ethyl alcohol, ethyl ether, benzene, pyrimidine and hexane (Lide, 1993; Eastman Chemical Co., 1993)

Vapor Density: 6.8 (Aldrich Chemical Co., Inc., 1995a)

Stability: Decomposition starts at 130˚C with major decomposition at 188˚C (Aldrich Chemical Co., Inc., 1995a); thermal decomposition products include benzene, *o*-aminodiphenyl, *p*-aminodiphenyl, diphenylamine and azobenzene (Mortimore *et al.*, 1979)

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Reactivity:	Incompatible with strong oxidizing agents; explodes when heated to 150˚C (Budavari, 1989); strongly explosive when shocked ; when heated to decomposition emits toxic fumes of NO_x (Sax & Lewis, 1989).
Technical Products and Impurity:	Diazoaminobenzene is available from Aldrich Chemical Company, Inc., at a purity level of 95% (Aldrich Chemical Co., Inc., 1995b).

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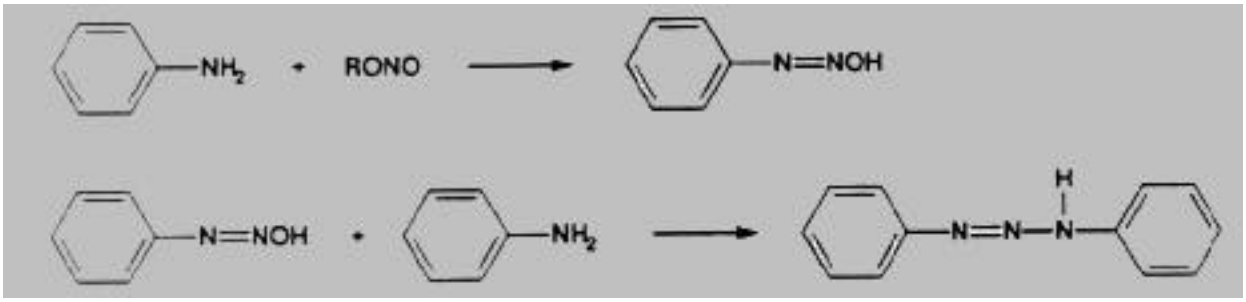
EXPOSURE INFORMATION

Production and Producers: Diazoaminobenzene (DAAB) can be made by diazotizing aniline dissolved in hydrochloric acid with sodium nitrite and then adding a concentrated solution of sodium acetate (Budavari, 1989). It can also be made by the interaction of nitrous acid and an alcoholic solution of aniline (Lewis, 1993). Smith and Ho (1990) reported that DAAB could be conveniently prepared by the rapid reaction of aniline with isoamyl nitrite with a product yield of 67%. They described the reaction by the following general scheme, where R = isoamyl:



DAAB is listed in the EPA's TSCA Inventory (STN International, 1995). No information was found reporting annual production volumes for DAAB. Barnhart (1982) listed Mobay Chemical (Bayer) as the sole manufacturer of DAAB as a blowing agent for rubber and plastics. Several producers/suppliers of this chemical were listed in chemical industry catalogs or directories; the following companies advertise the availability of DAAB in the United States (Aceto Corporation, 1994; Hunter, 1994; Aldrich Chemical Co., Inc., 1995b; Eastman Chemical Co., 1993):

Aldrich Chemical Company, Inc.
Eastman Chemical Company
Molecular Rearrangement International (MRI)
Pfaltz & Bauer, Div. of Aceto Corporation

One catalog supplier, Eastman, sells DAAB in bulk quantities (Eastman Chemical Co., 1993). The lack of information identifying producers of DAAB in chemical product directories may indicate that this chemical primarily occurs as a non-isolated intermediate formed and used in closed systems.

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Use Pattern: DAAB has three major use areas: intermediate, complexing agent, and polymer additive. Use as an intermediate is reported in several industry sectors, including organic synthesis, dye manufacture, and agrochemical manufacture (insecticides) (Lewis, 1993). For example, DAAB has been reported to be formed as a non-isolated intermediate in the preparation of iodobenzene from aniline (Smith & Ho, 1990). DAAB is also a versatile metal complexing agent (Barker *et al.*, 1991). In addition, it has been reported to show semiconducting properties and to be useful as a dopant for poly(methyl methacrylate) (PMMA) in semiconductor manufacture (Bolle *et al*., 1990; Shaaban *et al*., 1993).

As a polymer additive DAAB has been included in a patent as a coupler to promote adhesion of natural rubber to steel (Shemenski & Starinshak, 1982). In addition, Barnhart (1982) lists DAAB as a chemical blowing agent. DAAB is patented for use as a blowing agent in a variety of polymer systems, including the following:

- foamed or fiber-reinforced elastomeric resins (synthetic rubbers, phenolics) (Kumasaka & Horikoshi, 1983; Raad, 1993)
- heat-curable epoxy and urea-formaldehyde adhesive resins (Vankova & Soucek, 1988)
- polyurethane foam coating materials (Mochihara *et al*., 1987)
- thermal transfer inks and electrophotographic toners (polyesters/acrylics) (Shimura, 1988; Yamada *et al*., 1988).

Human Exposure: DAAB is reported to be hazardous to handle due to the potential for dangerous chemical reactions, including deflagration. The use of personal protective equipment, including NIOSH-approved respirator, chemical-resistant gloves, safety goggles and other protective clothing, to ensure minimization and control of human exposures has been recommended by the Aldrich Chemical Co., Inc., (1995a). Grewer and Klais (1987) reported on a number of accidents in the chemical industry caused by pressure rise associated with deflagration; DAAB was cited as one of the chemicals involved in this kind of hazard to workers although specific accidental exposures to the chemical in these incidents was not addressed.

The presence of DAAB as an impurity in certain food and cosmetic dyes could result in very low level consumer exposures by the oral or dermal route. DAAB has been identified as an impurity in the monoazo color additive, D & C Red No. 33 (CI 17200), which is

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permitted in the United States for use in ingested drugs, lipsticks, and externally applied drugs and cosmetics (Bailey, 1985). Bailey reported detecting DAAB in 9 of 11 samples of commercial products analyzed by reverse-phase high-performance liquid chromatography (HPLC); levels of up to 439 ppb were reported, with an average level of 99 ppb. Levels of DAAB detected in two "pharmacology samples" were reported as 68 and 110 ppb. DAAB has also been reported as a trace contaminant in FD&C Yellow No. 5, which is reportedly used in dietary products, including candy, beverages, frozen dairy desserts and baked goods (Palmer & Mathews, 1986). The Food and Drug Administration (FDA) in 21 CFR Part 74, issued a final rule amending the color additive regulations for FD&C Yellow No. 5 in foods and ingested drugs to permit an impurity level of DAAB of not more than 40 ppb. FD&C Yellow No. 5 is also permitted as an additive in externally applied drugs and cosmetics (Federal Register, 1986).

According to Noda and coworkers (1981) nitrites ingested by humans can release nitrite ions in the gastrointestinal tract or other biological fluids which can react with various amines giving rise to biologically active substances, including triazenes. This would suggest that exposures to aniline in humans who have nitrite ions present in their bodies could possibly result in biological transformations of the amine to DAAB; however, the concentrations involved make it highly improbable.

DAAB is not listed in the National Occupational Exposure Survey (NOES), which was conducted by the National Institute for Occupational Safety and Health (NIOSH) between 1981 and 1983.

Environmental Occurrence: DAAB has not been reported to occur naturally. No information was found in the available literature on occurrences of DAAB in environmental media.

Regulatory Status: No standards or guidelines have been set by NIOSH or OSHA for occupational exposure to or workplace maximum allowable levels of DAAB. The American Conference of Governmental Industrial Hygienists (ACGIH) has not recommended a Threshold Limit Value (TLV) or Biological Exposure Index (BEI) for this compound.

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EVIDENCE FOR POSSIBLE CARCINOGENIC ACTIVITY

Human Data: No epidemiological studies or case reports investigating the association of exposure to DAAB and cancer risk in humans were identified in the published literature.

Toxicological effects of chronic exposure to humans have not been thoroughly investigated, but acute dermal effects that have been reported include eye and skin irritation and central cyanosis unresponsive to oxygen therapy. Inhalation of DAAB causes irritation of the mucous membranes and upper respiratory tract, and dyspnea and tachypnea may occur. Ingestion can induce nausea and vomiting. DAAB is reported to be capable of producing methemoglobinemia following inhalation, skin absorption, or ingestion (Aldrich Chemical Co., Inc., 1995a; NLM, 1995a).

Animal Data: No 2-year carcinogenicity studies of DAAB in rats were identified in the published literature. However several older studies investigating sub-chronic/chronic effects of DAAB in mice dosed by the subcutaneous, oral, or dermal route, were reported. DAAB is an experimental tumorigen according to RTECS criteria (NLM, 1995b).

Shear and Stewart (1941) [unavailable] reported that no tumors were observed after repeated subcutaneous injections in strain A mice (PHS-149, 1963).

Kirby (1947a) reported that when 10 male and 10 female mice were injected subcutaneously with DAAB at 3 dose levels (2, 4 and 10 mg) in arachis oil over a period of up to 190 days, both males and females developed toxic nephritis. One mouse also developed marked hyaline (waxy) degeneration of the kidney, liver, and spleen; but the sites of injection had only cystic spaces with fibrous walls. One female mouse developed an anaplastic mammary tumor with metastases in the spleen, liver, and kidney; but the tumor was considered to be spontaneous. One mouse that survived until 329 days after the first injection had a hematoma near the injection site, but no sarcomas were seen in any mouse.

Otsuka administered 0.04 percent DAAB in olive oil in a diet consisting of olive oil (2 percent), unpolished rice (97 percent), and honey (1 percent) supplemented with some greens at dose levels ranging from 0.018-0.456 grams to 30 male and female mice; forestomach epithelial proliferation (hyperkeratosis and hyperplasia) developed in 22 mice

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that survived for 59 days, and 11 that survived 307-483 days showed papillomatous growths (Otsuka, 1935). No controls of any kind were reported by the author.

Kirby (1947a) administered DAAB at a dose of 50 mg/100 g basal diet to six male and six female mice. Two male mice and all the females were dead in 39 days, usually with degenerative changes in the liver and acute toxic damage to the kidneys affecting mainly the convoluted tubules. There were no signs of changes in the stomachs of the dead mice or those that survived 125 days. Additional male mice fed 100 mg DAAB/100 gm basal diet developed degenerative changes in the liver, but no stomach abnormalities were observed. Seven male and three female mice fed DAAB at a dose of 100 mg/100g of protein-restricted diet containing arachis oil also developed kidney and liver damage, but no stomach lesions were observed. No controls were reported for this study either.

In a dermal study, Kirby (1947a) painted the interscapular region of male and female mice with increasing concentrations of DAAB. The painting was begun on 19 mice (10 male, 8 female, 1 sex unreported) with a 0.5% solution of DAAB in acetone; after a time the strength was increased to 1%, later to 2% and finally to 5%. Degenerative changes in the liver and kidneys as well as degeneration in the spleens were observed in 11 mice (6 male and 5 female) succumbing before completion of the treatment. In the group of eight surviving mice (4 male, 3 female, 1 sex unreported) painted with 5% solution, squamous carcinoma developed in one male and one female. Six of the eight also developed horny upgrowths at the site of painting and the other two showed ulceration and marked hyperkeratosis. No controls in which acetone alone was applied dermally to mice were reported by the author. In another experiment, Kirby (1948) also applied a 5 percent solution of DAAB dissolved in a solution of 0.5 percent croton oil in acetone to the necks of stock mice three times a week for 545 days; croton oil was discontinued after 15 days and reintroduced after 167 days in one-third of the mice. In those mice given only initial croton oil, hyperkeratosis, with or without necrosis or ulceration, was common after 200 days, and the first skin papilloma was found in a mouse killed 320 days after painting. In 16 mice surviving more than 300 days' painting with 5 percent DAAB in acetone, three developed simple papilloma and three others developed squamous carcinoma at the site of painting. In six mice that were given DAAB initially plus subsequent croton oil and survived 300 days painting, three developed squamous papilloma and one developed squamous carcinoma at the treatment site. No controls in which either acetone or croton oil alone was applied dermally to mice were reported by the author. In a summary of both

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treatment groups, Kirby stated that, of 17 mice painted for more than 400 days, 5 developed low-grade squamous carcinoma and 5 others developed squamous papilloma at the site of painting. No metastases or spindle-cell metaplasias were observed. Three primary lung tumors (one adenoma in each group and one adenocarcinoma) that may have been spontaneous were observed. Kirby reported that croton oil painted concurrently with DAAB caused no increase in the incidence of skin tumors, nor did it increase the latent period. He concluded that his studies had confirmed the carcinogenicity of DAAB for mouse skin.

Kirby (1947b) reported that earlier Japanese studies, including Otsuka (1935), which appeared to demonstrate the carcinogenicity of azo dyes in rats were inadequate. The experiments were carried out using unbalanced rice diets and were uncontrolled with regard to constituents. No carcinogenicity studies more recent than Kirby's were found in the published literature, and the adequacy of the Kirby studies as a basis for classifying DAAB as to carcinogenicity is questionable due to small numbers of animals and lack of controls.

Short-Term Tests: There is limited information in the available literature on the genotoxicity of DAAB. DAAB was tested for mutagenic activity in the Ames/*Salmonella* assay. Zeiger and coworkers (1987) reported that this chemical, when tested with preincubation over a dose range of 0.1-10 µg/plate, did not induce an increase in the frequency of reversion in *S. typhimurium* strains TA100, TA1535, TA1537, and TA98 without metabolic activation. When tested over a dose range of 0.3-33 µg/plate, DAAB was mutagenic in *S. typhimurium* TA100, TA1537, and TA98, but not in TA1535 at 1-100 µg/plate, with metabolic activation.

Grant (1982) tabulated the results of a study by Kundu (1977) [unavailable] that utilized chromosomal aberrations in root tips of the onion *Allium cepa*. Although Grant did not specify the end point(s) (such as chromosome breaks and exchanges) used by Kundu, he reported that 1,250 ppm DAAB induced chromosome aberrations in *A. cepa*.

Metabolism: No studies on the metabolism of DAAB were found in the published literature. However, Williams (1959) reported that azo compounds can undergo a reaction sequence *in vivo* in which reduction of the azo link forms a hydrazo compound which can then be split to yield two aromatic amines. Since this pathway involves a flavin-dependent enzyme,

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a sufficient level of riboflavin as well as the presence of bacteria may be required for the transformation to occur.

Structure/Activity Relationships: Four triazenes structurally similar to diazoaminobenzene were screened for relevant information associating these related compounds with a mutagenic or carcinogenic effect. A summary of relevant data found in the published literature for three of these triazenes, 3,3-dimethyl-1-phenyltriazene (CAS RN 7227-91-0), azobenzene (CAS RN 103-33-3), and *p*-aminoazobenzene (CAS RN 60-09-3), is presented in Table 1 (2 pages); no data pertaining to these effects were found for a fourth compound 1,3-diphenyltriazene 1-oxide (CAS RN 3639-37-0).

All three of these structurally related chemicals shown in Table 1 exhibited some carcinogenic activity in laboratory animals. 3,3-Dimethyl-1-phenyltriazene induced mainly nervous system tumors by transplacental administration in rats. Azobenzene was sarcomagenic in the spleen of male and female rats and has been listed as a suspected cancer agent by Janssen Chimica. *p*-Aminoazobenzene induced a variety of tumors (bile duct, injection site, liver, and skin) when administered by several different routes to rats and mice. 3,3-Dimethyl-1-phenyltriazene also induced teratogenic effects when given as a single ip injection to rats.

Information on mutagenicity was also found for three of the structurally related compounds. In general, there is good evidence that 3,3-dimethyl-1-phenyltriazene is a gene mutagen. This compound was positive in *S. typhimurium* TA100 with S9 and several other mutagenicity studies which included the ability to induce SCEs and unscheduled DNA synthesis. However, it was negative in another study in *S. typhimurium* TA100 and five other strains with and without S9. Approximately half of the mutagenicity studies that are listed in Table 1 for azobenzene and *p*-aminoazobenzene were positive including one positive *S. typhimurium* study for each compound.

Table 1. Summary of Toxicological Evidence on Diazoaminobenzene and Structurally Related Compounds

Chemical Name [CAS RN] Structure	Carcinogenicity Data	Mutagenicity Data	Other Related Data
Diazoamino benzene [136-35-6]	caused stomach tumors in mice after administration in diet (Otsuka, 1935) caused skin tumors in mice after dermal application (Kirby, 1947a; Kirby, 1948) did not cause stomach tumors in male or female mice after administration in diet (Kirby, 1947a) did not cause injection site sarcomas in mice after sc injection (Kirby, 1947a)	negative in *S. typhimurium* TA100, TA1537, and TA98 without S9; negative in *S. typhimurium* TA1535 with and without S9; positive in *S. typhimurium* TA100, TA1537, and TA98 (Zeiger, 1987) positive in an *A. cepa* chromosome aberration assay (Grant, 1982)	
3,3-dimethyl-1-phenyl triazene [7227-91-0]	caused brain, peripheral nervous system and spinal cord tumors after gavage as well as sc administration; did not cause transplacental carcinogenesis in rats treated on day 15 of gestation; caused transplacental nervous system carcinogenesis in rats treated on day 23 of gestation (Preussman *et al.*, 1969; Druckrey, 1973 a, b) caused kidney, brain, and nerve tumors in BD rats when given sc or orally (Preussman *et al.*, 1969; 1974) caused transplacental carcinogenic effects in kidney, brain, and teeth (odontoma) when given ip to pregnant Sprague-Dawley rats on days 16, 18 and 20 of gestation (Frank *et al.*, 1992) caused brain, heart, kidney, ear, abdominal cavity tumors, and leukemia after tail vein administration in Sprague-Dawley rats (Kolar & Habs, 1984)	positive in *S. typhimurium* TA100 with S9 (Pool, 1978) negative in *S. typhimurium* TA1535, TA1536, TA1537, TA1538, TA98 and TA100 with and without S9 (Simmon, 1979) positive in a MN assay in mouse bone marrow (Jenssen & Ramel, 1980) positive for SCE *in vivo*; positive in a MN assay (Bauknecht *et al.*, 1977) positive for SCE in V79 cells with and without S9 (pH-dependent direct cytotoxicity) (Thust & Schneider, 1990) positive for dominant lethal mutations in *D. melanogaster* (Buchi, 1977) positive for CA in rat lymphocytes *in vivo* (Newton *et al.*, 1977) positive for *ad-3* mutants in *Neurospora crassa* (Ong & De Serres, 1973) positive for CA in V79 CH lung fibroblasts after 24 hours without S15; positive for CA in V79 lung fibroblasts with S15 for 3 hours (Ochi *et al.*, 1981) loss of ring-shaped X-chromosome in postmeiotic *Drosophila* (Vogel *et al.*, 1993)	induces teratogenic effects including skeletal deformities such as micrognathism, cleft palate, digital malformations, and nervous system hypoplasia when given as a single ip dose to pregnant Sprague-Dawley rats (Frank *et al.*, 1989a,b)

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azobenzene [103-33-3]	caused sarcomas in the spleen and other abdominal organs when administered in the diet to male and female F344 rats; negative for carcinogenicity in male and female $B6C3F_1$ mice (NCI, 1979) caused liver cell tumors in male but not female mice when administered by stomach tube; subcutaneous studies in mice and rats were negative, but could not be evaluated because the adequacy of the dose used could not be assessed (IARC, 1975) caused splenic sarcomas and fibrosis in male and female rats with some metastases to the peritoneal cavity and abdominal organs (Goodman *et al.*, 1984) listed as suspected cancer agent (Janssen Chimica, 1992)	positive in *S. typhimurium* TA100 with S9; negative in TA98, TA1535, and TA1537 with and without S9 (Haworth *et al.*, 1983) negative in *S. typhimurium* TA98 with and without S9; positive in TA100 with S9; negative for hepatocyte/DNA repair (Mori *et al.*, 1986) weakly positive for SOS with S9 (Nakamura *et al*., 1987) negative for CA and SCE in CHO cells (Bloom *et al.*, 1982)	NDF
p-aminoazobenzene [60-09-3]	caused hepatoma induction but did not cause bile duct adenocarcinoma or cholangioma when given in a diet containing casein and peeled potatoes as the main protein source to male Wistar rats (Kirby, 1947b) equivocal for adenocarcinoma when given as a single injection into kidney in *Rana pipiens* (Strauss & Mateyko, 1964) did not cause local or liver tumors when injected sc in mice fed a restricted or normal diet (IARC, 1975) caused skin tumors (papillomas, carcinomas and miscellaneous tumors) when painted on the dorsal skin of male albino rats; did not cause liver tumors (Fare, 1966) caused liver tumors in rats when given in diet; carcinogenic in rats when applied to skin; sc studies in mice and rats and ip injection in rats could not be evaluated because of the limited number of animals or inadequate duration of studies (IARC, 1975) caused liver tumors when given by ip injection prior to weaning in $B6C3F_1$ C3H/He and C57BL/6 male mice but did not cause tumors in F344 male rats (Delclos *et al.*, 1984)	negative for the production of X-linked recessive lethals in *Drosophila* (the purity of the compound was not defined) (IARC, 1975) positive in *S. typhimurium* TA98 and TA100 with but not without S9 (Miyagoshi *et al.*, 1985) positive in modified ML (Tk^+/Tk^-) mutation assay with S9 (Amacher & Turner, 1982) positive in *S. typhimurium* strains TA100, TA1538 and TA98 with S9; positive for hepatocyte UDS (Probst *et al.*, 1981; Watanabe & Hashimoto, 1981) negative for DNA repair in *Bacillus subtilis* and *E. coli* systems (Suter & Jaeger, 1982) negative in *S. typhimurium* TA1535 with and without S9 (Rosenkranz & Poirier, 1979)	the urine of rats, mice, or hamsters injected with *para*-aminoazobenzene contained N-hydroxy-N-acetylaminoazobenzene in conjugated form (IARC, 1975)

NDF: No data found; sc: subcutaneous; MN: micronucleus assay; SCE: sister chromatid exchange; CA: chromosomal aberrations; ip: intraperitoneal; ML: mouse lymphoma; UDS: unscheduled DNA synthesis; SOS: SOS function induction as indication of DNA damage to *S. typhimurium*.

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