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*Center for Epidemiology & Computational
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**Comments on the Draft NTP
Monograph on
Immunotoxicity Associated
with Exposure to
Perfluorooctanoic Acid
(PFOA) or Perfluorooctane
Sulfonate (PFOS)**



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Monograph on Immunotoxicity
Associated with Exposure to
Perfluorooctanoic Acid (PFOA) or
Perfluorooctane Sulfonate (PFOS)**

Prepared for

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Appendix A: *Curriculum vitae* of Dr. Ellen T. Chang, Sc.D.

Limitations and Disclosures

These comments summarize work performed to-date and present the findings resulting from that work. The findings presented herein are made to a reasonable degree of scientific certainty. I reserve the right to supplement these comments and to expand or modify opinions based on review of additional material as it becomes available through any additional work or review of additional work performed by others.

The preparation of these comments was supported by the 3M Company. 3M was not directly involved in the preparation of these comments and did not contribute to their content. I have been a consultant to 3M and other industry clients on issues related to perfluoroalkyl and polyfluoroalkyl substances.

Executive Summary

The comments herein address the Draft National Toxicology Program (NTP) Monograph on Immunotoxicity Associated with Exposure to Perfluorooctanoic Acid (PFOA) or Perfluorooctane Sulfonate (PFOS), dated June 6, 2016. In particular, these comments address scientific deficiencies in the hazard classification scheme used by NTP to reach the conclusion that PFOA and PFOS are “presumed to be an immune hazard to humans.” In addition, these comments call attention to the inconsistencies and methodological limitations in the human epidemiologic literature on the immunotoxicity of PFOA and PFOS; these problems further undermine NTP’s conclusion that these substances are “presumed to be an immune hazard to humans.”

The following points are discussed below:

- The hazard classification scheme inappropriately gives equal weight to “factors decreasing confidence” and “factors increasing confidence.” A high risk of bias, which is directly related to the internal validity of a study, should outweigh all other considerations when evaluating the degree of confidence in any scientific study or body of scientific evidence. For example, a large relative risk does not compensate for bias due to insufficient control for confounding, which can produce a spurious association.
- The hazard classification scheme gives undue weight to animal evidence in the absence of compelling human evidence. If the level of confidence in the relevant human evidence is low or inadequate, then an agent should be classified as a “suspected” rather than a “presumed” human health hazard.
- The human epidemiologic evidence on antibody-mediated immune suppression is not consistent, contrary to statements by NTP. If, as asserted by NTP, antibody responses are expected to vary by vaccine type, then responses to different vaccines should be treated as distinct health outcomes. In light of the dissimilarity of the vaccine types tested in published epidemiologic studies and the preponderance of statistically non-significant associations detected, the available epidemiologic evidence of an effect of PFOA or PFOS on the antibody response is inconsistent.

- NTP does not adequately acknowledge the impact of potential confounding on the validity and interpretability of the available epidemiologic studies. Confounding by other perfluoralkyl acids, other potential immunotoxicants or their surrogates, or various uncontrolled sociodemographic or lifestyle factors is likely, such that any of the observed associations with PFOA and PFOS could be spurious.
- Decrements in antibody-mediated immunity do not necessarily correspond with an increased risk of infectious disease. Moreover, most of the available epidemiologic evidence shows no significant association of PFOA or PFOS exposure with infectious disease. Therefore, it is unclear whether a decrement in the antibody response (especially given the lack of consistency of such an effect) without a corresponding increase in infectious disease susceptibility represents an adverse health outcome from a clinical and public-health perspective.

In light of the deficiencies in the hazard classification scheme and the inconsistent, sparse, and methodologically limited body of epidemiologic evidence, the classification of PFOA and PFOS as “presumed to be an immune hazard to humans” is inappropriate. Taking into account the diminished confidence in studies with a high risk of bias, including confounding, along with the inconsistencies and methodological limitations of the body of epidemiologic evidence, PFOA and PFOS arguably should be classified as “suspected to be an immune hazard to humans.”

My *curriculum vitae* is attached as Appendix A.

1. The hazard classification scheme inappropriately gives equal weight to “factors decreasing confidence” and “factors increasing confidence.”

NTP assesses the risk of bias in individual studies using a tool¹ developed by the NTP Office of Health Assessment and Translation (OHAT) to facilitate a standardized, systematic consideration of risk of bias across lines of scientific evidence (Table 3 of the Draft Monograph). Risk-of-bias questions are as follows:

1. Was administered dose or exposure level adequately randomized? (relevant to experimental animal studies, *in vitro* experimental studies, and human controlled trials)
2. Was allocation to study groups adequately concealed? (relevant to experimental animal studies, *in vitro* experimental studies, and human controlled trials)
3. Did selection of study participants result in the appropriate comparison group? (relevant to cohort, case-control, and cross-sectional studies)
4. Did study design or analysis account for important confounding and modifying variables? (relevant to cohort, case-control, and cross-sectional studies and case series)
5. Were experimental conditions identical across study groups? (relevant to experimental animal studies and *in vitro* experimental studies)
6. Were research personnel blinded to the study group during the study? (relevant to experimental animal studies, *in vitro* experimental studies, and human controlled trials)
7. Were outcome data complete without attrition or exclusion from analysis? (relevant to experimental animal studies, *in vitro* experimental studies, human controlled trials, and cohort, case-control, and cross-sectional studies)
8. Can we be confident in the exposure characterization? (relevant to all studies)
9. Can we be confident in the outcome assessment (including blinding of outcome assessors)? (relevant to all studies)
10. Were all measured outcomes reported? (relevant to all studies)
11. Were there no other potential threats to internal validity? (relevant to all studies)

¹ https://ntp.niehs.nih.gov/ntp/ohat/pubs/riskofbiastool_508.pdf

Answers to these questions are used to classify studies as having “definitely low risk of bias” where there is direct evidence of low risk-of-bias practices; “probably low risk of bias” where there is indirect evidence of low risk-of-bias practices or it is deemed that deviations from low risk-of-bias practices for these criteria during the study would not appreciably bias results, including consideration of direction and magnitude of bias; “probably high risk of bias” where there is indirect evidence of high risk-of-bias practices or there is insufficient information provided about relevant risk-of-bias practices; or “definitely high risk of bias” where there is direct evidence of high risk-of-bias practices (Table 4 of the Draft Monograph).

Risk of bias is directly related to the internal validity of a study—that is, whether an observed association between an exposure and an outcome in a study is likely to be credible or spurious. In the presence of a high risk of bias, the results of a given study cannot be relied upon as being valid. Consequently, if studies in a given scientific line of evidence are rated overall as having a high risk of bias, then their collective results cannot be trusted; in other words, a high risk of bias across a body of evidence renders that evidence essentially uninformative.

Given the central importance of validity in the consideration of any body of evidence, NTP’s confidence rating scale (Figure 2 of the Draft Monograph; NTP 2015, Step 5), which apparently gives equal weighting to factors decreasing confidence (risk of bias, unexplained inconsistency, indirectness, imprecision, and publication bias) and factors increasing confidence (large magnitude of effect, dose response, residual confounding, consistency, and other factors such as particularly rare outcomes), is inappropriate. Risk of bias, that is, scientific validity, should outweigh any other considerations in determining the degree of confidence in a body of scientific evidence. In the absence of scientific validity, none of the other factors considered as increasing or decreasing confidence are relevant:

- Consistent findings across animal models or species, across dissimilar populations, or across study design types may well be due to similar biases operating in multiple scenarios, whereas “unexplained inconsistency” may be due to different sources of bias in each scenario.

- Indirectness or lack of applicability of a given exposure or outcome is inconsequential, because untrustworthy results should not be used for extrapolation to other exposures or endpoints.
- The precision or imprecision of an inaccurate estimate is meaningless.
- Publication bias is a lesser concern than whether published results are incorrect.
- A large magnitude of effect or an apparent dose-response trend cannot be trusted to reflect reality.
- Residual confounding is simply one form of bias that can invalidate results and can contribute to a high risk of bias.
- Studies of rare outcomes, if invalid, are uninformative.

Thus, in the presence of a probably or definitely high risk of bias, it is unreasonable to rate the overall confidence in a body of scientific evidence as either high or moderate; confidence should be rated as low or very low, regardless of other factors that may increase or decrease confidence.

This issue is directly relevant to NTP's rating of overall confidence in experimental animal studies of antibody response for PFOA and PFOS, which in turn impacts NTP's hazard classification of the immunotoxicity of these chemicals. Specifically, NTP reports: "Half of the studies were rated probably high or definitely high risk of bias for exposure characterization (one of the Key Questions) due to use of PFOA <98% purity without independent confirmation. In addition, all of the studies in mammals were rated probably high risk of bias for lack of allocation concealment and lack of researcher blinding during the study." For PFOS, NTP states: "All studies were rated probably high risk of bias for outcome assessment due to lack of blinding of outcome assessors, one of the Key Questions. In addition, all of the studies were rated probably high risk of bias for lack of allocation concealment and lack of researcher blinding during the study." These ratings reflect what should properly be characterized as a low level of confidence in the animal evidence for an effect of PFOA or PFOS on the antibody-mediated immune response. Yet according to NTP's confidence rating scale, the high risk of bias is balanced out by evidence of a dose-response trend for both PFOA and PFOS, leading to a final confidence rating of "high" for the animal evidence (Tables 12 and 20 of the Draft

Monograph). As stated above, an apparent dose-response trend based on invalid data is not interpretable; thus, it is not scientifically reasonable for evidence of a dose-response trend to counterbalance a high risk of bias, or for a high or even moderate level of confidence to be placed in data that are likely to be biased.

If the animal data were more reasonably rated as having a low level of confidence due to the high risk of bias, then the final hazard conclusion regarding an effect of PFOA or PFOS on the antibody response, based on the moderate level of confidence in the human data and the low level of confidence in the animal data would be “*suspected to be an immune hazard to humans,*” rather than “presumed to be an immune hazard to humans.” If, however, the animal data were rated as having a moderate level of confidence due to the high risk of bias, then the final hazard conclusion, taking into account the moderate level of confidence in the human data, would be “*presumed to be an immune hazard to humans.*” Thus, the subjective impact of a high risk of bias on the confidence rating for a body of evidence has important implications in terms of the overall hazard classification for PFOA and PFOS.

2. The hazard classification scheme gives undue weight to animal evidence in the absence of compelling human evidence.

NTP's classification of confidence in the evidence for an effect of PFOA on hypersensitivity (Table 17 of the Draft Monograph) reveals another major deficiency in NTP's hazard classification scheme, namely, the undue weight given to the animal literature without supportive human literature for the evaluation of human health hazards. According to the hazard classification scheme used by NTP (Figure 4 of the Draft Monograph), low or inadequate confidence in the human literature, combined with high confidence in the animal literature, is sufficient for an agent to be classified as having a "presumed" human health effect—the same category assigned to agents with moderate confidence in both the human and animal literature, or moderate confidence in the human literature and high confidence in the animal literature.

The classification of a substance as "presumed" to be a human health hazard despite low or inadequate confidence in the relevant human evidence is inappropriate, regardless of the strength of the animal evidence. This hazard classification means that in the absence of sufficient or valid human data, accrual of animal data is sufficient to establish a substance as a "presumed" human health hazard. Yet numerous examples exist, as in the cases of thalidomide, saccharin, and untold numbers of new pharmaceutical compounds evaluated in early-phase clinical trials, where toxicity data in animals were found not to apply to humans. Thus, a more appropriate classification of an agent based on low or inadequate confidence in the human data, combined with high confidence in the animal data—as in the case of the relationship between PFOA exposure and hypersensitivity—is as a "suspected" rather than a "presumed" human health hazard.

3. The human epidemiologic evidence on antibody-mediated immune suppression is not consistent, contrary to statements by NTP.

NTP claims that the human data on the association between PFOA or PFOS and antibody response are “consistent,” based on “suppression in at least one measure of the anti-vaccine antibody response across multiple studies.” At the same time, NTP states that “different responses to different vaccines are expected and often observed in human and experimental animal data as antigens such as vaccines may stimulate different components of the immune system. The strength of an antibody response in terms of antibody level and length of time that an elevated/effective antibody response is maintained is known to differ across vaccines.” NTP acknowledges that only one vaccine, tetanus, was tested in more than one study, and that maternal serum PFOA and PFOS concentrations were not significantly associated with child anti-tetanus toxoid antibody levels in either study (Grandjean et al. 2012; Granum et al. 2013). If, as asserted by NTP, antibody responses are expected to differ by vaccine type, then each anti-vaccine antibody level should be evaluated as a distinct outcome, unless a uniform or nearly uniform pattern of antibody suppression is detected across all vaccine types, which is not the case.

Given the non-comparability of the particular vaccine types tested in each study and the heterogeneity of the associations detected—with the majority of associations tested in every study being statistically non-significant—the assertion that findings were “consistent” due to the detection of suppression of at least one measure of the anti-vaccine antibody response in each study amounts to cherry-picking of the literature. That is, focusing on one or a few significant findings from each study, while ignoring the majority of findings that were null, does not constitute a balanced evaluation of consistency. In fact, if antibody responses to different vaccines are considered as separate outcomes, then no significant association with any specific antibody response was observed in more than one study. This is the opposite of consistency.

NTP’s emphasis on selected statistically significant associations from each study is especially concerning when considered in light of multiple hypothesis testing and false positive rates. That

is, some studies tested a large number of associations (Grandjean et al. 2012; Looker et al. 2014; Stein et al. 2016), thereby increasing the expected number of statistically significant associations that would be expected by chance. Conversely, other studies included a relatively small number of subjects (Granum et al. 2013; Kielsen et al. 2016; Stein et al. 2016), thereby increasing the false positive rate, i.e., the probability that any observed statistically significant association is due to chance (Button et al. 2013). Moreover, multivariate regression models based on sparse data are known to be prone to yielding overestimated relative risks that are biased away from the null, a phenomenon known as sparse-data bias or small-sample bias (Greenland et al. 2000; Sullivan and Greenland 2013). The selective focus on isolated positive findings creates a distorted and misleading portrayal of the epidemiologic evidence (Kavvoura et al. 2007), thereby hindering proper interpretation of the overall consistency of results.

4. NTP does not adequately acknowledge the impact of potential confounding on the validity and interpretability of the available epidemiologic studies.

Key potential confounders of associations between PFOA or PFOS and immune outcomes in humans are identified by NTP as including age, sex, race/ethnicity, smoking, body mass index, alcohol consumption, variables that represent socioeconomic status, and exposure to other known or suspected immunotoxicants, such as polychlorinated biphenyls (PCBs) and other potentially immunomodulatory perfluoroalkyl acids (PFASs). NTP acknowledges:

There may be limited ability to differentiate effects of PFOA or PFOS from other PFASs given that there is likely to be co-exposure with other PFASs and there may be similar immunomodulatory effects of the different PFASs (e.g., suppression of the antibody response as discussed above). Therefore, unless a study controlled for other PFASs, studies were rated probably high risk of bias in accounting for potential confounders and modifiers because of the limited ability to differentiate effects of PFOA or PFOS from other PFASs.

Of the five independent studies of PFOA or PFOS with respect to antibody response (Grandjean et al. 2012 (with Mogensen et al. 2015); Granum et al. 2013; Looker et al. 2014; Kielsen et al. 2016; Stein et al. 2016), only one (Mogensen et al. 2015) controlled for potential confounding by other PFASs. Mogensen et al. (2015) evaluated confounding by other PFASs in the associations between child PFOA or PFOS levels at age 5 years and antibody responses at age 7 years, and they found that associations with each PFAS were attenuated after mutual adjustment. However, they did not control for other PFASs when estimating associations with maternal PFOA or PFOS levels, or with antibody responses age at 5 years. The same study (Grandjean et al. 2012) was also the only one to control for PCBs (although results did not reveal an apparent confounding effect). Therefore, all of the studies should have been rated as being at probably high risk of bias due to confounding. In fact, NTP explicitly stated that Grandjean et al. (2012), Granum et al. (2013), and Kielsen et al. (2016) were rated as being at probably high risk of bias due to confounding by other PFASs. Stein et al. (2016) was initially

classified by NTP as a study with low confidence due to its cross-sectional design, and it was not further down-graded for risk of bias due to inadequate confounder control. NTP stated that it did not downgrade Looker et al. (2014) for being at high risk of bias due to potential confounding because of the high concentrations of PFOA in this population and the lack of an observed association with PFOS, with the implication that confounding by other PFASs was not a problem in this study population. However, this reasoning overlooks the facts that PFASs other than PFOS were not measured in this population, that low levels of other PFASs can just as readily confound associations with either low or high levels of PFOA, and that other potential immunotoxicants or their surrogates, such as PCBs, dioxins, and furans, were not measured and, therefore, not accounted for as potential confounders.

Moreover, among the key potential confounders identified by NTP, Grandjean et al. (2012) also did not report having considered maternal alcohol consumption, maternal body size, or socioeconomic status as potential confounders; Granum et al. (2013) apparently did not consider maternal alcohol consumption; Kielsen et al. (2016) apparently did not consider smoking, body size, alcohol consumption, or socioeconomic status (although this small study of 12 subjects had limited capacity for confounder control); and Stein et al. (2016) apparently did not consider alcohol consumption or socioeconomic status. None of the studies except Stein et al. (2016) controlled for race/ethnicity, but those other studies were set in populations that were unlikely to have substantial racial/ethnic heterogeneity.

Thus, any of the observed associations with PFOA or PFOS in the available epidemiologic studies of antibody response could be spurious findings due to confounding by other PFASs, other potential environmental immunotoxicants, or various uncontrolled sociodemographic or lifestyle factors. The high risk of bias due to confounding translates into low certainty that the results of these studies are valid. At a basic level, observed associations cannot confidently be attributed specifically to PFOA or PFOS. In combination with the inconsistency of results across studies, discussed above, the high potential for confounding should contribute to a rating of “low confidence,” rather than “moderate confidence” as rated by NTP, in the epidemiologic evidence for a suppressive effect of PFOA or PFOS in the antibody response.

5. Decrements in antibody-mediated immunity do not necessarily correspond with an increased risk of infectious disease.

None of the epidemiologic studies of antibody response demonstrated an increased risk of clinically recognizable infectious diseases as a consequence of a diminished antibody response. Other epidemiologic studies that explicitly examined associations of PFOA or PFOS exposure with infectious disease outcomes yielded inconsistent and mostly null results (Tables 13, 14, and 21 of the Draft Monograph; reviewed by Chang et al. 2016). Moreover, three of the five studies of antibody response examined adolescents or adults who largely had already been immunized against the antigens of interest prior to the immunization event under study (Looker et al. 2014; Kielsen et al. 2016; Stein et al. 2016), further obscuring the clinical significance of differences in antibody titers. Thus, the clinical and public-health interpretation of findings from the antibody response studies is unclear. Stein et al. (2016) acknowledged: “The clinical relevance of lower vaccine antibody concentrations at the levels we report – a 13% decrease for rubella with a doubling of PFOS concentration – is uncertain, particularly because this finding was among a subset of children who had antibody levels high enough to be considered protected against disease.”

NTP acknowledges: “It is unclear if this level of antibody reduction would affect the immune response to a viral or bacterial challenge for these individuals.” Nevertheless, NTP states that “any lowering of the antibody response may be considered adverse on a population level such that individuals with lower antibody levels *may be* less able to mount a defense against viruses or bacteria (WHO 2012)” (emphasis added). Such an adverse clinical effect, however, remains speculative. As expressed by a clinical immunologist, “if an abnormality is noted but it does not predict disease, then at best time and money are wasted, and at worst a patient is informed erroneously that he or she is sick or will get sick when this is not true, thereby breaking the rule of *primum non nocere* – above all do no harm” (Chang et al. 2016). Thus, whether a subclinical decrement in the antibody response, at the degree observed in these studies, represents a true “adverse health effect” remains debatable.

6. Conclusions

In summary, the hazard classification scheme used by NTP underplays the importance of a high risk of bias in invalidating scientific results, and it also underappreciates the potential impact of confounding on the reported association between PFOA or PFOS exposure and antibody response in humans. At the same time, NTP overvalues animal evidence in the presence of weak human evidence, and it exaggerates the consistency of results from the few available epidemiologic studies of antibody response. The net effect of these problems is the inappropriate classification of PFOA and PFOS as “presumed to be an immune hazard to humans.” If NTP more appropriately downgraded the level of confidence in studies with a high risk of bias, including those susceptible to uncontrolled confounding, and if NTP more accurately characterized the inconsistency of the epidemiologic evidence on antibody response and the weak epidemiologic evidence on hypersensitivity, then the proper hazard classification of PFOA and PFOS would arguably be “suspected to be an immune hazard to humans.”

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Appendix A

***Curriculum vitae* of Dr. Ellen
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Dr. Chang has 17 years of experience in designing, conducting, and interpreting epidemiologic studies, with a particular focus on studies of cancer and other chronic diseases. She provides scientific consultation on the potential human health effects of various chemicals (such as dioxins, chlorinated solvents, pesticides, PCBs, and perfluoroalkyl and polyfluoroalkyl substances), air pollutants, metals and metalloids, fibers, pharmaceuticals, medical devices, electromagnetic fields, and nutrients. She has expertise in qualitatively and quantitatively synthesizing the weight of epidemiologic evidence on causal effects of environmental exposures.

Dr. Chang's recent projects include evaluations of the epidemiologic evidence on glyphosate, TCDD, and perchloroethylene in association with non-Hodgkin lymphoma and other cancers; perfluoroalkyl and polyfluoroalkyl substances in association with immune-related conditions; fine particulate matter in association with all-cause mortality; and organophosphate insecticides in association with birth and developmental outcomes. Dr. Chang also frequently conducts and coordinates analyses of cancer incidence, mortality, and survival in population-based cancer registries.

Dr. Chang has led original research studies of cancers of the head and neck, nasopharynx, stomach, liver, lung and bronchus, skin, breast, uterus, ovary, prostate, thyroid, and lymphatic system. These studies focused on a wide range of exposures including genetic variation, physical activity, body size, diet and nutrition, alcohol consumption, tobacco smoking, ultraviolet radiation, immunologic biomarkers, microbial infections, use of nonsteroidal anti-inflammatory drugs and other medications, use of hormone therapy and oral contraceptives, reproductive factors, medical history, family structure, and demographic characteristics. In addition, Dr. Chang has conducted cancer surveillance research at one of the U.S. National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) registries, and contributed to community-based research on hepatitis B and liver cancer awareness, detection, prevention, and medical management at the Asian Liver Center at Stanford

University.

Dr. Chang earned her undergraduate degree in English and American literature and language from Harvard College. She earned her Sc.D. (Doctor of Science) in epidemiology with a minor in biostatistics from the Harvard School of Public Health, and she completed a post-doctoral fellowship at the Karolinska Institute. She is a member of the Stanford Cancer Institute and a Consulting Assistant Professor in the Division of Epidemiology, Department of Health Research and Policy at the Stanford University School of Medicine. Dr. Chang has published more than 150 peer-reviewed research articles and reviews, and eight book chapters.

CREDENTIALS & PROFESSIONAL HONORS

Sc.D., Epidemiology, Harvard University, 2003

A.B., English and American Literature and Language, Harvard University, 1998

National Cancer Institute Minority Investigators Workshop on Behavioral Methodologies Fellowship, 2007

New York Academy of Sciences (NYAS) Science Alliance Program Membership, 2005–2006

National Institutes of Health Ruth L. Kirschstein National Research Service Award, 2004–2005

American Association for the Advancement of Science (AAAS)/Science Program for Excellence in Science Membership, 2004–2005

Harvard University Sheldon Traveling Fellowship, 2003–2004

Harvard School of Public Health Department of Epidemiology Seiden Scholarship, 2001–2003

Harvard University Pforzheimer Public Service Fellowship, 1999–2003

National Cancer Institute/Harvard School of Public Health Cancer Epidemiology Pre-Doctoral Training Program Fellowship, 1998–2002

LANGUAGES

PUBLICATIONS

Peer-Reviewed Publications

Chang ET, Liu Z, Hildesheim A, Liu Q, Cai Y, Zhang Z, Chen G, Xie SH, Cao SM, Shao JY, Jia WH, Zheng Y, Liao J, Chen Y, Lin L, Ernberg I, Vaughan TL, Adami HO, Huang G, Zeng Y, Zeng YX, Ye W. Active and passive smoking and risk of nasopharyngeal carcinoma: a population-based case-control study in southern China. *Am J Epidemiol* (in press).

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Chang ET, So SK. Chapter 9. Chronic hepatitis B and liver cancer: The greatest health disparity between Asian and non-Asian Americans. In: Praeger Handbook of Asian American Health: Taking Notice and Taking Action. Bateman WB, Abesamis-Mendoza N, Ho-Asjoe H (eds), Santa Barbara, CA: ABC-CLIO, 2009: 177–199.

Wakelee HA, Gomez SL, Chang ET. Sex differences in lung cancer susceptibility: A smoke screen? *Lancet Oncol* 2008; 9(7): 609–610.

Chang ET, So SK. Re: Ten largest racial and ethnic health disparities in the United States Based on Healthy People 2010 Objectives by Keppel. *American Journal of Epidemiology* 2007; 166(9): 1105–1106.

Gomez SL, Clarke CA, Chang ET, Keegan THM, West DW, So SK, Glaser SL. Response to McCracken et al.: Cancer Surveillance Among Asian Americans, CA: *Cancer J Clin* 2007 Aug 28 (eLetter).

Chang ET, Adami HO. Chapter 8. Nasopharyngeal carcinoma. In: *Textbook of Cancer Epidemiology*, 2nd Edition. Adami HO, Hunter D, Trichopoulos D (eds), New York: Oxford University Press, 2008: 175–195.

Mueller NE, Grufferman S, Chang ET. Chapter 2. The epidemiology of Hodgkin lymphoma. In: *Hodgkin Lymphoma*, 2nd Edition. Hoppe RT, Armitage JO, Diehl V, Mauch PM, Weiss LM (eds), Philadelphia, PA: Lippincott Williams & Wilkins, 2007: 7–23.

Chang ET, Clarke CA, Glaser SL. Making sense of seasonal fluctuations in lymphoma diagnosis. *Leuk Lymphoma* 2007; 48(2): 223–224.

Chang ET, Hedelin M, Adami HO, Grönberg H, Bälter KA. Re: Zinc supplement use and risk of prostate cancer. *Journal of the National Cancer Institute* 2004; 96(14): 1108.

Birmann B, Chang ET, Mueller NE. Chapter 2. Epidemiology of lymphoma. In: *Malignant Lymphomas*. Grossbard ML (ed), Hamilton, Ontario: BC Decker, Inc., 2002: 31–46.

Abstracts, Posters, and Presentations

Chen VL, Podlaha O, Estevez J, Li B, Le A, Vutien P, Chang ET, Pflanz S, Jiang Z, Ge D, Gaggar A, Nguyen MH. High serum soluble intracellular adhesion molecule 1 (sICAM-1) concentration is associated with hepatocellular carcinoma development in hepatitis B virus, hepatitis C virus, and non-viral liver disease: Multiplex analysis of 51 cytokines and other serum markers. Abstract and presentation at Digestive Disease Week 2016, San Diego, California, May 21–24, 2016.

Estevez J, Chen VL, Podlaha O, Li B, Le A, Vutien P, Chang ET, Jiang Z, Pflanz S, Ge D, Gaggar A, Nguyen MH. Differential cytokine profiles in patients with hepatocellular carcinoma related to hepatitis B virus (HBV) and hepatitis C virus (HCV) infection. Abstract and presentation at Digestive Disease Week 2016, San Diego, California, May 21–24, 2016.

Chang ET. Weight-of-evidence synthesis in epidemiology: What's the bottom line? Presentation at DRI Toxic Torts and Environmental Law Seminar, New Orleans, Louisiana, March 17–18, 2016.

Cheng VL, Vutien P, Li B, Podlaha O, Chang ET, Jiang Z, Ge D, Gaggar A, Nguyen MH. Differential serum cytokine profiles in patients with hepatitis B virus (HBV), hepatitis C virus (HCV), and non-viral non-autoimmune liver disease, with or without hepatocellular carcinoma (HCC). Abstract and poster at The AASLD Liver Meeting 2015, San Francisco, California, November 13–17, 2015.

Plenary speaker. NPC international incidence and risk factors. 7th International Biannual Symposium on Nasopharyngeal Carcinoma 2015, Yogyakarta, Indonesia, June 3–6, 2015.

Session chair for plenary session on genetics and epigenetics of nasopharyngeal carcinoma. 7th International Biannual Symposium on Nasopharyngeal Carcinoma 2015, Yogyakarta, Indonesia, June 3–6, 2015.

Invited speaker. Reanalysis of the Diesel Exhaust in Miners Study (DEMS) cohort. Mario Negri Institute for Pharmacological Research, Milan, Italy, March 12, 2015.

Epstein MM, Chang ET, Zhang Y, Fung T, Batista JL, Ambinder RF, Zheng T, Mueller NE, Birmann BM. Diet patterns and risk of Hodgkin lymphoma in a population-based case-control study. Abstract and presentation at 2014 InterLymph Annual Meeting, Los Angeles, CA, June 17–20, 2014.

Hjalgrim H, Monnereau A, Glaser SL, Chang E. Risk factors for classical Hodgkin lymphoma. Presentation at 2013 InterLymph Annual Meeting, Dijon, France, June 24–26, 2013.

Birmann BM, Epstein MM, Chang ET, Zhang Y, Fung T, Kasperzyk J, Ambinder RF, Zheng T, Mueller NE. Dietary patterns and risk of Hodgkin lymphoma in a population-based case-control study [Abstract 491-S]. *American Journal of Epidemiology* 2013; 177(11Suppl): S123. Poster at 46th Annual Society for Epidemiologic Research (SER) Meeting, Boston, MA, June 18–21, 2013.

Gao L, Chang E, Nelson D, Vutien P, Rosenberg-Hassan Y, Nguyen MH. Serum cytokine profiles and hepatocellular carcinoma (HCC) in patients with chronic hepatitis C. Poster at Digestive Disease Week, Orlando, FL, May 18–21, 2013.

Vutien P, Chang E, Nelson D, Gao L, Rosenberg-Hassan Y, Nguyen MH. Serum cytokine profiles in patients with hepatitis B virus (HBV) infection and associated hepatocellular carcinoma (HCC). Poster at Digestive Disease Week, Orlando, FL, May 18–21, 2013.

Monnereau A, Glaser SL, Chang ET. Ultraviolet radiation (UVR) and risk of Hodgkin lymphoma: A pooled analysis. Presentation at InterLymph Consortium Annual Meeting, Washington, DC, June 7, 2012.

Invited speaker. Gastric cancer incidence patterns in California Hispanics. Northern California Cancer Registrars Association Conference, Fremont, CA, December 14, 2011.

Ai W, Chang E, Fu K, Fish K, Weisenburger DD, Keegan T. Racial/ethnic patterns of NK/T cell lymphoma in California: A population-based study. Abstract at International Conference on Malignant Lymphoma, Lugano, Switzerland, June 15–18, 2011.

Colevas AD, Clarke CA, Lichtensztajn D, Chang ET. A population-based evaluation of incidence trends in oropharynx cancer focusing on socioeconomic status, sex, and race/ethnicity. Poster at American Society of Clinical Oncology (ASCO) Annual Meeting, Chicago, IL, June 4–8, 2010.

Clément-Duchêne C, Xu X, Gomez SL, Chang ET, West DW, Wakelee HA, Gould MK. Survival among never and ever smokers with lung cancer in the Cancer Care Outcomes Research and Surveillance Consortium (CanCORS) study. Poster at American Society of Clinical Oncology (ASCO) Annual Meeting, Chicago, IL, June 4–8, 2010.

Chang ET. Ultraviolet radiation exposure & sensitivity and risk of Hodgkin lymphoma: A pooled analysis. Presentation at InterLymph Consortium 9th Annual Meeting, Washington, DC, April 15, 2010.

Chang ET, Nguyen BH, So SK. Motivations for hepatitis B and liver cancer prevention in Bay Area Chinese Americans. Poster at Stanford Cancer Center Members' Retreat, Menlo Park, CA, April 7, 2010.

Wakelee HA, Chang ET, Shema SJ, Reynolds P, Clément-Duchêne C, Wiencke J, Gomez SL. Survival after non-small cell lung cancer in never-smoking Asian/Pacific Islander and Latina women. *J Thoracic Oncol* 2009; 4(9 Suppl 1): s310 (Abstr#A7.6). Oral presentation at 13th World Conference on Lung Cancer, San Francisco, CA, July 31–August 4, 2009.

Chang ET, Kasperzyk JL, Birmann BM, Kraft P, Zheng T, Mueller NE. One-carbon metabolism nutrients and genes and Hodgkin lymphoma risk. Oral presentation at InterLymph Consortium 8th Annual Meeting, Vancouver, British Columbia, July 19–22, 2009.

Marshall SF, Chang ET, Clarke CA, Cress R, Deapen D, Horn-Ross PL, Largent J, Neuhausen S, Reynolds P, Templeman C, Bernstein L. Hormone therapy before diagnosis and breast cancer survival in the 10 California Teachers Study. Abstract at San Antonio Breast Cancer Symposium, San Antonio, TX, December 10–14, 2008.

Telli ML, Kurian AW, Chang ET, Keegan THM, McClure LA, Ford JM, Gomez SL. Differences in breast cancer subtype distribution exist among ethnic subgroups of Asian women in California. Abstract at San Antonio Breast Cancer Symposium, San Antonio, TX, December 10–14, 2008.

Telli ML, Kurian AW, Chang ET, Keegan THM, Ford JM, Gomez SL. Asian race and breast cancer subtypes: a study from the California Cancer Registry. Poster at 44th American Society for Clinical Oncology (ASCO) Annual Meeting, Chicago, IL, May 30–June 3, 2008.

Invited speaker. Hepatitis B and liver cancer prevention in Asian/Pacific Islander Americans. Department of Epidemiology, Harvard School of Public Health, Boston, MA, April 1, 2008.

Chen JJ, Bergin M, Chang ET, So SK. A model HBV catch-up immunization and education project in Qinghai, China. Workshop presentation at 42nd National Immunization Conference, Atlanta, GA, March 17–20, 2008.

Bergin M, Rao A, Chang ET, So SK. Motivating youth to take action in public health: 5th Annual Youth Leadership Conference on Asian and Pacific Islander Health. Poster at 42nd National Immunization Conference, Atlanta, GA, March 17–20, 2008.

Invited speaker. Integration of population sciences with clinical research. Cancer Clinical Trials Forum, Stanford University School of Medicine, Stanford, California, July 18, 2007.

Chang ET, Nguyen BH, So SK. Determinants of hepatitis B awareness and prevention in Chinese Americans. Poster and presentation at Stanford Cancer Center retreat, Menlo Park, CA, March 12, 2007.

Chang ET, Lin SY, So SK. The Jade Ribbon Campaign: Hepatitis B virus screening and education in Asian/Pacific Islander Americans. Presentation at 2006 National Asian American Pacific Islander Health Summit, San Jose, CA, September 15, 2006.

Chang ET, Canchola AJ, Lee VS, Clarke CA, Reynolds P, Horn-Ross PL, and the California Teachers Study Investigators. Wine and other alcohol consumption and risk of ovarian cancer in the California Teachers Study cohort. Abstract and poster at 2nd North American Congress of Epidemiology, Seattle, WA, June 21–24, 2006.

Invited speaker. The role of the Epstein-Barr virus in Hodgkin lymphoma. Viruses and Cancer Symposium, Harvard School of Public Health, Boston, MA, May 5, 2006.

Invited speaker. The changing racial/ethnic burden of liver cancer in the Greater San Francisco Bay Area. Greater Bay Area Cancer Registry Certified Tumor Registrars Meeting, Fremont, CA, April 5, 2006.

Invited speaker. The role of the Epstein-Barr virus in Hodgkin lymphoma. Department of Epidemiology, University of Michigan School of Public Health, Ann Arbor, MI, January 12, 2005.

Invited speaker. Department of Epidemiology, University of Washington School of Public Health and Community Medicine, and Seattle Epidemiologic Research and Information Center, Seattle, WA, August 17, 2004.

Levin LI, Lennette ET, Ambinder RF, Chang ET, Rubertone M, Mueller NE. Prediagnosis Epstein-Barr virus serologic patterns in relation to the molecular status of Hodgkin's lymphoma in young adults. Abstract and poster at American Association for Cancer Research International Conference on Molecular and Genetic Epidemiology of Cancer, Waikoloa, HI, January 18–23, 2003.

Chang ET, Ambinder RF, Weir EG, Borowitz M, Mann RB, Zheng T, Mueller NE. Inverse association between nursery school and Hodgkin's lymphoma, independent of EBV tumor status. Abstract and poster at 10th Biennial Meeting of the International Association for Research on Epstein-Barr Virus and Associated Diseases, Cairns, Australia, July 16–21, 2002.

Levin LI, Lennette ET, Ambinder RF, Chang ET, Rubertone M, Mueller NE. Prediagnosis Epstein-Barr virus serologic patterns in EBV-positive and EBV-negative Hodgkin's lymphoma. Abstract and poster at 10th Biennial Meeting of the International Association for Research on Epstein-Barr Virus and Associated Diseases, Cairns, Australia, July 16–21, 2002.

Chang ET, Ambinder RF, Zheng T, Mueller NE. Inverse association between childhood history of nursery school and Hodgkin's lymphoma in a population-based case-control study. *Leuk Lymphoma* 2001; 42(Suppl 2): 39-40. Abstract and poster at 5th International Symposium on Hodgkin's Lymphoma, Cologne, Germany, September 22–25, 2001.

Chang ET, Ambinder RF, Zheng T, Mueller NE. Inverse association between aspirin use and Hodgkin's lymphoma in a population-based case-control study. *Leuk Lymphoma* 2001; 42(Suppl 2): 41. Abstract and poster at 5th International Symposium on Hodgkin's Lymphoma, Cologne, Germany, September 22–25, 2001.

Chang ET, Birmann B, Ambinder RF, Zheng T, Mueller NE. Serum sCD23 levels in Hodgkin's disease patients are higher in EBV genome-positive than EBV genome-negative cases. Abstract, poster, and presentation at 9th Biennial Meeting of the International Association for Research on Epstein-Barr Virus and Associated Diseases, New Haven, Connecticut, June 22–27, 2000.

PRIOR EXPERIENCE

Research Scientist, Cancer Prevention Institute of California, 2005–2012

Consulting Assistant Investigator, Department of Health Policy Research, Palo Alto Medical Foundation Research Institute, 2008–2012

Chief Epidemiologist, Asian Liver Center at Stanford University, 2006–2011

PROFESSIONAL AFFILIATIONS

American Association for Cancer Research

Society for Epidemiologic Research

ACADEMIC APPOINTMENTS

Consulting Assistant Professor, Division of Epidemiology, Department of Health Research and Policy, Stanford University School of Medicine, 2007–present

Consulting Assistant Professor, Division of Epidemiology, Department of Health Research and Policy, Stanford University School of Medicine, 2007–present

Member, Stanford Cancer Institute, 2005–present

