

Exhibit 111

General Causation Expert Report of Howard Hu, M.D., M.P.H., Sc.D.

Prepared by:

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I am writing in response to your request for a medical expert opinion on (a) whether there is a causal relationship between exposure to trichloroethylene (TCE), tetrachloroethylene (PCE), and/or benzene in the water at Camp Lejeune and Non-Hodgkin Lymphoma (NHL); (b) where such a causal relationship mentioned in (a) exists, what the likely dose-response relationship at least as likely as not would be at certain levels of exposure; and (c) the potential impact of combined exposure to trichloroethylene (TCE), tetrachloroethylene (PCE), and benzene.

Background & Qualifications

I am a physician-scientist, board-certified internist and board-certified preventive medicine (occupational medicine) specialist, with a doctoral degree in epidemiology. As my Curriculum Vitae reflects, my current academic appointments are Professor of Population and Public Health Sciences (tenured) of the Department of Population and Public Health Sciences, Keck School of Medicine of the University of Southern California (USC), and Adjunct Professor in the University of Michigan School of Public Health. I am currently on a 1-year sabbatical from USC (July 1, 2024 – June 30, 2025) after having served as the Flora L. Thornton Chair of the Department since July of 2020. Previously, I had been the Founding Dean of the Dalla Lana School of Public Health and Professor of Environmental Health, Epidemiology, Global Health, and Medicine (tenured), University of Toronto (2012–2018); the NSF International Endowed Chair of the Department of Environmental Health Sciences, Professor of Environmental Health, Epidemiology and Medicine (tenured), Founding Director of the NIH/NIEHS Environmental Health Core Sciences Center, and Associate Physician at the University of Michigan and University of Michigan Health System (2006–2012); and Professor of Occupational & Environmental Medicine (tenured), Founding Director of the NIH/NIEHS Center for Children's Environmental Health, Director of the Occupational Medicine Residency at the Harvard School of Public Health and Associate Physician in the Brigham & Women's Hospital in Boston (1988–2006).

In terms of specific scientific expertise, since 1990, I have led multi-institutional and international teams of scientists, students, and fellows devoted to investigating the environmental, nutritional, social, psychosocial, genetic, and epigenetic determinants of chronic disease and impaired child development in population-based studies in the United States, Mexico, India (where I was a senior faculty Fulbright Scholar, 2000–2001), China, and elsewhere around the world. Our research team's work has generated over 350 publications in the peer-reviewed literature and won several awards, such as the 1999 Progress and Achievement Award from the U.S. NIH/NIEHS, the 2009 Linus Pauling Lifetime Achievement Award, the 2011 Award of Excellence from the American Public Health Association, and the 2015 John Goldsmith Award for Outstanding Contributions from the International Society for Environmental Epidemiology. In my current position, I am continuing NIH-funded environmental birth cohort research while also co-leading the Global Burden of Disease-Pollution, Climate, and Health initiative, which aims to improve understanding of pollution's "footprint" on the global burden of disease. I also lead several epidemiological studies related to the COVID-19 pandemic. In terms of service that capitalizes on my expertise, I served on, among other entities, the Board of Population and Public Health Practice of the Institute of Medicine of the National Academy of Sciences; on the Board of Environmental Studies and

Toxicology of the National Research Council; on the External Advisory Council of the U.S. National Institute for Environmental Health Sciences; and on the Energy Research Committee of the Health Effects Institute. I am currently serving as the Chair of the Scientific Advisory Board of the Marilyn Brachman Hoffman Foundation; on the Board of Directors of Pure Earth; on the Board of Directors of Wellness Equity Alliance; and as the Co-Chair of the Research Council of the Public Health Foundation of India.

In terms of expertise and service specific to environmental exposures and cancer, I trained specifically in toxicology, environmental health, environmental epidemiology, and cancer epidemiology as part of my Masters in Public Health coursework in occupational health and Doctorate in Science coursework in epidemiology, all at the Harvard School of Public Health. I collaborated on environmental and molecular epidemiologic studies of cancer as they relate, for example, to environmental risk factors for bladder cancer,^{2,3} lung cancer,⁴ prostate cancer,⁵ hepatic steatosis,⁶ and non-alcoholic fatty liver disease⁷ (known risk factors for liver cancer), and genetic susceptibility factors related to B-cell lymphomas.⁸ I served on the Advisory Board for the Cancer Epidemiology Education in Special Populations Program at the University of Michigan School of Public Health, have lectured on translational research opportunities and challenges for cancer research, and served as the Principal Investigator of an investigation of

² Guo HR, Chiang HS, Hu H, Lipsitz SR, Monson RR. Arsenic in drinking water and incidence of urinary cancers. *Epidemiology*. 1997 Sep;8(5):545-50. PubMed PMID:9270957.

³ Hu H, Markowitz SB. A case-study of industrial bladder cancer. *Einstein Quarterly Review of Biology and Medicine* 1982;1:29-35.

⁴ Guo HR, Wang NS, Hu H, Monson RR. Cell type specificity of lung cancer associated with arsenic ingestion. *Cancer Epidemiol Biomarkers Prev*. 2004 Apr;13(4):638-43. PubMed PMID: 15066930.

⁵ Tse LA, Lee PMY, Ho WM, Lam AT, Lee MK, Ng SSM, He Y, Leung KS, Hartle JC, Hu H, Kan H, Wang F, Ng CF. Bisphenol A and other environmental risk factors for prostate cancer in Hong Kong. *Environ Int*. 2017 Oct;107:1-7. doi: 10.1016/j.envint.2017.06.012. Epub 2017 Jun 20. PubMed PMID: 28644961.

⁶ Betanzos-Robledo L, Cantoral A, Peterson KE, Hu H, Hernández-Ávila M, Perng W, Jansen E, Ettinger AS, Mercado-García A, Solano-González M, Sánchez B, Téllez-Rojo MM. Association between cumulative childhood blood lead exposure and hepatic steatosis in young Mexican adults. *Environ Res*. 2021 May;196:110980. doi: 10.1016/j.envres.2021.110980. Epub 2021 Mar 7. PMID: 33691159; PMCID: PMC8119339.

⁷ Stratakis N, Golden-Mason L, Margetaki K, Zhao Y, Valvi D, Garcia E, Maitre L, Andrusaityte S, Basagana X, Borràs E, Bustamante M, Casas M, Fossati S, Grazuleviciene R, Haug LS, Heude B, McEachan RRC, Meltzer HM, Papadopoulou E, Roumeliotaki T, Robinson O, Sabidó E, Urquiza J, Vafeiadi M, Varo N, Wright J, Vos MB, Hu H, Vrijheid M, Berhane KT, Conti DV, McConnell R, Rosen HR, Chatzi L. In Utero Exposure to Mercury Is Associated With Increased Susceptibility to Liver Injury and Inflammation in Childhood. *Hepatology*. 2021 Sep;74(3):1546-1559. doi: 10.1002/hep.31809. Epub 2021 Aug 30. PMID: 33730435; PMCID: PMC8446089.

⁸ Bashash M, Connors JM, Gascoyne RD, Meissner B, Schuetz JM, Leach S, Slack GW, Berry R, Hu H, Sehn LH, Brooks-Wilson AR, Spinelli JJ. Genetic polymorphism at BCL2 as a predictor for rituximab, cyclophosphamide, doxorubicin, vincristine and prednisone efficacy in patients with diffuse large B-cell lymphoma. *Haematologica*. 2017 May;102(5):e199-e202. doi: 10.3324/haematol.2016.159087. Epub 2017 Feb 2. PubMed PMID: 28154089; PubMed Central PMCID: PMC5477624.

cancer and the environment funded by Health Canada.⁹

I am also familiar with the process of systematic review as it pertains to assessing the scientific literature and coming to conclusions regarding causality, having collaborated on 3 iterations of the systematic review of behavioral, environmental, occupational, and metabolic risk factors as part of the Global Burden of Disease Project (e.g., GBD, 2015¹⁰), a systematic review of the potential health impacts of unconventional oil and development,¹¹ a systematic review of lead exposure in low- and middle-income countries,¹² and an on-going systematic review of lead exposure and anti-social behavior.¹³ Several of these have included dose-response modeling, an exercise in which I have also engaged with collaborators on studies of the relationship between prenatal fluoride exposure and offspring cognitive outcomes.^{14,15}

Methodology

To conduct this expert evaluation, I reviewed the relevant peer-reviewed scientific literature and reports of international and national agencies. I evaluated each relevant study and report by looking at several factors, including statistical significance, potential for bias, and the quality of methodology and study design, among other criteria described throughout this report where relevant. Where studies provide incomplete, contradictory, or outlier results, I determined the appropriate relevance to be given to a particular study, based on my years of training and experience, and using criteria discussed below and throughout this report where relevant. Where

⁹ 5/15/2015-5/15/2019. Health Canada; PI, “A Community-based First Nation Study of Cancer and the Environment in Northern Ontario”.

¹⁰ GBD 2013 Risk Factors Collaborators, Forouzanfar MH, Alexander L, Anderson HR...Hu H...Lopez AD, Vos T, Murray CJ. Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet*. 2015 Dec 5;386(10010):2287-323. doi: 10.1016/S0140-6736(15)00128-2. Epub 2015 Sep 11. PubMed PMID: 26364544; PubMed Central PMCID: PMC4685753.

¹¹ HEI Energy Research Committee (Hu: Member). Potential Human Health Effects Associated with Unconventional Oil and Gas Development: A Systematic Review of the Epidemiology Literature. Health Effects Institute, September 2019. Available at: <https://hei-energy.org/publication/potential-human-health-effects-associated-unconventional-oil-and-gas-development>

¹² Ericson B, Hu H, Nash E, Ferraro G, Sinitsky J, Taylor MP. Blood lead levels in low-income and middle-income countries: a systematic review. *Lancet Planet Health*. 2021 Mar;5(3):e145-e153. doi: 10.1016/S2542-5196(20)30278-3. PMID: 33713615.

¹³ Shaffer RM, Forsyth JE, Ferraro G, Till C, Carlson LM, Hester K, Haddock A, Strawbridge J, Lanfear CC, Hu H, Kirrane E. Lead exposure and antisocial behavior: A systematic review protocol. *Environ Int*. 2022 Aug 4;168:107438. doi: 10.1016/j.envint.2022.107438. Epub ahead of print. PMID: 35994796.

¹⁴ Grandjean P, Hu H, Till C, Green R, Bashash M, Flora D, Tellez-Rojo MM, Song P, Lanphear B, Budtz-Jørgensen E. A Benchmark Dose Analysis for Maternal Pregnancy Urine-Fluoride and IQ in Children. *Risk Anal*. 2021 Jun 8. doi: 10.1111/risa.13767. Epub ahead of print. PMID: 34101876.

¹⁵ Grandjean P, Meddis A, Nielsen F, Beck IH, Bilenberg N, Goodman CV, Hu H, Till C, Budtz-Jørgensen E. Dose dependence of prenatal fluoride exposure associations with cognitive performance at school age in three prospective studies. *Eur J Public Health*. 2023 Oct 5:ckad170. doi: 10.1093/eurpub/ckad170. Epub ahead of print. PMID: 37798092.

national and international agency reports rely on the “weight-of-the-evidence” methodology, I evaluated their application of established medical and scientific methodology, and their ultimate conclusions, based on my years of training and experience. I then identified and relied upon the studies and reports that, in my opinion, comprise the most rigorous and authoritative documents relevant to the issues inherent in this evaluation. Such evidence will be cited in this report as appropriate.

Based on this review and critical evaluation of the relevant literature, I produced this report analyzing the causal relationship between TCE, PCE, benzene, and Non-Hodgkin Lymphoma (NHL) along six dimensions: (1) the association between TCE and cancer, generally; (2) the association between TCE and NHL; (3) the association between PCE and cancer, generally; (4) the association between PCE and NHL; (5) the association between benzene and cancer, generally; and (6) the association between benzene and NHL. I also addressed the issue of dose-response relationships at low levels of exposure and considered the association of exposure to TCE, PCE, and benzene (i.e., in combination) with NHL. My review and critical evaluation of the relevant scientific literature in this report follows the same methodologies as I would employ in my own research and practice.

My expert opinions regarding the causal relationships between TCE, PCE, benzene, and NHL are based on my years of training, research, and experience, applying established medical and scientific methodologies, including the “Bradford Hill” considerations. These are a well-established set of considerations used in evaluating the strength between a risk factor and health outcome in order to make judgements about causality. As Sir Bradford Hill himself stated, each of the considerations are not required items on a checklist but are rather factors to be considered as part of a causation analysis. Throughout this report, I identify the considerations that weigh most heavily in my analysis of a given study or report and in my ultimate conclusions regarding causality.

The general practice among researchers and physicians in my field, which I apply in this report, is to consider all the relevant evidence in light of the factors and relevant context regarding study design and associated limitations and sources of bias. This almost always requires considering evidence that is probabilistic and susceptible to random error because such is the nature of scientific research. Nevertheless, it is common and appropriate to make determinations about potential causal relationships using deduction and/or extrapolation and to state the degree of certainty attached to those conclusions. In this report, the following represents my opinions based on a reasonable degree of medical and scientific certainty.

Standard Applied

It is my understanding that the Camp Lejeune Justice Act of 2022, the controlling statute in this case, states that a plaintiff must “show one or more relationships between the water at Camp Lejeune and the harm.” “To meet that burden of proof . . . a party shall produce evidence showing that the relationship between exposure to the water at Camp Lejeune and the harm is— (A) sufficient to conclude that a causal relationship exists; or (B) sufficient to conclude that a causal relationship is at least as likely as not.” It is also my understanding that the water at Camp

Lejeune was contaminated with, among other potential chemicals, benzene, TCE, PCE, and vinyl chloride.

The Agency for Toxic Substances and Disease Registry (ATSDR) released on January 13, 2017 a report titled *ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases*. I reviewed the ATSDR Report in forming my opinions in this case. The ATSDR Report used four categories to classify the strength of the evidence for a causal relationship between the chemicals in the water at Camp Lejeune and various harms. The “Sufficient” and “Equipoise and Above” categories of this classification scheme employ the same language as the Camp Lejeune Justice Act: “1. Sufficient: The evidence is sufficient to conclude that a causal relationship exists. 2. Equipoise and Above: The evidence is sufficient to conclude that a causal relationship is at least as likely as not, but not sufficient to conclude that a causal relationship exists.” The ATSDR then proceeded to explain how, in its view, each of these categories could be met.

I express some of my opinions in this report under a “more likely than not” standard. Based on my review of the Camp Lejeune Justice Act’s “at least as likely as not standard,” the ATSDR’s “equipoise and above” classification, and my own research and knowledge of “equipoise” in scientific literature, the opinions I express under the “more likely than not” standard *necessarily exceed* the “at least as likely as not” standard under the Camp Lejeune Justice Act.

Summary of Opinions

In summary, from my review of the literature and additional scientific reports related to trichloroethylene (TCE), tetrachloroethylene (PCE), benzene, and Non-Hodgkin Lymphoma (NHL), it is my opinion that (1) TCE is a human carcinogen; (2) TCE more likely than not can cause NHL; (3) PCE is a human carcinogen; (4) PCE at least as likely as not, if not more likely than not, can cause NHL; (5) benzene is a human carcinogen; (6) benzene more likely than not can cause NHL. Furthermore, it is my opinion that (7) the quantitative risk associated with being exposed to the combination of TCE, PCE, and benzene with respect to NHL is at least as likely as not additive or higher.

Discussion of Opinions

I. TCE, Cancer, and NHL

My opinion that TCE is a human carcinogen and a carcinogen specifically for NHL is based on (1) review of and agreement with the 2014 monograph published by the International Agency for Research on Cancer (IARC), as well as my review of its underlying sources/studies, on its assessment of TCE’s carcinogenicity, as well as review of IARC’s assessment of TCE as a risk factor specifically for NHL; (2) review of and agreement with the 2015 monograph published by the U.S. National Toxicology Program (NTP) on its assessment of TCE’s carcinogenicity, as well as my review of its underlying source/studies, as well as review of the NTP’s assessment of TCE as a risk factor specifically for NHL; (3) my assessment of the scientific literature that has appeared since the 2014 IARC monograph and 2015 NTP

monograph, which provided additional evidence on TCE as a risk factor specifically for NHL, including both toxicological and epidemiological evidence; and (4) consideration of the evidence in relation to the Bradford Hill considerations.

A. Background

1. Trichloroethylene (TCE)

TCE is a colorless, volatile liquid. Liquid TCE evaporates quickly into the air. It is non-flammable and has a sweet odor. The two major uses of TCE are as a solvent to remove grease from metal parts and as a chemical that is used to make other chemicals, especially the refrigerant HFC-134a. TCE has also been used as an extraction solvent for greases, oils, fats, waxes, and tars; by the textile processing industry to scour cotton, wool, and other fabrics; in dry cleaning operations; and as a component of adhesives, lubricants, paints, varnishes, paint strippers, pesticides, and cold metal cleaners.

Human exposure to TCE can occur through TCE-contaminated air, water, food, or soil, or through direct skin contact. A relatively common exposure route to TCE is via drinking TCE-contaminated water, as well as the inhalation of TCE released into the air from TCE-contaminated water (e.g., when showering). One can also be exposed to TCE through dermal contact, such as swimming in a pool, showering, or washing hands with TCE-contaminated water. Occupational exposures may occur in the degreasing industry or other industries where TCE is produced or used via breathing in TCE-contaminated air or by contact of the chemical with skin. Some TCE is released into the air by its evaporation from products such as adhesives, paints, and coatings; and through its evaporation from TCE-contaminated soil at landfills. TCE exposure can also occur through the consumption of TCE-contaminated foods, by contact with consumer products containing TCE, and by direct contact with TCE-contaminated soil.

I note that on October 23, 2023, the EPA proposed a ban on TCE based on the risks it poses for causing cancer, neurotoxicity, and reproductive toxicity¹⁶.

2. Non-Hodgkin Lymphoma (NHL)

Non-Hodgkin Lymphoma (NHL) is a type of cancer that starts in lymphocyte cells, which are part of the body's immune system. There are many different types of NHL, and they are generally divided into those that involve B-lymphocyte cells (which produce antibodies, that, in turn, attach to germs like bacteria, viruses, or other infectious agents, marking them for destruction) or T-lymphocyte cells (which destroy germs or abnormal cells in the body, including cancerous cells). NHL can start anywhere in the body's lymphatic system, including lymph nodes, the spleen, bone marrow, the thymus, adenoids and tonsils, or lymph tissue in the digestive tract. Some forms of NHL are indolent (slow-growing); others are aggressive, and, if untreated, can quickly spread to other parts of the lymphatic system and other organs, such as the

¹⁶ US EPA. Biden-Harris Administration Proposes Ban on Trichloroethylene to Protect Public from Toxic Chemical Known to Cause Serious Health Risks. Available at: doi:10.1001/jamanetworkopen.2024.11987; accessed November 14, 2024.

liver or brain.

B-cell Non-Hodgkin Lymphomas are the most common¹⁷ and include several subtypes:

Diffuse Large B-Cell Lymphoma (DLBCL) is the most prevalent subtype. DLBCL can occur at any age but is most commonly diagnosed in older adults, with the average age at diagnosis being in the mid-60s. It typically presents as a rapidly enlarging mass in a lymph node, either deep within the body, such as in the chest or abdomen, or in a more accessible area like the neck or armpit. In some cases, it may originate in other parts of the body, including the intestines, bones, brain, or spinal cord. Although DLBCL is considered an aggressive, fast-growing lymphoma, it often responds effectively to treatment.¹⁸

Mantle Cell Lymphoma (MCL) is often advanced and widespread at diagnosis, commonly involving the lymph nodes, bone marrow, and spleen. While it is considered a fast-growing lymphoma, it tends to grow more slowly than other aggressive lymphomas and typically does not respond as well to treatment.¹⁹ Managing MCL can be particularly challenging, though a stem cell transplant may be a treatment option in certain cases.²⁰

Marginal Zone Lymphoma (MZL) account for 5% to 10% of lymphomas and are typically slow-growing (indolent).²¹ While patients with MZL often have long survival times, the disease is usually incurable, with relapses occurring over many years. A small percentage of MZLs may transform into diffuse large B-cell lymphoma (DLBCL), which requires more aggressive treatment.²² Subtypes of MZL include nodal, gastric mucosa-associated lymphoid tissue (MALT), extragastric MALT, Mediterranean abdominal lymphoma, and splenic marginal zone

¹⁷ American Cancer Society. Types of Non-Hodgkin Lymphoma. American Cancer Society website. <https://www.cancer.org/cancer/types/non-hodgkin-lymphoma/about/b-cell-lymphoma.html>. Accessed November 21, 2024.

¹⁸ American Cancer Society. B-cell Lymphoma. American Cancer Society website. <https://www.cancer.org/cancer/types/non-hodgkin-lymphoma/about/b-cell-lymphoma.html>. Updated January 12, 2024. Accessed November 21, 2024.

¹⁹ American Cancer Society. B-cell Lymphoma. American Cancer Society website. <https://www.cancer.org/cancer/types/non-hodgkin-lymphoma/about/b-cell-lymphoma.html>. Updated January 12, 2024. Accessed November 21, 2024.

²⁰ American Cancer Society. Treating B-cell Lymphoma. American Cancer Society website. <https://www.cancer.org/cancer/types/non-hodgkin-lymphoma/treating/b-cell-lymphoma.html>. Updated January 12, 2024. Accessed November 21, 2024.

²¹ American Cancer Society. B-cell Lymphoma. American Cancer Society website. <https://www.cancer.org/cancer/types/non-hodgkin-lymphoma/about/b-cell-lymphoma.html>. Updated January 12, 2024. Accessed November 21, 2024.

²² Leukemia & Lymphoma Society. Marginal Zone Lymphoma Facts. Leukemia & Lymphoma Society website. https://www.lls.org/sites/default/files/2022-06/PS84_MZL_2022_FINAL.pdf. Published June 2022. Accessed November 21, 2024.

lymphoma, each arising from distinct tissues and presenting unique characteristics.²³

Chronic Lymphocytic Leukemia (CLL) is one of the most common types of leukemia in adults, originating in the early forms of white blood cells (lymphocytes) in the bone marrow. This cancer typically begins in B lymphocytes and progresses as leukemia cells accumulate in the bone marrow and spread to the bloodstream. Over time, these cells may also infiltrate other parts of the body, including the lymph nodes, liver, and spleen. CLL usually develops slowly, with many individuals remaining symptom-free for years, but it is often harder to cure compared to acute leukemias.²⁴ While most people with CLL can live with the disease for many years, the condition compromises the immune system, increasing susceptibility to infections.²⁵ Historically considered a leukemia, CLL was reclassified in 2001 by the World Health Organization (WHO) as a subtype of NHL, recognizing CLL and Small Lymphocytic Lymphoma (SLL) as different manifestations of the same disease.²⁶ The rationale for this reclassification was based on the recognition that CLL and SLL involve the same type of malignant B-cell (small lymphocyte), with the main difference being where the cancer cells are found – in the blood/bone marrow for CLL and in lymph nodes for SLL. This change was reflected in epidemiological studies and cancer registries starting in the early 2000s. This classification has persisted in subsequent revisions of the WHO system and is now widely accepted in clinical practice and research.²⁷

B. IARC's Assessment of TCE and Cancer Risk, Specifically NHL

The International Agency for Research on Cancer (IARC) is a specialized cancer agency of the World Health Organization (WHO). IARC promotes international and interdisciplinary collaboration in cancer research, bringing together skills in epidemiology, laboratory sciences, and biostatistics to identify the causes of cancer so that preventive measures may be adopted, and the burden of disease and associated suffering reduced. The IARC Monographs program is a core element of the Agency's portfolio of activities, with international expert working groups applying rigorous procedures for the scientific review and evaluation of carcinogenic hazards. Given the potential importance and impact of the classification of an agent, consideration has

²³ National Cancer Institute. Non-Hodgkin Lymphoma Treatment (PDQ®)—Patient Version. National Cancer Institute website. <https://www.cancer.gov/types/lymphoma/patient/adult-nhl-treatment-pdq>. Updated August 15, 2024. Accessed November 21, 2024

²⁴ American Cancer Society. What Is Chronic Lymphocytic Leukemia (CLL)? American Cancer Society website. <https://www.cancer.org/cancer/types/chronic-lymphocytic-leukemia/about/what-is-cll.html>. Updated January 12, 2024. Accessed November 21, 2024.

²⁵ American Cancer Society. Living as a Chronic Lymphocytic Leukemia Survivor. American Cancer Society website. <https://www.cancer.org/cancer/types/chronic-lymphocytic-leukemia/after-treatment/follow-up.html>. Updated January 12, 2024. Accessed November 21, 2024.

²⁶ Howlader N, Morton LM, Feuer EJ, Besson C, Engels EA. Contributions of Subtypes of Non-Hodgkin Lymphoma to Mortality Trends. *Cancer Epidemiol Biomarkers Prev*. 2016 Jan;25(1):174-9. doi: 10.1158/1055-9965.EPI-15-0921. Epub 2015 Oct 15. PMID: 26472423; PMCID: PMC4713325.

²⁷ Al-Hamadani M, Habermann TM, Cerhan JR, Macon WR, Maurer MJ, Go RS. Non-Hodgkin lymphoma subtype distribution, geodemographic patterns, and survival in the US: A longitudinal analysis of the National Cancer Data Base from 1998 to 2011. *Am J Hematol*. 2015 Sep;90(9):790-5. doi: 10.1002/ajh.24086. Epub 2015 Jul 27. PMID: 26096944.

long been given to managing conflicts of interest on the part of all participants in working group meetings. In particular, since 2006, for enhanced transparency, IARC has strengthened conflict-of-interest management, delineated the distinct roles of different participants (working group members, invited specialists, representatives, observers, IARC Secretariat), and ensured that the working groups follow a robust process for identifying, evaluating, and disclosing conflicts of interest.²⁸ The result, in my opinion, is that IARC monographs are thought of as being rigorous and, in general, authoritative, with conclusions made in the monographs carrying substantial weight in the scientific community.

In 2014, based on the deliberations of a working group of experts that met from October 2–9, 2012, IARC published a monograph (Volume 106) that addressed the carcinogenicity of several chlorinated agents, including TCE.²⁹ With regards to TCE and toxicological evidence, the monograph authors reviewed a large body of experimental evidence in animals (rodents), including those involving the oral administration of TCE and inhalation of TCE; and mechanistic data on TCE's metabolism and genotoxicity; and non-genotoxic mechanisms of TCE-related carcinogenesis. IARC concluded that TCE metabolites are genotoxic, and that TCE is immunotoxic.

With regards to TCE and epidemiological evidence, the monograph authors reviewed a large body of evidence including both cohort and case-control studies (as well as ecological studies). The cohort studies included those with a particular focus on studies of workers in the dry-cleaning industry, the aircraft and aerospace industries, and other manufacturing industries in which TCE was used as a solvent. The case-control studies included those from Europe, Canada, and the United States. The working group also reviewed several meta-analyses of the epidemiological literature on risk of cancer among persons exposed to TCE, selecting those that were recent and comprehensive, and assembled, presented, or analyzed the literature in ways beyond the text of the individual publications.

Overall, based on its review, the IARC working group concluded that there was sufficient evidence in experimental animals for the carcinogenicity of TCE; and that there was sufficient evidence in humans for the carcinogenicity of TCE. "Sufficient evidence" under the IARC classification is the highest classification level for these two categories. This combination of findings resulted in TCE being designated as a Group I carcinogen, i.e., it is carcinogenic to humans (the highest overall IARC classification). With regards to specific cancers, the working group concluded that TCE causes cancer of the kidney; and that a positive association has been

²⁸ Samet JM, Chiu WA, Coglianò V, Jinot J, Kriebel D, Lunn RM, Beland FA, Bero L, Browne P, Fritschi L, Kanno J, Lachenmeier DW, Lan Q, Lasfargues G, Le Curieux F, Peters S, Shubat P, Sone H, White MC, Williamson J, Yakubovskaya M, Siemiatycki J, White PA, Guyton KZ, Schubauer-Berigan MK, Hall AL, Grosse Y, Bouvard V, Benbrahim-Tallaa L, El Ghissassi F, Lauby-Secretan B, Armstrong B, Saracci R, Zavadil J, Straif K, Wild CP. The IARC Monographs: Updated Procedures for Modern and Transparent Evidence Synthesis in Cancer Hazard Identification. *J Natl Cancer Inst.* 2020 Jan 1;112(1):30-37. doi: 10.1093/jnci/djz169. PMID: 31498409; PMCID: PMC6968684.

²⁹ IARC. *Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents*. Volume 106, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. 2014. International Agency for Research on Cancer, Lyons, France. WHO Press, World Health Organization.

observed between exposure to TCE and NHL (and liver cancer). The working group based its assessment of TCE and NHL on eight independent cohort studies and eight case-control studies, as well as the published results of several meta-analyses of cohort and case-control studies reporting statistically significant meta-relative risks, such as a 2006 study by Mandel et al.³⁰ that demonstrated a meta-relative risk of 2.33 (95% CI: 1.39–3.91) for TCE and NHL studies with higher-quality exposure data; and a 2011 study by Scott and Jinot³¹ that found a meta-relative risk of 1.43 (95% CI: 1.13–1.82) for TCE and NHL studies when focusing on the highest exposure categories in 10 studies. I have independently reviewed and assessed these peer-reviewed studies relied upon by IARC.

C. NTP's Assessment of TCE and Cancer Risk, Specifically NHL

The National Toxicology Program (NTP) is an interagency program within the Public Health Service of the U.S. Department of Health and Human Services, administratively based in the National Institute of Environmental Health Sciences of the U.S. National Institutes of Health. NTP conducts testing, research, and analysis of agents of concern to identify toxic and biological effects, thereby providing information that strengthens the science base and informs decisions by health regulatory and research agencies to safeguard public health. NTP evaluates cancer hazards by following a rigorous multistep process and using established criteria to review and integrate the scientific evidence from published human, experimental animal, and mechanistic studies.³² Draft monographs undergo external peer review before they are finalized and published. The result, in my opinion, is that, similar to IARC monographs, NTP monographs are thought of as being rigorous and, in general, authoritative, with the conclusions made in the monographs carrying substantial weight.

In 2015, the NTP published a monograph³³ on TCE as part of its reports on carcinogens. In its review, the NTP focused on three types of cancer: kidney cancer, NHL, and liver cancer, using systematic review methods to identify studies, evaluate study quality, integrate evidence

³⁰ Mandel JH, Kelsh MA, Mink PJ, Alexander DD, Kalmes RM, Weingart M, Yost L, Goodman M. Occupational trichloroethylene exposure and non-Hodgkin's lymphoma: a meta-analysis and review. *Occup Environ Med*. 2006 Sep;63(9):597-607. doi: 10.1136/oem.2005.022418. Epub 2006 Apr 27. PMID: 16644896; PMCID: PMC2078160.

³¹ Scott CS, Jinot J. Trichloroethylene and cancer: systematic and quantitative review of epidemiologic evidence for identifying hazards. *Int J Environ Res Public Health*. 2011 Nov;8(11):4238-72. doi: 10.3390/ijerph8114238. Epub 2011 Nov 9. PMID: 22163205; PMCID: PMC3228569.

³² NTP. *Handbook for Preparing Report on Carcinogens Monographs*. National Toxicology Program, National Institute of Environmental Health Sciences, U.S. Department of Health and Human Services, July 20, 2015. Available at: https://ntp.niehs.nih.gov/sites/default/files/ntp/roc/handbook/roc_handbook_508.pdf ; accessed February 3, 2024.

³³ NTP. *Report on Carcinogens: Monograph on Trichloroethylene*. National Toxicology Program, National Institute of Environmental Health Sciences, U.S. Department of Health and Human Services, January, 2015. Available at: https://ntp.niehs.nih.gov/sites/default/files/ntp/roc/monographs/finaltce_508.pdf ; accessed February 4, 2024.

across studies, and integrate evidence across data streams (human, animal, and mechanistic data). Using established criteria, NTP reached conclusions regarding the strength of the evidence for each of the three cancer types and on the recommended listing status of trichloroethylene in the Report on Carcinogens. It is standard practice for scientists to refer to NTP's monographs where available.

Overall, the NTP concluded from its cancer hazard evaluation that there was sufficient evidence to designate TCE as a human carcinogen based on epidemiology studies together with supporting evidence from toxicological, toxicokinetic, and mechanistic studies demonstrating the biological plausibility of its carcinogenicity in humans. In relation specifically to NHL, the NTP noted with respect to experimental animal studies that exposure to TCE by inhalation or gavage caused lymphoma in mice, and that TCE causes immunomodulation in both people and animals, and immunomodulation is linked to NHL. However, NTP noted that the potential association between trichloroethylene-induced immune effects and lymphoma has not been tested directly in either humans or animals. With respect to the epidemiological evidence, NTP concluded that epidemiological studies provide limited evidence that TCE exposure causes NHL, based on positive associations in several studies and evidence for increased risk of NHL across studies combined in two meta-analyses. However, alternative explanations for the associations, such as chance or confounding, could not reasonably be ruled out. Under the NTP classification scheme, this put TCE into an intermediately-ranked category of evidence.³⁴ It carries a meaning similar to the term "limited evidence" used by IARC, i.e., a positive association has been observed between exposure to the agent and cancer but that other explanations for the observations (technically termed "chance," "bias," or "confounding") could not be ruled out with reasonable confidence.³⁵ Having reviewed the IARC and NTP monographs and the studies/sources underlying the monographs, I concur with the evaluations they made (at the time of their publication) regarding the relationship between TCE and NHL.

³⁴ Under the NTP classification scheme the main categories are: 1. Known to be a Human Carcinogen: This classification is used when there is sufficient evidence from human studies that a substance causes cancer; 2. Reasonably Anticipated to be a Human Carcinogen: This category is used when there is limited evidence from human studies and/or sufficient evidence from animal studies. It suggests that a substance may cause cancer in humans, but the evidence is not as strong as for the first category; 3. Not Classifiable as to its Carcinogenicity to Humans: This is used when the evidence is inadequate to make a determination about the substance's potential to cause cancer. Source: NTP (National Toxicology Program). 2021. Report on Carcinogens, Fifteenth Edition. Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service. <https://ntp.niehs.nih.gov/go/roc15> (EndNote XML) DOI: <https://doi.org/10.22427/NTP-OTHER-1003>.

³⁵ NTP (National Toxicology Program). 2021. Report on Carcinogens, Fifteenth Edition. Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service. <https://ntp.niehs.nih.gov/go/roc15> (EndNote XML) DOI: <https://doi.org/10.22427/NTP-OTHER-1003>.

D. Literature That Has Appeared Since the IARC and NTP Reports on the Evidence Surrounding the Potential of TCE to Cause NHL

1. Epidemiological Evidence

Around the time of, and subsequent to, when the IARC and NTP reports appeared, there has been a number of epidemiological studies and experimental studies that pertain to TCE as a potential cause of NHL. Regarding epidemiological studies, of particular relevance are systematic reviews and meta-analyses.

Systematic reviews of epidemiological evidence are increasingly being conducted and used by academics, non-government organizations, industry, and regulators to characterize health hazards and risks posed by exposure to environmental exposures.³⁶ This trend reflects the increasing recognition of the utility of systematic methods for providing a rigorous methodology for aggregating and summarizing evidence in support of regulatory and policy decisions. The earliest versions, such as the approach developed by the Cochrane Collaboration, focused on the results of randomized trials in order to help clinical decision-making.³⁷ However, given that environmental risks are not amenable to randomized trials (as they would be unethical to perform in humans) as well as other factors, approaches to systematic reviews have been developed that are specific to environmental risks, such as the U.S. National Toxicology Program Office of Health Assessment and Translation (NTP OHAT) handbook,^{38,39} the Navigation Guide,⁴⁰ and the Conduct of Systematic Reviews in Toxicology and Environmental Health Research guidelines (COSTER).⁴¹ In general, elements that are common to such systematic reviews are the

³⁶ Whaley P, Halsall C, Ågerstrand M, Aiassa E, Benford D, Bilotta G, Coggon D, Collins C, Dempsey C, Duarte-Davidson R, FitzGerald R, Galay-Burgos M, Gee D, Hoffmann S, Lam J, Lasserson T, Levy L, Lipworth S, Ross SM, Martin O, Meads C, Meyer-Baron M, Miller J, Pease C, Rooney A, Sapiets A, Stewart G, Taylor D. Implementing systematic review techniques in chemical risk assessment: Challenges, opportunities and recommendations. *Environ Int.* 2016 Jul-Aug;92-93:556-64. doi: 10.1016/j.envint.2015.11.002. Epub 2015 Dec 11. PMID: 26687863; PMCID: PMC4881816.

³⁷ Shah HM, Chung KC. Archie Cochrane and his vision for evidence-based medicine. *Plast Reconstr Surg.* 2009 Sep;124(3):982-988. doi: 10.1097/PRS.0b013e3181b03928. PMID: 19730323; PMCID: PMC2746659.

³⁸ Rooney AA, Boyles AL, Wolfe MS, Bucher JR, Thayer KA. Systematic review and evidence integration for literature-based environmental health science assessments. *Environ Health Perspect.* 2014 Jul;122(7):711-8. doi: 10.1289/ehp.1307972. Epub 2014 Apr 22. PMID: 24755067; PMCID: PMC4080517.

³⁹ NTP OHAT, 2019: Handbook for Conducting a Literature-Based Health Assessment Using OHAT Approach for Systematic Review and Evidence Integration. US National Toxicology Program Office of Health Assessment and Translation. Available online at <https://ntp.niehs.nih.gov/pubhealth/hat/review/index-2.html>, accessed on January 14, 2024.

⁴⁰ Woodruff TJ, Sutton P. The Navigation Guide systematic review methodology: a rigorous and transparent method for translating environmental health science into better health outcomes. *Environ Health Perspect.* 2014 Oct;122(10):1007-14. doi: 10.1289/ehp.1307175. Epub 2014 Jun 25. PMID: 24968373; PMCID: PMC4181919.

⁴¹ Whaley P, Aiassa E, Beausoleil C, Beronius A, Bilotta G, Boobis A, de Vries R, Hanberg A, Hoffmann

incorporation of several specific steps, i.e., the development of a protocol, the use of comprehensive search strategies, the employment of a Cochrane-derived risk of bias approach to appraising study quality, and the use of a Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach to rating the certainty of the evidence.⁴²

In reviewing the evidence, if a critical mass of studies is identified of which the studies are sufficiently comparable as to render the combined result meaningful, systematic reviews will often then undertake meta-analyses. Meta-analyses statistically synthesize evidence from multiple research studies with the goal of providing summaries of the evidence that account for and transcend the limitations of individual studies, and that result in quantitative estimates of the average effect across studies and the effect's statistical significance and variability.^{43,44}

In my opinion, given the accumulation of epidemiological studies on the topic of TCE exposure and NHL, employing a systematic review method and, where appropriate, meta-analyses of the associated data represent the optimal approach for weighing the epidemiological evidence in relation to my task of assessing whether there is a causal relationship between exposure to TCE and NHL. Such an approach is also generally accepted in the scientific community. Systematic reviews are complex, multi-disciplinary projects that typically take 12–36 months to conduct, followed by many additional months required to select the studies identified during the review for meta-analysis, perform the meta-analysis, generate a summary manuscript, and steer the manuscript through peer-review towards publication. Multiple investigators are typically involved to ensure a diversity of input, with a mechanism for resolving differences of opinion. Thus, instead of attempting to conduct my own systematic review and, if appropriate, meta-analysis, it is strategically useful to focus on the several systematic reviews (with meta-analyses) relevant to the subject of TCE and NHL that have recently appeared in the peer-reviewed scientific literature.

In the ensuing discussion of the results of these systematic reviews and meta-analyses, certain terms deserve explanation, as they are relatively technical and go beyond basic familiarity with epidemiological terminology. As noted earlier, among the essential steps of systematic

S, Hunt N, Kwiatkowski CF, Lam J, Lipworth S, Martin O, Randall N, Rhomberg L, Rooney AA, Schünemann HJ, Wikoff D, Wolffe T, Halsall C. Recommendations for the conduct of systematic reviews in toxicology and environmental health research (COSTER). *Environ Int.* 2020 Oct;143:105926. doi: 10.1016/j.envint.2020.105926. Epub 2020 Jul 9. PMID: 32653802.

⁴² Morgan RL, Thayer KA, Bero L, Bruce N, Falck-Ytter Y, Gherzi D, Guyatt G, Hooijmans C, Langendam M, Mandrioli D, Mustafa RA, Rehfuss EA, Rooney AA, Shea B, Silbergeld EK, Sutton P, Wolfe MS, Woodruff TJ, Verbeek JH, Holloway AC, Santesso N, Schünemann HJ. GRADE: Assessing the quality of evidence in environmental and occupational health. *Environ Int.* 2016 Jul-Aug;92-93:611-6. doi: 10.1016/j.envint.2016.01.004. Epub 2016 Jan 27. PMID: 26827182; PMCID: PMC4902742.

⁴³ Pigott, T. D., & Polanin, J. R. (2020). Methodological Guidance Paper: High-Quality Meta-Analysis in a Systematic Review. *Review of Educational Research*, 90(1), 24-46. <https://doi.org/10.3102/0034654319877153>.

⁴⁴ Tanner-Smith EE, Grant S, Mayo-Wilson E. Modern Meta-Analytic Methods in Prevention Science: Introduction to the Special Issue. *Prev Sci.* 2022 Apr;23(3):341-345. doi: 10.1007/s11121-022-01354-8. Epub 2022 Feb 16. PMID: 35171463.

reviews is an assessment of study quality. One of the tools used for such an assessment in the studies discussed below is the Newcastle–Ottawa Scale (NOS),⁴⁵ which rates each study based on three components: methodological quality, comparability of each study, and the outcomes and statistical analysis approach. Another systematic review characteristic that is rated is the potential for publication bias, i.e., the tendency to include or exclude relevant studies depending on the nature and direction of the results (rather than the characteristics associated with the study design). A common example of publication bias is if a study is more likely to be included if the results are significant. One of the tools used for this purpose in the systematic reviews discussed below is the funnel plot and Egger’s test,⁴⁶ by which the funnel plot provides a visual diagram (of effect sizes from regression models plotted against their standard errors), and the Egger’s test provides a statistical tool for determining the magnitude of potential publication bias by which the standardized effect sizes are regressed on their precisions (in the absence of publication bias, the regression intercept is expected to be zero). A third systematic review characteristic that is rated and worth mentioning is heterogeneity, an assessment of the differences between studies of their underlying target parameters.⁴⁷ Results of multiple studies always differ to some degree owing to, for example, differences in study target populations or targeted effects, survey recruitment and administration methods, measurement instruments, doses of interventions (if a review involves intervention trials), timing of outcome measurements, and/or analytical methods, including covariate adjustments. A high rating on a scale of heterogeneity weakens somewhat the ability to interpret the results. The most popular data-based measure of study heterogeneity, the I^2 statistic, is quoted in the meta-analyses below, and approaches its maximum of 1 when heterogeneity substantially exceeds within-study sampling and measurement variability. Another test of heterogeneity cited in the meta-analyses below is the chi-square-based Q-test, which assesses whether observed differences in results are compatible with chance alone. A low P value (or a large chi-squared statistic relative to its degree of freedom) provides evidence of heterogeneity effects (variation in effect estimates beyond chance). Finally, the Hedges d statistic is used to represent the overall effect size (and associated statistical significance) of the impact of a risk factor on the outcome of interest in systematic reviews.

2. Systematic Reviews and Meta-Analyses: TCE and NHL

In 2011, Scott and Jinot⁴⁸ published a systematic review and meta-analysis of TCE and cancer that combined 24 cohort and case-control studies. The authors found statistically elevated risks of NHL in association with TCE with respect to overall exposure, for which the relative

⁴⁵ Shaheen N, Shaheen A, Ramadan A, Hefnawy MT, Ramadan A, Ibrahim IA, Hassanein ME, Ashour ME, Flouty O. Appraising systematic reviews: a comprehensive guide to ensuring validity and reliability. *Front Res Metr Anal.* 2023 Dec 21;8:1268045. doi: 10.3389/frma.2023.1268045. PMID: 38179256; PMCID: PMC10764628.

⁴⁶ Lin L, Chu H. Quantifying publication bias in meta-analysis. *Biometrics.* 2018 Sep;74(3):785-794. doi: 10.1111/biom.12817. Epub 2017 Nov 15. PMID: 29141096; PMCID: PMC5953768.

⁴⁷ Imrey PB. Limitations of Meta-analyses of Studies With High Heterogeneity. *JAMA Netw Open.* 2020 Jan 3;3(1):e1919325. doi: 10.1001/jamanetworkopen.2019.19325. PMID: 31922554.

⁴⁸ Scott CS, Jinot J. Trichloroethylene and cancer: systematic and quantitative review of epidemiologic evidence for identifying hazards. *Int J Environ Res Public Health.* 2011 Nov;8(11):4238-72. doi: 10.3390/ijerph8114238. Epub 2011 Nov 9. PMID: 22163205; PMCID: PMC3228569.

risk estimate was 1.23 (95% CI: 1.07, 1.42) and with respect to the highest exposure group, for which the relative risk estimate was 1.43 (95% CI: 1.13, 1.82). Among the limitations were issues of study heterogeneity, potential publication bias, and somewhat weak dose-response relationships.

Although not the result of a systematic review, Cocco et al. (2013)⁴⁹ was able to pool the results of four case-control studies that contributed to the International Lymphoma Epidemiology Consortium. As a result, in my opinion, given the relatively large sample size it afforded as well as the relatively rigorous methodology employed by the parent studies, it deserves mention. Overall, the pooled study population included 3,788 NHL cases and 4,279 controls. The risk of NHL and its major subtypes associated with TCE exposure was calculated with unconditional logistic regression and polytomous regression analysis, adjusting by age, gender, and study. As noted by the authors, the risk of follicular lymphoma (FL)—a subtype of NHL—but not NHL overall or other subtypes, increased by probability ($p=0.02$) and intensity level ($p=0.04$), and with the combined analysis of four exposure metrics assumed as independent ($p=0.004$). After restricting the analysis to the most likely exposed study subjects, risk of NHL overall, FL and chronic lymphocytic leukemia (CLL)⁵⁰ were elevated and increased by duration of exposure ($p=0.009$, $p=0.04$ and $p=0.01$, respectively) and with the combined analysis of duration, frequency, and intensity of exposure ($p=0.004$, $p=0.015$ and $p=0.005$, respectively). Although based on small numbers of exposed, risk of all the major NHL subtypes, namely diffuse large B-cell lymphoma (DLBCL), FL, and CLL, showed increases in risk ranging 2–3.2-fold in the highest category of exposure intensity. Based on the Q-test, no significant heterogeneity in risk was detected by major NHL subtypes or by study.

Also published in 2013 was a systematic review and meta-analysis of TCE in relation to lymphatic and hematopoietic cancers conducted by Karami et al.⁵¹ and colleagues from the U.S. National Cancer Institute. The meta-analysis for NHL incorporated results for 293 NHL cases from 12 cohort (228 cases from 10 ‘TCE-exposure’ and 65 cases from two ‘chlorinated solvent-exposure’) studies and 8,140 NHL cases from 12 case-control (6,095 cases from nine ‘TCE-exposure’ and 2,045 cases from three ‘chlorinated solvent-exposure’) studies. Among the rigorous methods used by the authors that improved upon previous reviews was a focus on TCE exposures classified by an industrial hygienist, which decreased the potential for exposure

⁴⁹ Cocco P, Vermeulen R, Flore V, Nonne T, Campagna M, Purdue M, Blair A, Monnereau A, Orsi L, Clavel J, Becker N, de Sanjosé S, Foretova L, Staines A, Maynadié M, Nieters A, Miligi L, 't Mannetje A, Krickler A, Brennan P, Boffetta P, Lan Q, Rothman N. Occupational exposure to trichloroethylene and risk of non-Hodgkin lymphoma and its major subtypes: a pooled InterLymph [correction of IinterLymph] analysis. *Occup Environ Med*. 2013 Nov;70(11):795-802. doi: 10.1136/oemed-2013-101551. Epub 2013 Jul 23. PMID: 23881218.

⁵⁰ As noted earlier, until 2001, CLL was classified as a leukemia but since then, beginning with the World Health Organization, CLL has more commonly been characterized as a NHL subtype.

⁵¹ Karami S, Bassig B, Stewart PA, Lee KM, Rothman N, Moore LE, Lan Q. Occupational trichloroethylene exposure and risk of lymphatic and haematopoietic cancers: a meta-analysis. *Occup Environ Med*. 2013 Aug;70(8):591-9. doi: 10.1136/oemed-2012-101212. Epub 2013 May 30. PMID: 23723297.

misclassification⁵². A significantly raised summary estimate for NHL was seen for all cohort and case-control ‘TCE-exposure’ studies combined (N=19; relative risk (RR)=1.32, 95% CI 1.14 to 1.54; $I^2=25.20$; p-heterogeneity=0.12) and for cohort ‘TCE-exposure’ studies (N=10; RR=1.52, 95% CI 1.29 to 1.79; $I^2=7.09$; p-heterogeneity=0.63). The results were stronger when confined to incident (v. mortality) studies: NHL Rel Risk =1.66 (95% CI: 1.29-2.14; $I^2=3.19$; p-heterogeneity=0.36). No evidence of publication bias, assessed using the Begg and Egger tests as well as funnel plots, was seen for analyses involving NHL cohort and case-control studies combined. In the four cohort studies that examined duration of exposure, a significant summary risk estimate of 1.56 (95% CI 1.02 to 2.40; $I^2=4.66$; p-heterogeneity=0.20) was seen among workers who had a longer duration of exposure (≥ 6.25 years, ≥ 5 years, ≥ 2 years) compared with those with a shorter exposure duration (< 6.25 years, < 5 years, < 2 years) (RR=1.30, 95% CI 0.92 to 1.84; $I^2=3.75$; p-heterogeneity>0.29), which constitutes evidence of a dose-response relationship. Finally, the results were even stronger when confined to 3 studies that used urine biomarkers to classify exposure: NHL relative risk=2.15 (95% CI: 1.34-3.45; $I^2=1.47$; p-heterogeneity=0.48), which further reduced the potential for exposure misclassification.

More recently, Odutola et al.⁵³ published in 2021 a systematic review and meta-analysis of occupational exposures, including TCE, in relation to follicular lymphoma (FL; a subtype of NHL). The systematic review focused on 10 cohort and 37 case-control studies that quantified FL risk in relation to any exposure to one or more occupational groups or agents. Of these, 8 cohort and 19 case-control studies examined dose-response relationships. The authors found that exposure to chlorinated solvents (ever v. never) in 4 studies was associated with a relative risk of FL of 1.35 (95% CI: 1.09-1.68; $I^2 = 0.0$) with no statistical evidence of heterogeneity ($I^2 = 0.0\%$, $p = 0.702$).

3. Mechanistic Evidence

In my opinion, a number of mechanistic studies (both in humans and animals, and in vitro) that deserve consideration were published beginning in 2012 that were either not included by or published after the 2014 IARC and 2015 NTP reports on TCE. In 2012, Jiao et al.⁵⁴ published a molecular epidemiology case-control study of 518 NHL cases and 597 controls among women in Connecticut that tested the hypothesis that the association between solvent

⁵² Exposure misclassification refers to errors in determining or assigning levels or degrees of exposure (e.g., “low”, “medium”, “high”) for subjects in a study. In general, in an epidemiological study, exposure misclassification that is random tends to dilute the true relationship between an exposure and an outcome. By contrast, some forms of exposure misclassification can introduce a form of bias that, depending on specifics, can falsely promote the illusion of a relationship, or, alternatively, promote the illusion of a lack of a relationship, between an exposure and an outcome.

⁵³ Odutola MK, Benke G, Fritschi L, Giles GG, van Leeuwen MT, Vajdic CM. A systematic review and meta-analysis of occupational exposures and risk of follicular lymphoma. *Environ Res*. 2021 Jun;197:110887. doi: 10.1016/j.envres.2021.110887. Epub 2021 Feb 17. PMID: 33607095.

⁵⁴ Jiao J, Zheng T, Lan Q, Chen Y, Deng Q, Bi X, Kim C, Holford T, Leaderer B, Boyle P, Ba Y, Xia Z, Chanock SJ, Rothman N, Zhang Y. Occupational solvent exposure, genetic variation of DNA repair genes, and the risk of non-Hodgkin's lymphoma. *Eur J Cancer Prev*. 2012 Nov;21(6):580-4. doi: 10.1097/CEJ.0b013e328351c762. PMID: 22430443; PMCID: PMC3397155.

exposure and risk of NHL was modified by genetic variations in DNA repair genes. Among their findings was that the *MGMT* allelic variant (rs12917) modified the association between chlorinated solvents and risk of diffuse large B-cell lymphoma ($P_{\text{for interaction}} = 0.0027$) and follicular lymphoma ($P_{\text{for interaction}} = 0.0024$).

Deng et al. (2013)⁵⁵ extended the work of Jiao et al. with an updated data set of 601 incident cases of NHL and 717 controls among women in Connecticut, this time, testing the hypothesis that the association between solvent exposure and risk of NHL was modified by genetic variations in immune genes. The study found that polymorphism in interleukin 10 (IL10; rs1800890) modified the association between occupational exposure to organic solvents and the risk for diffuse large B-cell lymphoma ($P_{\text{for interaction}} = 0.0058$). The results remained statistically significant after adjustment for false discovery rate. Compared with women who were never occupationally exposed to any organic solvents, women who were exposed to organic solvents at least once had a significantly increased risk for diffuse large B-cell lymphoma if they carried the IL10 (rs1800890) TT genotype (odds ratio=3.31, 95% confidence interval: 1.80–6.08), but not if they carried the AT/AA genotype (odds ratio=1.14, 95% confidence interval: 0.72–1.79).

In a molecular epidemiology study in a different population, Zhang et al. (2013)⁵⁶ tested the hypothesis that TCE exposure would be associated with markers of immune dysfunction among 80 TCE-exposed workers in comparison to 45 unexposed controls matched by age and gender. They found that the exposed workers had about a 17.5% decline in serum levels of IgG compared with the unexposed controls ($P = 0.0002$). Similarly, serum levels of IgM were reduced by about 38% in the workers exposed to TCE compared with the unexposed controls ($P < 0.0001$). Serum levels of both IgG and IgM were significantly decreased in workers exposed to TCE levels below 12 ppm, the median exposure level.

In an in vitro study, Varshney et al. (2014)⁵⁷ examined the genotoxic potential of trichloroacetic acid, a persistent metabolite of TCE. Lymphocytes were obtained from 3 healthy human donors and exposed to 25, 50, and 100 $\mu\text{g/ml}$ concentrations of TCA separately. TCA exposure was found to result in chromosomal anomalies and the formation of micronuclei in lymphocytes. Chromosome analysis revealed a dose-dependent effect and significant induction of chromosomal anomalies. Chromatid breaks/chromosome breaks, fragments, and chromatid

⁵⁵ Deng Q, Zheng T, Lan Q, Lan Y, Holford T, Chen Y, Dai M, Leaderer B, Boyle P, Chanock SJ, Rothman N, Zhang Y. Occupational solvent exposure, genetic variation in immune genes, and the risk for non-Hodgkin lymphoma. *Eur J Cancer Prev.* 2013 Jan;22(1):77-82. doi: 10.1097/CEJ.0b013e328354d2c1. PMID: 22609637; PMCID: PMC3469764.

⁵⁶ Zhang L, Bassig BA, Mora JL, Vermeulen R, Ge Y, Curry JD, Hu W, Shen M, Qiu C, Ji Z, Reiss B, McHale CM, Liu S, Guo W, Purdue MP, Yue F, Li L, Smith MT, Huang H, Tang X, Rothman N, Lan Q. Alterations in serum immunoglobulin levels in workers occupationally exposed to trichloroethylene. *Carcinogenesis.* 2013 Apr;34(4):799-802. doi: 10.1093/carcin/bgs403. Epub 2012 Dec 30. PMID: 23276795; PMCID: PMC3616671.

⁵⁷ Varshney M, Chandra A, Chauhan LK, Goel SK. In vitro cytogenetic assessment of trichloroacetic acid in human peripheral blood lymphocytes. *Environ Sci Pollut Res Int.* 2014 Jan;21(2):843-50. doi: 10.1007/s11356-013-1949-6. Epub 2013 Jun 30. PMID: 23812791.

exchanges were commonly observed.

Lee et al. (2019)⁵⁸ evaluated the association between occupational exposure to TCE and serum levels of microRNAs (miRNA) in a molecular epidemiology study of TCE exposed workers and unexposed controls. MiRNAs are a group of regulatory RNAs that exert regulatory functions that result in gene silencing. As such, aberrant miRNA expression profiles are well known to play a critical role in tumorigenesis and tumor development, including the development of lymphomas through altered regulation of target oncogenes or tumor suppressors. Lee et al. found that seven miRNAs showed significant differences between exposed and unexposed workers at FDR (false discovery rate) < 0.20. miR-150-5p and let-7b-5p also showed significant inverse exposure-response associations with TCE exposure (Ptrend=0.002 and 0.03, respectively). The authors noted that miR-150-5p is involved in B-cell receptor pathways and let-7b-5p plays a role in the innate immune response processes that are potentially important in the etiology of NHL.

In a molecular epidemiology study, Phillips et al. (2019)⁵⁹ conducted an epigenome-wide association study (EWAS) in TCE exposed workers. Across individual CpG probes, genomic regions, and globally (i.e., the 450K methylome), they investigated differences mean DNA methylation and differences in variability of DNA methylation between 73 control (< 0.005 ppm TCE), 30 lower exposed (< 10 ppm TCE), and 37 higher exposed (≥ 10 ppm TCE) subjects' white blood cells. They found that TCE exposure was associated with increased methylation variation globally (Kruskal-Wallis p-value = 3.75e-3) and in 25 CpG sites at a genome-wide significance level (Bonferroni p-value < 0.05). They identified a 609 base pair region in the TRIM68 gene promoter that exhibited hypomethylation with increased exposure to TCE (FWER = 1.20e-2). Also, genes that matched differentially variable CpGs were enriched in the 'focal adhesion' biological pathway (p-value = 2.80e-2). All in all, human exposure to TCE was associated with epigenetic alterations in genes involved in cell-matrix adhesions and interferon subtype expression and in genes related to cancer development.

Finally, Hosgood et al.⁶⁰ (2022) conducted a molecular epidemiology study of serum

⁵⁸ Lee KM, Bassig BA, Zhang L, Vermeulen RC, Hu W, Wong JYY, Qiu C, Wen C, Huang Y, Purdue MP, Ji BT, Li L, Tang X, Rothman N, Smith MT, Lan Q. Association between occupational exposure to trichloroethylene and serum levels of microRNAs: a cross-sectional molecular epidemiology study in China. *Int Arch Occup Environ Health*. 2019 Nov;92(8):1077-1085. doi: 10.1007/s00420-019-01448-x. Epub 2019 Jun 3. PMID: 31161417; PMCID: PMC6953905.

⁵⁹ Phillips RV, Rieswijk L, Hubbard AE, Vermeulen R, Zhang J, Hu W, Li L, Bassig BA, Wong JYY, Reiss B, Huang Y, Wen C, Purdue M, Tang X, Zhang L, Smith MT, Rothman N, Lan Q. Human exposure to trichloroethylene is associated with increased variability of blood DNA methylation that is enriched in genes and pathways related to autoimmune disease and cancer. *Epigenetics*. 2019 Nov;14(11):1112-1124. doi: 10.1080/15592294.2019.1633866. Epub 2019 Jun 26. PMID: 31241004; PMCID: PMC6773405.

⁶⁰ Hosgood HD, Rahman ML, Blansky D, Hu W, Davitt M, Wen C, Huang Y, Tang X, Li L, Smith MT, Zhang L, Vermeulen R, Rothman N, Bassig BA, Lan Q. Targeted proteomic scan identifies alteration of serum proteins among workers occupationally exposed to low levels of trichloroethylene. *Environ Mol Mutagen*. 2022 Dec;63(8-9):423-428. doi: 10.1002/em.22518. PMID: 36346153.

proteins known to be related to carcinogenesis among 42 TCE exposed and 34 unexposed factory workers. Exposure to TCE was associated with lower levels of tumor necrosis factor receptor superfamily member 17 (TNFRSF17; $\beta = -.08$; p-value = .0003) and kynureninase (KYNU; $\beta = -.10$, p-value = .002). These proteins also showed a significant dose-response relation across the unexposed, low exposed, and high exposed workers (all p-trends < .001, false discovery rate [FDR] < 0.20). Pathway analysis of TCE-related proteins showed significant enrichment (FDR < 0.05) for several inflammatory and immune pathways. TCE exposure was associated with TNFRSF17, a key B-cell maturation antigen that mediates B-cell survival and KYNU, an enzyme that plays a role in the T-cell mediated immune response. Given that altered immunity is an established risk factor for NHL, the authors interpreted their findings as supporting the biological plausibility of linking TCE exposure with NHL.

E. Bradford Hill Considerations

Having reviewed the evidence regarding the association between TCE and NHL, it is useful to assess this evidence according to the Bradford Hill factors.

BRADFORD HILL FACTORS

1. Strength of association: A small association does not mean that there is not a causal effect, though the larger the association, the more likely that it is causal.
2. Consistency: Consistent findings observed by different persons in different places with different samples strengthens the likelihood of an effect.
3. Specificity: Causation is likely if a very specific population at a specific site and disease with no other likely explanation. The more specific an association between a factor and an effect is, the bigger the probability of a causal relationship. However, Hill cautioned against overemphasizing specificity in light of the fact that some environmental factors can cause more than one disease.
4. Temporality: The effect has to occur after the cause (and if there is an expected delay between the cause and expected effect, then the effect must occur after that delay).
5. Biological gradient: Greater exposure should generally lead to greater incidence of the effect. However, in some cases, the mere presence of the factor can trigger the effect. In other cases, an inverse proportion is observed: greater exposure leads to lower incidence.
6. Plausibility: A plausible mechanism between cause and effect is helpful (but Hill noted that knowledge of the mechanism is limited by current knowledge).
7. Coherence: Coherence between epidemiological and laboratory findings increases the likelihood of an effect. However, Hill noted that "... lack of such [laboratory] evidence cannot nullify the epidemiological effect on associations".
8. Experiment: "Occasionally it is possible to appeal to experimental evidence".
9. Analogy: The effect of similar factors may be considered.

1. **Strength of association:** In terms of strength of association, in addition to the meta-analyses discussed above that demonstrate a significant association between TCE and NHL, several of the individual epidemiological studies provide evidence of a strong association between TCE

exposure and NHL. Among them are the case-control study by Hardell et al. (1994)⁶¹ that found an Odds Ratio for the association of TCE with NHL of 7.2 (95% CI: 1.3–42); an occupational cohort study by Hansen et al. (2001)⁶² that found that workers exposed to TCE had an elevated Standardized Incidence Ratio (SIR) for NHL of 3.5 (95% CI: 1.3–6.1); and an occupational cohort study by Raaschou-Nielsen et al.⁶³ (2003) that found in a subcohort of 14,360 presumably highly exposed workers, the SIR for NHL was 1.5 (95% CI: 1.2–2.0). Moreover, in the meta-analysis conducted by Karami et al. of TCE cohort studies that used bio-monitoring to estimate TCE exposure, which likely afforded more precise estimates of exposure, the relative risk of NHL was 2.15 (95% CI: 1.34–3.45; $I^2=1.47$; p-heterogeneity=0.48). Thus, in my opinion, the evidence in support of this criterion is strong.

2. **Consistency:** As can be seen in the most recent meta-analysis conducted by Karami et al. (2013), the findings of the cohort and case-control studies that found statistically elevated risks of NHL in relation to TCE exposure demonstrate consistency across the USA and several countries of Europe, based on different population samples, and different investigators. Thus, in my opinion, the evidence in support of this criterion is strong.
3. **Specificity:** The epidemiological evidence discussed in this analysis is specific to the relationship of TCE and NHL and limited to those studies in which TCE exposure can be distinguished from other exposures and NHL could be distinguished from other hematological malignancies. Thus, in my opinion, the evidence in support of this criterion is strong.
4. **Temporality:** In the cohort studies that found a significant association between TCE exposure and NHL, including those discussed above and reviewed in the meta-analyses discussed above, exposure preceded disease, providing evidence of temporality. Additionally, studies that have adjusted for a lag period in the onset of cancer have found a stronger association between TCE and NHL, which aligns with the latency period one would expect between exposure and the onset of a cancer such as NHL. Thus, in my opinion, the evidence in support of this criterion is strong.
5. **Biological Gradient:** As noted in the systematic review and meta-analysis conducted by Karami et al. (2013), several studies demonstrated a dose-response relationship with respect to duration of TCE exposure and NHL that are consistent with a biological gradient. In my opinion, the evidence in support of this criterion is present, but modest.

⁶¹ Hardell L, Eriksson M, Degerman A. Exposure to phenoxyacetic acids, chlorophenols, or organic solvents in relation to histopathology, stage, and anatomical localization of non-Hodgkin's lymphoma. *Cancer Res.* 1994 May 1;54(9):2386-9. PMID: 8162585.

⁶² Hansen J, Raaschou-Nielsen O, Christensen JM, Johansen I, McLaughlin JK, Lipworth L, Blot WJ, Olsen JH. Cancer incidence among Danish workers exposed to trichloroethylene. *J Occup Environ Med.* 2001 Feb;43(2):133-9. doi: 10.1097/00043764-200102000-00012. PMID: 11227631.

⁶³ Raaschou-Nielsen O, Hansen J, McLaughlin JK, Kolstad H, Christensen JM, Tarone RE, Olsen JH. Cancer risk among workers at Danish companies using trichloroethylene: a cohort study. *Am J Epidemiol.* 2003 Dec 15;158(12):1182-92. doi: 10.1093/aje/kwg282. PMID: 14652303.

6. **Biological Plausibility:** The mechanistic, experimental, and animal evidence are supportive based on evidence of genotoxicity, immunosuppression, autoimmunity, and biomarkers consistent with cancer changes related to miRNA, epigenetics, and chromosomal aberrations. Thus, in my opinion, the evidence in support of this criterion is strong.
7. **Coherence:** The correspondence between the epidemiological evidence and the experimental toxicological evidence noted by IARC, and confirmed by my review, is consistent with coherence.
8. **Experimental Evidence:** Experimental epidemiological evidence is not available for human exposures to TCE (and would be unethical to accomplish). However, it is important to note that the toxicological evidence regarding TCE's genotoxic and immunotoxic mechanisms are rooted in numerous sound experimental studies.
9. **Analogy:** There is similarity between the likely mechanisms of action of TCE with other immunotoxic agents known to induce lymphoma, e.g., azathioprine given for inflammatory bowel disease (Pasternak et al., 2013), which supports this viewpoint.

F. Conclusion: TCE Exposure More Likely Than Not Causes NHL

In conclusion, the epidemiological evidence of the association between TCE and NHL is relatively limited, due, in part, to the fact that NHL is a relatively rare disease and that other than history, there are no tools (e.g., biological markers) with which to assess historical and/or cumulative exposures to TCE; and even if histories are available, the ability to disentangle exposure to TCE from other solvents is typically limited. These factors account for the limited conclusions reached by the IARC and NTP reports with regards to TCE and NHL. On the other hand, substantial epidemiological and mechanistic evidence has appeared subsequent to the IARC and NTP deliberations that generated their reports, including, (a) the systematic review and meta-analyses conducted by Karami et al. (2013) and Odutola et al. (2021), and (b) the recent biomarker epidemiology studies of TCE's genotoxicity, immunotoxicity, autoimmunity, and chromosomal aberrations, miRNA perturbations, and epigenetic changes consistent with carcinogenesis. In my opinion, the analysis of the currently assembled evidence using the Bradford Hill framework makes it more likely than not that TCE is a cause of NHL (and therefore also exceeds the standard of "at least as likely as not" prescribed by the Camp Lejeune Justice Act).

II. PCE, Cancer, and NHL

A. Background

1. Tetrachloroethylene (a.k.a. perchloroethylene; PCE)

Tetrachloroethylene (a.k.a. perchloroethylene, PCE) is a non-flammable colorless liquid. Other names for tetrachloroethylene besides perchloroethylene and PCE include PERC, tetrachloroethene, and perchlor. In the 1950's, about 80% of PCE was used in dry-cleaning, and 15% in metal-cleaning and vapour degreasing. By the 1980's, the pattern of usage was changing,

with about 50% of PCE used for dry-cleaning, 28% for chemical intermediates, and 10–15% for metal cleaning and degreasing. With the continuing decline of its use for dry-cleaning, PCE is now used primarily for producing fluorocarbons (ATSDR, 2019).

B. IARC's Assessment of PCE and Cancer Risk

A description of the International Agency for Research on Cancer (IARC), a specialized cancer agency of the World Health Organization (WHO), was provided earlier in the discussion surrounding TCE in Section I.B.

The 2014 monograph that IARC published on the deliberations of a working group of experts that met from October 2–9, 2012 to address the case of TCE (as noted in Section I.B.) also discussed the evidence with respect to PCE. Although in comparison to TCE, the chemistry of PCE is very similar, its metabolism is quite distinct, particularly with respect to the much lower ability of PCE to be metabolized by the CYP-dependent oxidation pathway and its more lipophilic properties (leading to greater sequestration in fat). With regards to PCE and toxicological evidence, the monograph authors reviewed a large body of experimental evidence in animals (rodents), including those involving the administration of PCE orally, by inhalation, by skin application, and by intraperitoneal injection. The working group also reviewed mechanistic data on PCE's metabolism, toxicokinetics, and genotoxicity; and non-genotoxic mechanisms of PCE-related carcinogenesis.

With regards to PCE and epidemiological evidence, the monograph authors reviewed a body of evidence including both cohort and case-control studies (as well as ecological studies). The cohort studies included those with a particular focus on studies of workers in the dry-cleaning industry and other industries in which PCE was used as a solvent. The case-control studies included those from Europe, Canada, and the United States. The working group also commented on a meta-analysis of research on 8 populations that appeared in the epidemiological literature on risk of pancreatic cancer among dry-cleaning workers, noting that a summary risk was found of 1.4 (95% CI, 1.1–2.4), with the caveat that exposure to other solvents also occurred in the populations involved.

Overall, based on its review, the IARC working group concluded that there was sufficient evidence in experimental animals for the carcinogenicity of PCE; and that there was limited evidence in humans for the carcinogenicity of PCE. “Sufficient” evidence under the IARC classification is the highest level for these two categories, and “limited evidence” is IARC's second highest classification level. This combination of findings resulted in PCE being designated as a Group 2A carcinogen, i.e., it is *probably* carcinogenic to humans.

With regards to specific cancers, the working group focused on epidemiological studies of dry-cleaning workers in the United States and in four Nordic countries, given that they were likely to involve exposures primarily to PCE (since PCE became the most commonly used dry-cleaning solvent in the 1950s), and not mixtures of solvents. The cancer outcome that the working group felt emerged to be most consistently associated with PCE was bladder cancer. Methodologically, the studies were mostly able to control for potential confounding by smoking;

limitations included the fact that employment in dry-cleaning was in most cases the only indicator of exposure to PCE, the number of exposed cases was small, and support for an exposure-response relationship was lacking.

With respect specifically to NHL, the working group observed that there were 3 cohort studies that showed an increased risk; however, they were based on small numbers, with the largest study that had the best control of potential confounders not finding an increased risk of NHL. The working group also noted that the case control studies of NHL did not find significant associations between PCE and NHL.

In terms of other cancers, statistically significant increases in mortality from cancer of the esophagus, kidney cancer, and cervical cancer were noted in some studies, but overall, the evidence base was considered inconsistent and limited based on lack of control for confounders, small sample sizes, and other issues.

C. NTP's Assessment of PCE and Cancer Risk, NHL

The U.S. Department of Health and Human Services released the 15th Report on Carcinogens (RoC) in January 2021. The RoC is a congressionally mandated, science-based, public health document that the National Toxicology Program (NTP) prepares for the HHS Secretary. This cumulative report currently includes 256 listings of agents, substances, mixtures, and exposure circumstances that are known or reasonably anticipated to cause cancer in humans. In the 15th Report, the NTP stated that PCE "...is *reasonably anticipated to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in experimental animals." The discussion of evidence underlying the statement parallels that of the 2014 IARC report; however, the list of references makes it clear that the information from IARC that appears in the entry on PCE in the January 2021 NTP report (the most recent such report available publicly) was the report by IARC in 1995, i.e., *not IARC's more recent 2014 report*. The 2021 NTP report did not comment on the potential impact of PCE on specific cancers, including NHL.

D. EPA's December 2020 Discussion of the Risk Evaluation for PCE

In December of 2020, the U.S. EPA published its risk evaluation for PCE.⁶⁴ In the evaluation, the EPA stated that PCE is considered "likely to be carcinogenic in humans" by all routes of exposure based on conclusive evidence in animals and suggestive evidence in humans. As with the 2014 IARC report, the EPA stated that there is conclusive animal evidence of the carcinogenicity of PCE, administered by ingestion or inhalation. With respect to the weight of the epidemiological evidence, EPA reviewed 38 studies in detail, including 18 cohort studies of 15 unique cohorts, 17 case-control studies of 14 unique population groups, and 3 ecological studies. Based on the review, the EPA stated that "There is a pattern of epidemiological evidence associating PCE exposure with

⁶⁴ US EPA. *Risk Evaluation for Perchloroethylene (Ethene, 1,1,2,2-Tetrachloro)*. Office of Chemical Safety and Pollution Prevention, U.S. Environmental Protection Agency, #740-R1-8011, December 2020.

NHL.”

With regards to the dose-response relationship for PCE and cancer, the EPA noted that no available human studies of cancer were found to be suitable for dose-response assessment. Thus, as an alternative, the EPA relied on rodent bioassays to construct a dose-response assessment based on the relevance to humans of the tumors reported in the rodent bioassays. Human cancer risks were estimated from the rodent dose-response data using the linear non-threshold model.

E. Additional Recent Literature That Has Appeared on the Evidence Surrounding the Potential of PCE to Cause Cancer, NHL

As far as I am aware and based upon my own search, apart from the studies reviewed by the EPA in its 2020 report (which I have independently assessed), there have been no epidemiological or mechanistic studies with new data published on the subject of PCE and cancer, including NHL. However, in 2022, Goodman et al.⁶⁵ published a systematic review of the evidence relating specifically to PCE and NHL. The authors focused on the studies that had been previously reviewed by the U.S. EPA in its 2020 Risk Report, given that no additional studies relevant to the potential for PCE to cause NHL had since been published. In their opinion, none of the studies were high quality in terms of all four quality elements considered to be critical, i.e., quality of study design, quality of exposure measurements, ascertainment of at least three exposure levels, and control of potential confounders. The authors also raised criticisms of other methodological study limitations of each study. They noted that the reported risk estimates were mostly null, ranged widely from below to above 1, and often had extremely wide confidence intervals (CIs), indicating unstable risk estimates. In addition, they were of the opinion that there was no consistent evidence of dose-response. Based on their interpretation of the studies, a brief review of the experimental and mechanistic data, and applying the Bradford Hill considerations, Goodman et al. concluded that the evidence does not support an association between PCE exposure and NHL. However, as discussed below, I disagree with their interpretation of the literature.

F. My Interpretation of the Epidemiological Evidence

In my opinion, the epidemiological evidence relevant to the relationship between PCE exposure and NHL is modest in volume and hampered by a number of limitations typical of this kind of research, such as the relatively small number of studies conducted that had the ability to focus specifically on PCE (v. exposures that included other solvents), the limited ability of the relevant studies to assess individual exposures with precision and accuracy, issues associated with comparison populations (e.g., the Health Worker bias that may result from comparing occupationally-exposed populations to the general population), and limited ability to control for confounders.

⁶⁵ Goodman JE, Ticknor RC, Zhou J. Systematic review of perchloroethylene and non-Hodgkin's lymphoma. *Glob Epidemiol.* 2022 Jun 16;4:100077. doi: 10.1016/j.gloepi.2022.100077. PMID: 37637029; PMCID: PMC10446115.

Nevertheless, in my opinion, the epidemiological evidence generally trends in favor of a finding that it is at least as likely as not that there is a positive, and indeed causal, relationship. As noted in the 2020 EPA report, the epidemiological evidence consists of 18 cohort studies of 15 unique cohorts, 17 case-control studies of 14 unique population groups, and 3 ecological studies. Among the evidence are five cohort studies that, in my opinion (as well as that of the 2020 EPA Report, but in contrast to Goodman et al. 2022) used a relatively high-quality exposure assessment methodology, i.e., Anttila et al. (1995),⁶⁶ Boice et al. (1999),⁶⁷ Radican et al. (2008),⁶⁸ Calvert et al., (2011),⁶⁹ and Seldén and Ahlborg (2011).⁷⁰ Boice et al. (1999) conducted a retrospective cohort mortality study of aircraft manufacturing workers. The SMRs for 40 cause of death categories were computed for the total cohort and for subgroups defined by sex, race, position in the factory, work duration, year of first employment, latency, and occupational groups. Factory job titles were classified as to likely use of chemicals, and internal Poisson regression analyses were used to compute mortality risk ratios for categories of years of exposure to chromate, TCE, PCE, and mixed solvents, with unexposed factory workers serving as referents. Of the 45,323 workers studied, 3,176 and 2,631 had intermittent or routine exposure specifically to PCE, respectively. The overall SMR for the association between exposure specific to PCE and NHL was 1.7 (95% CI: 0.73 to 3.34), with SMRs in relation to 0, 1-4, and ≥ 5 years of exposure of 1.00 (referent category; 32 cases), 1.11 (6 cases), and 1.41 (10 cases), respectively. Anttila et al. (1995) studied Standardized Incidence Rates (SIRs) of cancer among 3,974 Finnish workers exposed to halogenated hydrocarbons in which exposure levels to PCE had been specifically determined by repeatedly measured concentrations of PCE in blood for each worker over a 10-year period. The authors found overall increased SIRs for NHL of 1.1 (95% CI: 0.15 to 4.3) and 2.55 (95% CI: 1.17 to 4.84) in association with exposures that were 0–9 years and 10 years since first-measured exposure occurred, respectively. Radican et al. (2008) studied 14,455 workers at an aircraft maintenance facility, using information on exposures, chemicals, jobs, processes, worker compensation files, histories, and telephone books of the facility, organization charts, technical orders and position descriptions, walk through surveys and interviews of long term employees to derive exposure estimates for each job-organization combination. Using multivariable Cox models, they found that the association between exposure

⁶⁶ Anttila A, Pukkala E, Sallmén M, Hernberg S, Hemminki K. Cancer incidence among Finnish workers exposed to halogenated hydrocarbons. *J Occup Environ Med.* 1995 Jul;37(7):797-806. doi: 10.1097/00043764-199507000-00008. PMID: 7552463.

⁶⁷ Boice JD Jr, Marano DE, Fryzek JP, Sadler CJ, McLaughlin JK. Mortality among aircraft manufacturing workers. *Occup Environ Med.* 1999 Sep;56(9):581-97. doi: 10.1136/oem.56.9.581. PMID: 10615290; PMCID: PMC1757791.

⁶⁸ Radican L, Blair A, Stewart P, Wartenberg D. Mortality of aircraft maintenance workers exposed to trichloroethylene and other hydrocarbons and chemicals: extended follow-up. *J Occup Environ Med.* 2008 Nov;50(11):1306-19. doi: 10.1097/JOM.0b013e3181845f7f. PMID: 19001957; PMCID: PMC2763375.

⁶⁹ Calvert GM, Ruder AM, Petersen MR. Mortality and end-stage renal disease incidence among dry cleaning workers. *Occup Environ Med.* 2011 Oct;68(10):709-16. doi: 10.1136/oem.2010.060665. Epub 2010 Dec 16. PMID: 21172794.

⁷⁰ Seldén AI, Ahlborg G Jr. Cancer morbidity in Swedish dry-cleaners and laundry workers: historically prospective cohort study. *Int Arch Occup Environ Health.* 2011 Apr;84(4):435-43. doi: 10.1007/s00420-010-0582-7. Epub 2010 Oct 1. PMID: 20886350; PMCID: PMC3058547.

to PCE and NHL had a hazard ratio of 2.32 among males (95% CI: 0.75–7.15, 5 cases) and 2.35 (95% CI: 0.52–10.71; 2 cases). The association of PCE with all lymphatic/hematopoietic cancers had a hazard ratio of 1.92 among males (95% CI: 1.00–3.69). Calvert et al. (2011) studied the mortality experience of 1,704 dry cleaning workers, for whom PCE was the specific dominant chemical exposure. Standardized Mortality Ratios (SMR) in relation to the general population were calculated, demonstrating, for workers whose exposure was confined to PCE, an SMR for NHL of 2.46 (95% CI: 0.90 to 5.36, 6 cases). Given that SMR analyses using the generation population as the reference are subject to the Healthy Worker Effect (which applies to studies of cancer, including hematopoietic cancers) (Kirkeleit et al., 2013),⁷¹ the SMR in Calvert et al. (2011) is likely to be an underestimate. Selden and Ahlorg (2011) studied the cancer incidence experience of a Swedish national cohort of 10,389 dry-cleaning and laundry workers in relation to the Swedish general population, finding a significantly elevated Standardized Incidence Rate for NHL among the dry-cleaning workers of 2.05 (95% CI: 1.30–3.07; 23 cases in men). (The interpretation of this result was somewhat complicated by the finding of an elevated rate of NHL among the laundry workers, who were not known to have been exposed to PCE, as well).

In addition, in contrast to the opinion rendered by Goodman et al., there was evidence in Boice et al. (1999), Seidler et al. (2007),⁷² and Vlaanderen et al. (2013)⁷³ of exposure-response gradients with higher NHL risks observed in the higher or highest exposure categories based on PCE-specific exposure measures. As discussed earlier, this evidence was seen in the study by Boice et al. (1999) in terms of progressively higher SMRs in relation to years of exposure. Seidler et al. (2007) studied 710 cases of malignant lymphoma in relation to local general population controls. A structured personal interview was used to elicit a complete occupational history, including job task-specific information, which was used by a trained occupational physician (blinded to disease status) to assess levels of exposure to PCE and other hydrocarbons. For PCE, the adjusted Odds Ratio for the association of NHL with PCE exposure was flat over the lowest 3 categories of exposure, but then elevated at a rate of 3.4 (95% CI: 0.7–17.3; based on 6 cases) at the highest exposure category. Vlaanderen et al. (2013) studied national cancer registry data from four Nordic Countries, which included occupational information for exposure to PCE and other exposures based on country, time period, and job-specific exposures via a job-exposure matrix. They found higher hazard ratios for NHL that were significantly elevated among men and women combined (H: 1.23, 95% CI: 1.00 to 1.52), among men in association with increasing cumulative exposure (HR: 1.54, 95% CI: 0.99 to 2.42) and among men based on a metric of Intensity x Prevalence of exposure (HR: 1.74, 95% CI: 1.15 to 2.64).

⁷¹ Kirkeleit J, Riise T, Bjørge T, Christiani DC. The healthy worker effect in cancer incidence studies. *Am J Epidemiol.* 2013 Jun 1;177(11):1218-24. doi: 10.1093/aje/kws373. Epub 2013 Apr 17. PMID: 23595008.

⁷² Seidler A, Möhner M, Berger J, Mester B, Deeg E, Elsner G, Nieters A, Becker N. Solvent exposure and malignant lymphoma: a population-based case-control study in Germany. *J Occup Med Toxicol.* 2007 Apr 2;2:2. doi: 10.1186/1745-6673-2-2. PMID: 17407545; PMCID: PMC1851965.

⁷³ Vlaanderen J, Straif K, Pukkala E, Kauppinen T, Kyrrönen P, Martinsen JI, Kjaerheim K, Tryggvadottir L, Hansen J, Sparén P, Weiderpass E. Occupational exposure to trichloroethylene and perchloroethylene and the risk of lymphoma, liver, and kidney cancer in four Nordic countries. *Occup Environ Med.* 2013 Jun;70(6):393-401. doi: 10.1136/oemed-2012-101188. Epub 2013 Feb 27. PMID: 23447073.

Other studies relevant to the association of PCE with NHL include Lipworth et al. (2011),⁷⁴ which continued the follow-up of workers studied by Boice et al. (1999) and found a marginally significant increase in risk of death due to NHL among workers with routine or intermittent exposure to PCE (SMR = 1.43, 95% CI = 1.00–1.98) based on 36 observed cases and 25.1 expected. In a New York state cohort studied by (Silver et al., 2014),⁷⁵ the overall hourly male workers from the cohort showed a significant increase in mortality due to NHL (SMR = 1.49, 95% CI = 1.15–1.89, 65 observed). The HR for NHL risk for the measure of cumulative exposure to PCE relative to internal referents was 1.25 (95% CI = 0.90–1.73). Results from other newer studies were not informative, primarily due to small numbers of NHL cases with exposure to PCE (Bulka et al., 2016; Christensen et al., 2013; Morales-Suárez-Varela et al., 2013; Ruckart et al., 2013).

Overall, in my opinion, the weight of the epidemiologic evidence tilts toward there being at least as likely, if not more than likely, a causal relationship between PCE and NHL. That my opinion differs from that of Goodman et al. reflects the fact that even though “systematic reviews” aim at increasing the rigor of an overall analysis by applying a more consistent set of rules towards weighing the value of individual studies, judgment and appreciating nuances and patterns are still required in determining and applying those weights—a critique of Goodman et al. that was raised by Savitz (2022)⁷⁶ and published in tandem with Goodman et al.’s article. Thus, for example, in contrast to the opinions expressed by Goodman et al. (2022), as discussed above, my interpretation of the evidence aligns with that of the EPA in finding evidence of high-quality exposure assessments and dose-response relationships in a number of the studies, whereas Goodman et al. did not. Another issue with respect to Goodman et al. is internal consistency. For example, as noted by LaKind et al. (2022)⁷⁷ in another critique, even though Goodman et al. stated that none of the reviewed studies was of high or medium quality for exposure assessment and confounding, they then included all studies equally in their discussion of evidence synthesis. Even then, the evidence synthesis by Goodman et al. is flawed, an opinion shared by Weed (2022) in another critique that was published in tandem with Goodman et al. Weed noted that:

Goodman et al. did not, in fact, determine that all the epidemiological studies on PCE and NHL were of low quality based on their four-characteristic quality approach, and seven studies to be “50% high quality and 50% low quality.” The

⁷⁴ Lipworth L, Sonderman JS, Mumma MT, Tarone RE, Marano DE, Boice JD Jr, McLaughlin JK. Cancer mortality among aircraft manufacturing workers: an extended follow-up. J Occup Environ Med. 2011 Sep;53(9):992-1007. doi: 10.1097/JOM.0b013e31822e0940. PMID: 21866047.

⁷⁵ Silver SR, Pinkerton LE, Fleming DA, Jones JH, Allee S, Luo L, Bertke SJ. Retrospective cohort study of a microelectronics and business machine facility. Am J Ind Med. 2014 Apr;57(4):412-24. doi: 10.1002/ajim.22288. Epub 2013 Dec 24. PMID: 24375784; PMCID: PMC4548877.

⁷⁶ Savitz DA. Commentary on Methodologic choices in synthesizing epidemiologic evidence to assess perchloroethylene and non-Hodgkin's lymphoma. Glob Epidemiol. 2022 Oct 18;4:100089. doi: 10.1016/j.gloepi.2022.100089. PMID: 37637026; PMCID: PMC10445977.

⁷⁷ LaKind JS, Burns CJ, Mattison DR. Commentary: Systematic reviews and observational epidemiology: The more things change.... Glob Epidemiol. 2022 Oct 17;4:100088. doi: 10.1016/j.gloepi.2022.100088. PMID: 37637020; PMCID: PMC10446007.

results of these seven “50% high quality and 50% low quality” studies are shown in the table below:

Author, Year	RR Estimate	Exposure
Boice, 1999	1.41 (0.67–3.00)	>5 years
Bove, 2014a	1.14(0.56–2.32)	High Cumulative
Lipworth, 2011	1.02 (0.53–1.99)	>5 years
Lynge, 2006	0.66 (0.36–1.22)	>10 years
Morales, 2003	2.48 (0.52–11.90)	>Median Exposure
Ruckart, 2013	1.4 (0.3–5.6)	High Exposure
Seidler, 2007	3.2 (0.6–16.7)	> 78.8 ppm-years

In contrast to the Goodman et al. conclusion regarding this evidence taken as a whole, the somewhat better-quality studies reveal relative risk estimates above one at relatively high exposure levels (with one exception), albeit imprecise with confidence intervals that include 1.0. Had Goodman et al. performed exploratory meta-analyses with these studies it is entirely possible, even reasonable, that the results would have included an elevated risk with confidence interval that excludes 1.0. It follows that a conclusion that an association may exist between PCE and NHL seems reasonable at least at relatively high exposure levels. In the end, Goodman et al.’s claim of “no association” is as overstated as they believe the conclusions are from the three organizations involved.

I also note that confounding by life-style factors in these studies is an unlikely explanation for the observed results because common behaviors, such as smoking and alcohol use, are not strong risk factors for NHL.^{78,79} Moreover, there is little reason to suspect that PCE exposure in the studies reviewed above is associated with other identified risk factors for NHL that could have served as confounders, such as overweight/obesity, exposure to radiation, previous treatment with chemotherapy, weakened immune system, personal history of autoimmune diseases, family history of NHL or lymphohematopoietic cancer, and certain infections. No such associations make intuitive sense nor have they been reported in the literature, and without evidence of such associations, there is no reason to suspect that such factors can be confounders of the relationship between PCE and NHL, since, by definition, a confounder needs to be associated with *both* the exposure and the outcome of interest.

⁷⁸ Besson H, Brennan P, Becker N, Nieters A, De Sanjosé S, Font R, Maynadié M, Foretova L, Cocco PL, Staines A, Vornanen M, Boffetta P. Tobacco smoking, alcohol drinking and non-Hodgkin's lymphoma: A European multicenter case-control study (Epilymph). *Int J Cancer*. 2006 Aug 15;119(4):901-8. doi: 10.1002/ijc.21913. PMID: 16557575.

⁷⁹ Morton LM, Hartge P, Holford TR, Holly EA, Chiu BC, Vineis P, Stagnaro E, Willett EV, Franceschi S, La Vecchia C, Hughes AM, Cozen W, Davis S, Severson RK, Bernstein L, Mayne ST, Dee FR, Cerhan JR, Zheng T. Cigarette smoking and risk of non-Hodgkin lymphoma: a pooled analysis from the International Lymphoma Epidemiology Consortium (interlymph). *Cancer Epidemiol Biomarkers Prev*. 2005 Apr;14(4):925-33. doi: 10.1158/1055-9965.EPI-04-0693. PMID: 15824165.

Finally, Goodman et al. downplayed the evidence with regards to biological plausibility, stating that although there was experimental evidence for the genotoxicity of PCE metabolites, the evidence was specific to rodent liver and kidney cancers. Without evidence that demonstrates that PCE (or its metabolites) can contribute specifically to NHL, Goodman et al. opined that the evidence was weak, and they also stated that there is no evidence that supports any other biologically-plausible mode of action by which PCE could cause NHL. However, PCE has been shown to be genotoxic to peripheral blood lymphocytes in vitro, causing chromosomal aberrations and the formation of micronuclei in peripheral blood lymphocytes in vitro (Kocaman et al., 2021).⁸⁰ PCE exposure has also been found to be associated with primary DNA damage to leukocytes among workers exposed to PCE (from working as dry cleaners) in comparison to non-exposed workers (Azimi et al., 2017).⁸¹ Inhalation exposure to PCE has been shown to cause mononuclear-cell leukemia in rats of both sexes (NTP, 2015).⁸² Since leukemia and lymphoma involve cells that originate in the bone marrow, damage to circulating blood cells likely indicate effects on their precursors in the bone marrow. Altogether, in my opinion, these studies provide evidence supporting the biological plausibility of PCE causing NHL.

G. Bradford Hill Considerations

Having reviewed the evidence regarding the association between PCE and NHL, it is useful to assess this evidence according to the Bradford Hill factors.

1. Strength of association: In terms of strength of association, I take note of several of the epidemiological studies that, as discussed earlier, are of relatively high quality and that demonstrate increased risks of NHL in relation to PCE exposure. One such study is Antilla et al. (1995), which studied Finnish workers using repeated measures of PCE to provide relatively precise measures of PCE exposure, and which found a statistically significant elevated standardized incidence rate for NHL of 2.55 (based on 9 cases; 95% CI: 1.17 to 4.84) in association with exposures that were 10 years since first measured exposure occurred. Selden and Ahlorg's national cohort study of Swedish dry-cleaning workers found a statistically significant elevated Standardized Incidence Rate of NHL of 2.05 (based on 23 cases; 95% CI: 1.30–3.07; 23 cases) in men. Although Radican et al. did not have enough NHL cases to match the statistical power of the previous two studies, the hazard ratio point estimate for the association of PCE and NHL was 2.32 among males (based on 5 cases; 95% CI: 0.75 – 7.15); similarly, the 6 cases of NHL found among PCE-exposed dry cleaning workers by Calvert et al. (2011) conferred a Standardized Mortality Ratio of 2.46 (95% CI: 0.90 to 5.36). An Odds Ratio

⁸⁰ Kocaman AY, Asfuroğlu K. The genotoxic effects of perchloroethylene in human peripheral blood lymphocytes and the possible ameliorative role of α -tocopherol. *Environ Sci Pollut Res Int*. 2021 Aug;28(29):39576-39586. doi: 10.1007/s11356-021-13523-3. Epub 2021 Mar 24. PMID: 33763835.

⁸¹ Azimi M, Bahrami MR, Rezaei Hachesu V, Zavar Reza J, Mihanpour H, Zare Sakhvidi MJ, Mostaghaci M. Primary DNA Damage in Dry Cleaners with Perchloroethylene Exposure. *Int J Occup Environ Med*. 2017 Oct;8(4):224-231. doi: 10.15171/ijoem.2017.1089. PMID: 28970597; PMCID: PMC6679606.

⁸² National Toxicology Program. 15th Report on Carcinogens [Internet]. Research Triangle Park (NC): National Toxicology Program; 2021 Dec 21. Tetrachloroethylene: CAS No. 127-18-4. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK590938/>.

of 3.4 (95% CI: 0.7 to 17.3; based on 6 cases) was found in the case control study conducted by Seidler et al. (2017). Overall, I find this sufficient evidence to provide moderately strong evidence for strength of association.

2. Consistency: As noted in the discussion above, the higher-quality studies predominantly found elevated risk of NHL in association with PCE (especially at the higher/highest levels of exposure), whether or not they individually were statistically significant at the $p < 0.05$ level. These studies were conducted by different investigators in different populations spanning different countries (United States, Finland, Germany, Sweden, and the Nordic countries [Iceland, Norway, Sweden, Finland]). In my view, this meets the expectation for consistency.

3. Specificity: The epidemiological evidence discussed in this analysis is specific to the relationship between PCE and NHL and limited to those studies in which PCE exposure can be distinguished from other exposures and NHL could be distinguished from other hematological malignancies. Thus, in my opinion, the evidence in support of this criterion is strong.

4. Temporality: In the cohort studies that found a significant association between PCE exposure and NHL, including those discussed above, exposure preceded disease, providing evidence of temporality. Additionally, studies that have adjusted for a lag period in the onset of cancer (e.g., Antilla et al., 1995), found a stronger association between PCE and NHL, which aligns with the latency period one would expect between exposure and the onset of a cancer such as NHL. Thus, in my opinion, the evidence in support of this criterion is strong.

10. Biological Gradient: As noted earlier, there was evidence of an exposure-response gradient with respect to PCE exposure and NHL in Boice et al. (1999), Seidler et al. (2007), and Vlaanderen et al. (2013). In my opinion, the evidence in support of this criterion is modest, but present.

11. Biological Plausibility: As discussed earlier, there is both in vitro and evidence from human studies that PCE is genotoxic to peripheral blood lymphocytes, causing chromosomal aberrations and formation of micronuclei. PCE has also been shown to cause mononuclear-cell leukemia in rats, which indicates that the effects are also likely to bone marrow stem cells, which in turn, are the progenitor cells for lymphomas as well as leukemias. In my opinion, put together, the evidence provides modest support for biological plausibility.

12. Coherence: The correspondence between the epidemiological evidence and the experimental toxicological evidence, in my opinion, is consistent with coherence.

13. Experimental Evidence: Direct experimental epidemiological evidence is not available for human exposures to PCE (and would be unethical to accomplish). However, it is important to note that the toxicological evidence regarding PCE's genotoxicity are rooted in sound in vitro studies.

14. Analogy: There is similarity between the likely mechanisms of action of PCE with other genotoxic exposures known to induce lymphoma, e.g., benzene (see discussion elsewhere in

this report) and polycyclic aromatic hydrocarbons (Barnes et al., 2018),⁸³ which supports this viewpoint.

H. Conclusion: PCE Exposure Is At least as Likely as Not to Cause NHL

In conclusion, as with TCE, the epidemiological evidence of the association between PCE and NHL is relatively limited, due, in part, to the fact that NHL is a relatively rare disease and that other than history, there are no tools (e.g., biological markers) with which to assess historical and/or cumulative exposures to PCE; and even if histories are available, the ability to disentangle exposure to PCE from other solvents is typically limited. These factors account for the limited conclusions reached by the IARC and NTP reports with regards to PCE and NHL. On the other hand, in my opinion, as reflected by my discussion based on the Bradford Hill viewpoints, enough epidemiological and mechanistic evidence exists in the scientific literature to provide support for PCE as a cause of NHL sufficient to surpass the “at least as likely as not” standard in this case.

III. Benzene, Cancer, and NHL

My opinion that benzene is a human carcinogen and a carcinogen specifically for NHL is based on (1) review of the 2018 monograph published by the International Agency for Research on Cancer (IARC), as well as its underlying studies/sources, on its assessment of benzene’s carcinogenicity and benzene as a risk factor specifically for NHL that resulted from the October 10–17, 2017 meeting in Lyon, France, of the IARC Working Group on the Evaluation of Carcinogenic Risks to Human; (2) review of several epidemiological and toxicological studies published after the 2017 meeting of the IARC Working Group that assessed benzene’s carcinogenicity and as a risk factor specifically for NHL; (3) my assessment of the evidence in relation to the Bradford Hill considerations.

A. Background

Benzene is a colorless liquid with a sweet odor. It evaporates into air very quickly and dissolves slightly in water. It is found in air, water, and soil, and although there are some natural sources of benzene (e.g., volcanoes and forest fires), industrial processes, emissions from burning coal and oil, motor vehicle exhaust, evaporation from gasoline service stations (and leaking underground gasoline-storage tanks), and tobacco smoke are the main sources of benzene that appear in the environment.⁸⁴ Vapors from products that contain benzene, such as glues and paints, can also be a source of exposure. Benzene can pass into air from water and soil surfaces. Once in the air, benzene reacts with other chemicals and breaks down within a few days. But benzene in water and soil breaks down more slowly.

⁸³ Barnes JL, Zubair M, John K, Poirier MC, Martin FL. Carcinogens and DNA damage. *Biochem Soc Trans.* 2018 Oct 19;46(5):1213-1224. doi: 10.1042/BST20180519. Epub 2018 Oct 3. PMID: 30287511; PMCID: PMC6195640.

⁸⁴ ATSDR. *Toxicological Profile for Benzene*. Agency for Toxic Substances and Disease Registry. U.S. Centers for Disease Control and Prevention. August, 2007.

People are exposed to and can be affected by benzene due to its ability to enter the body through inhalation, ingestion, and skin (dermal) absorption. Benzene that contaminates a residential water system confers the opportunity for absorption via all 3 pathways. For example, studies of home exposures to benzene-contaminated water have demonstrated that in addition to being a source of exposure via ingestion, a typical 20-minute shower was associated with dermal exposure as well as the sustained volatilization of benzene into the air over the next hour.⁸⁵ In terms of toxicokinetics⁸⁶, benzene is readily absorbed following inhalation, oral, and dermal exposure (although benzene absorption from the skin is partly offset by the evaporation of benzene from the skin surface). Once absorbed, benzene is rapidly distributed throughout the body and tends to accumulate in fatty tissue. The liver plays an important role in benzene toxicity by metabolizing benzene, thereby producing conjugated metabolites that are excreted in urine, but also several reactive metabolites which are toxic, especially to the hematopoietic system. Benzene's carcinogenicity has been attributed to its metabolism to hydroquinone and then 1,4-benzoquinone.⁸⁷

Benzene exposure at high levels can cause injury to the lungs⁸ and possibly the heart and brain.⁸⁸ However, the primary target organ is the hematological system, both at high levels of exposure as well as low levels of exposure. Inhalational exposures exceeding an 8-hour time-weighted average (TWA) of 1 ppm for several months to several years is widely acknowledged to be capable of resulting in clinical pancytopenia, aplastic anemia and leukemia.^{77,89,90}

B. IARC's Assessment of Benzene, Risk of Cancer, and Specifically NHL

As noted earlier, the International Agency for Research on Cancer (IARC) is a specialized cancer agency of the World Health Organization (WHO). IARC promotes international and inter-disciplinary collaboration in cancer research, bringing together skills in epidemiology, laboratory sciences, and biostatistics to identify the causes of cancer so that preventive measures may be adopted and the burden of disease and associated suffering reduced (see Section I.B. for fuller description of IARC and IARC Monographs).

⁸⁵ Lindstrom AB, Highsmith VR, Buckley TJ, Pate WJ, Michael LC. Gasoline-contaminated ground water as a source of residential benzene exposure: a case study. *J Expo Anal Environ Epidemiol*. 1994 Apr-Jun;4(2):183-95. PMID: 7549473.

⁸⁶ ATSDR. *Toxicological Profile for Benzene*. Agency for Toxic Substances and Disease Registry. U.S. Centers for Disease Control and Prevention. August, 2007.

⁸⁷ Smith MT, Zhang L, Jeng M, Wang Y, Guo W, Duramad P, Hubbard AE, Hofstadler G, Holland NT. Hydroquinone, a benzene metabolite, increases the level of aneusomy of chromosomes 7 and 8 in human CD34-positive blood progenitor cells. *Carcinogenesis*. 2000 Aug;21(8):1485-90. PMID: 10910948.

⁸⁸ Hu J, Yu E, Liao Z. Changes in cognitive function and related brain regions in chronic benzene poisoning: a case report. *Ann Transl Med*. 2021 Jan;9(1):81. doi: 10.21037/atm-20-6597. PMID: 33553374; PMCID: PMC7859828.

⁸⁹ IARC. 1982. IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Vol. 29: Some industrial chemical and dyestuffs. Benzene. Lyon, France: World Health Organization, International Agency for Research on Cancer, 93-148.

⁹⁰ IRIS. 2007. Benzene. Integrated Risk Information System. Washington, DC: U.S. Environmental Protection Agency. <http://www.epa.gov/iris/subst/index.html>. May 1, 2007.

In 2018, based on the deliberations of a working group of experts that met from October 10–17, 2017, IARC published a monograph (Volume 120) that addressed the carcinogenicity of benzene in which it concluded that benzene, in general, is a category I (i.e., “proven”) carcinogen.⁹¹ In its summary of the evidence with regards specifically to NHL, IARC stated the following in the main text and associated footnote (pp. 291–292):

Overall, the Working Group concluded that the evidence of carcinogenicity for NHL is limited; however, a small minority of the Working Group concluded that the evidence of carcinogenicity is sufficient for NHL... The meta-analysis of five incidence studies revealed a statistically significant association between benzene exposure and chronic lymphocytic leukemia (CLL⁹²; pooled relative risk estimate, 1.53; 95% CI, 1.04–2.25). The relative risk estimates of four of these five studies were above 1.6; only one study found a slightly lower risk estimate of 1.40 (95% CI, 0.90–2.19). Therefore, the CLL risk is comparable to the AML (acute myelogenous leukemia) risk (pooled risk estimate, 1.45; 95% CI, 0.96–2.17 when combining the six incidence studies). Chance and confounding can be ruled out with reasonable confidence. Most studies show a positive association between benzene exposure and NHL incidence. Among the incidence studies, there are three high-quality cohort studies, which reveal clearly increased NHL risks: one found a relative risk of 3.90 (95% CI, 1.31–11.57) for ever versus never exposure to benzene (not including CLL and MM), another reported a hazard ratio of 1.86 (95% CI, 1.17–2.96) including MM but not CLL, and a third reported a hazard ratio of 1.49 (95% CI, 0.90–2.47) for the whole NHL group (including MM and CLL). Most older studies, as well as some newer studies, did not include MM and CLL in the NHL group. However, the inclusion of CLL and MM in the NHL entity in these studies should not have changed the finding of a clear association between benzene and NHL, for the following two reasons. First, studies allowing for a direct comparison between MM and the (remaining or total) NHL group give no overall indication for lower MM risks compared with NHL risks as a whole. Second, as noted above, CLL risks are also clearly elevated.

In addition, with respect to mechanistic evidence, IARC concluded that the evidence, including the evidence in humans, is strong that benzene: is metabolically activated to electrophilic metabolites; induces oxidative stress and associated oxidative DNA damage; is genotoxic, inducing DNA damage and chromosomal changes; is immunosuppressive; and causes hematotoxicity.

I have independently reviewed and assessed the studies/sources underlying the IARC Monograph.

⁹¹ IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Benzene. Lyon (FR): International Agency for Research on Cancer; 2018. PMID: 31769947.

⁹² Considered by the IARC Working Group as a subgroup of NHL).

C. Literature That Has Appeared Since the IARC Reports on the Evidence Surrounding the Potential of Benzene to Cause NHL

1. Epidemiological Evidence

As discussed earlier (in Section I.D.), systematic reviews and meta-analyses are of particular relevance to the present task as they use a rigorous methodology for aggregating and summarizing evidence that accounts for and transcends the limitations of individual studies, and that results in quantitative estimates of the average effect across studies and the effect's statistical significance and variability.

With respect to the relationship between benzene exposure and NHL, two recent systematic reviews with meta-analyses stand out that appeared after the 2018 IARC monograph on benzene. Rana et al. (2021)⁹³ conducted one such study that used criteria to specifically focus on the relationship between benzene and NHL, excluding studies that grouped benzene with other solvents, or that combined NHL with other cancer types. Their screening resulted in a meta-analysis that included 20 case-control studies and 8 cohort studies, with a total of 9,587 patients with NHL. Overall, they found that high exposure to benzene was associated with a significantly increased meta-relative risk of 1.33 (95% CI: 1.13–1.57). The meta-RR rose to 1.51 (1.22–1.87) in studies that provided results specifically for highly exposed individuals. They also found an elevated risk for diffuse large B-cell lymphoma (a major NHL subtype as noted earlier), with an RR of 1.67 (1.01–2.77) and increased risks for follicular lymphoma (1.47 [0.95–2.27]) and hairy cell leukaemia (1.77 [0.99–3.16]), though they were not statistically significant. Funnel plot, Egger's test ($p=0.77$), and Begg's test ($p=0.98$) did not show evidence of publication bias. In the paper's discussion section, the authors noted in contrast to previous meta-analyses that did not find a statistically significant association between benzene and NHL, their paper included studies covering a longer period of follow-up (1984–2015; the other studies had follow-up only until 2009). In addition, the authors noted that, in contrast to the IARC review conducted in 2017 (and published in 2018), their search of the literature was more systematic and thorough, thereby identifying additional studies for evaluation not included in the IARC analysis; moreover, the meta-analysis conducted by IARC was confined to CLL (not NHL, generally).

Following Rana et al., another systematic review and meta-analysis was published by Liu and Wang (2022).⁹⁴ Using an approach similar to that of Rana et al. (2021) but with some differences in methods and application, the authors selected for the meta-analysis 5 case control studies and 9 cohort studies that included 1,994 cases of NHL. The Odds Ratio (OR) of NHL in the benzene-exposed population compared with nonexposed population was 1.23 ($P=0.03$; 95% CI: 1.01–1.51), with moderate heterogeneity ($I^2=63.47\%$). People with high exposure to benzene showed a higher risk of NHL (OR =1.81; 95% CI: 1.34–2.43), and there was a low degree of

⁹³ Rana I, Dahlberg S, Steinmaus C, Zhang L. Benzene exposure and non-Hodgkin lymphoma: a systematic review and meta-analysis of human studies. *Lancet Planet Health*. 2021 Sep;5(9):e633-e643. doi: 10.1016/S2542-5196(21)00149-2. Epub 2021 Aug 25. PMID: 34450064; PMCID: PMC9109598.

⁹⁴ Liu Y, Wang J. Benzene exposure increases the risk of non-Hodgkin's lymphoma: a systematic review and meta-analysis of observational studies. *Transl Cancer Res*. 2022 Jun;11(6):1750-1761. doi: 10.21037/tcr-22-1434. PMID: 35836544; PMCID: PMC9273682.

heterogeneity ($I^2=27.56\%$). Of interest to these authors, since they were based in China, is that the risk of benzene exposure in China (OR =2.48; 95% CI: 1.24–4.94) was higher than that in Europe (OR =1.19; 95% CI: 0.89–1.59), the United Kingdom (OR =1.07; 95% CI: 0.92–1.26), and the United States (OR =1.24; 95% CI: 0.80–1.92) .

2. Mechanistic Evidence

Subsequent to the 2018 IARC report on benzene, Guo et al. (2020)⁹⁵ published a systematic review of the evidence on the association of benzene with chronic inflammatory and immunosuppressive outcomes which included several studies published in 2018 or later (i.e., after the 2017 meeting that led to the 2018 IARC report). In the review, the authors found it evident that benzene induces immunosuppressive effects on the adaptive immune system and activation of the innate immune system to cause inflammation. In particular, benzene significantly lowers the number of white blood cells, particularly lymphocytes such as CD4+ T-cells, B-cells and natural killer cells, and increases proinflammatory biomarkers at low levels of exposure. These effects are pathways known to be relevant to the causation of lymphoma.

D. Bradford Hill Factor Analysis

Having reviewed the evidence regarding the association between benzene and NHL, it is useful to assess this evidence according to the Bradford Hill considerations.

- 1. Strength of association:** In terms of strength of association, in addition to the meta-analyses discussed above that demonstrate a significant association between benzene and NHL, several of the individual epidemiological studies provide evidence of a strong association between benzene exposure and NHL. Among them are a cohort study by Wong et al., (1987)⁹⁶ that found a Relative Risk of 4.12 (95% CI: 1.11–10.55); a cohort study by Hayes et al., (1996)⁹⁷ that found a Relative Risk of 4.70 (95%CI: 1.20–18.10); a case-control study by Fabbro-Peray et al. (2001)⁹⁸ that found an Odds Ratio of 5.70 (95% CI: 1.40–23.20); and case-control study by Xu et al. (2003)⁹⁹ that found an Odds Ratio of 2.78 (95% CI: 1.68–

⁹⁵ Guo H, Ahn S, Zhang L. Benzene-associated immunosuppression and chronic inflammation in humans: a systematic review. *Occup Environ Med.* 2020 Sep 16:oemed-2020-106517. doi: 10.1136/oemed-2020-106517. Epub ahead of print. PMID: 32938756; PMCID: PMC7960562.

⁹⁶ Wong O. An industry wide mortality study of chemical workers occupationally exposed to benzene. II. Dose response analyses. *Br J Ind Med.* 1987 Jun;44(6):382-95. doi: 10.1136/oem.44.6.382. Erratum in: *Br J Ind Med* 1987 Nov;44(11):776. PMID: 3606967; PMCID: PMC1007839.

⁹⁷ Hayes RB, Yin SN, Dosemeci M, Li GL, Wacholder S, Chow WH, Rothman N, Wang YZ, Dai TR, Chao XJ, Jiang ZL, Ye PZ, Zhao HB, Kou QR, Zhang WY, Meng JF, Zho JS, Lin XF, Ding CY, Li CY, Zhang ZN, Li DG, Travis LB, Blot WJ, Linet MS. Mortality among benzene-exposed workers in China. *Environ Health Perspect.* 1996 Dec;104 Suppl 6(Suppl 6):1349-52. doi: 10.1289/ehp.961041349. PMID: 9118919; PMCID: PMC1469764.

⁹⁸ Fabbro-Peray P, Daures JP, Rossi JF. Environmental risk factors for non-Hodgkin's lymphoma: a population-based case-control study in Languedoc-Roussillon, France. *Cancer Causes Control.* 2001 Apr;12(3):201-12. doi: 10.1023/a:1011274922701. PMID: 11405325.

⁹⁹ Xu CG, Zheng SP, Huang J, Wu JX. [A case-control study for assessing the relation between the

14.32). Thus, in my opinion, the evidence in support of this criterion is strong.

2. **Consistency:** As can be seen in the summary provided by Rana et al. (2021; Table 1) of cohort and case-control studies of benzene in relation to NHL by location, the epidemiology demonstrates consistency across the United States, Canada, several countries in Europe, Australia, and China based on different population samples, and different investigators. Thus, in my opinion, the evidence in support of this criterion is strong.
3. **Specificity:** The epidemiological evidence discussed in this analysis is specific to the relationship of benzene and NHL and limited to those studies in which benzene exposure can be distinguished from other exposures and NHL could be distinguished from other hematological malignancies. Thus, in my opinion, the evidence in support of this criterion is strong.
4. **Temporality:** In the cohort studies that found a significant association between benzene exposure and NHL, including those discussed above and reviewed in the meta-analyses discussed above, exposure preceded disease, providing evidence of temporality. Additionally, studies that have adjusted for a lag period in the onset of cancer have found a stronger association between benzene and NHL, which aligns with the latency period one would expect between exposure and the onset of a cancer such as NHL. Thus, in my opinion, the evidence in support of this criterion is strong.
5. **Biological Gradient:** In the systematic review and meta-analysis conducted by Rana et al. (2021) discussed earlier, in the sensitivity analysis that was conducted, the authors detected a 34% increased meta-Relative Risk of NHL when comparing studies that considered benzene in terms of an ever exposure group to studies that considered benzene in terms of high exposure groups (the meta-RR increased from 1.17 to 1.51)¹⁰⁰. This is consistent with demonstration of a dose-response relationship, i.e., biological gradient. In my opinion, the evidence in support of this criterion is present.
6. **Biological Plausibility:** The mechanistic, experimental and animal evidence are supportive based on evidence of benzene being metabolically activated to electrophilic metabolites; and that benzene induces oxidative stress and associated oxidative DNA damage; is genotoxic, inducing DNA damage and chromosomal changes; is immunosuppressive and promotes inflammation; and causes hematotoxicity. Thus, in my opinion, the evidence in support of this criterion is strong.
7. **Coherence:** The correspondence between the epidemiological evidence and the experimental

incidence of malignant lymphomas and environmental factors in Sichuan province]. *Zhonghua Liu Xing Bing Xue Za Zhi*. 2003 Oct;24(10):875-8. Chinese. PMID: 14575598.

¹⁰⁰ Rana I, Dahlberg S, Steinmaus C, Zhang L. Benzene exposure and non-Hodgkin lymphoma: a systematic review and meta-analysis of human studies. *Lancet Planet Health*. 2021 Sep;5(9):e633-e643. doi: 10.1016/S2542-5196(21)00149-2. Epub 2021 Aug 25. PMID: 34450064; PMCID: PMC9109598. (Discussion section, and Figure 3).

toxicological evidence noted by IARC is consistent with coherence.

8. **Experimental Evidence:** Experimental epidemiological evidence is not available for human exposures and benzene (and would be unethical to accomplish). However, it is important to note that the toxicological evidence regarding benzene's genotoxic and immunotoxic mechanisms are rooted in numerous sound experimental studies.
9. **Analogy:** There is similarity between the likely mechanisms of action of benzene with other immunotoxic agents known to induce lymphoma, e.g., azathioprine given for inflammatory bowel disease (Pasternak et al., 2013), which supports this viewpoint.

E. Conclusion: Benzene Exposure More Likely Than Not Causes NHL

In conclusion, as is true for the relationship between TCE and NHL and PCE and NHL that is discussed above, the epidemiological evidence of the association between benzene and NHL is relatively limited, due, in part, to the fact that NHL is a relatively rare disease and that other than history; there are no tools (e.g., biological markers) with which to assess historical and/or cumulative exposures to benzene; and even if histories are available, the ability to disentangle exposure to benzene from other solvents is typically limited. Nevertheless, the epidemiological evidence that is available, together with (a) the recent systematic review and meta-analyses conducted by Rana et al. (2021) and Liu and Wang (2022), (b) the mechanistic studies of benzene's genotoxicity, immunotoxicity, inflammatory effects, and chromosomal aberrations that are consistent with carcinogenesis; and (c) the analysis of the assembled evidence using the Bradford Hill framework, in my opinion, makes it more likely than not that benzene is a cause of NHL. As stated above, this means that the evidence for a causal relationship between benzene exposure and NHL exceeds the "at least as likely as not" standard prescribed by the Camp Lejeune Justice Act.

IV. Dose-Response Relationships at Low Levels of Exposure and Camp Lejeune

An issue that is common to each of the exposures discussed above (TCE, PCE, Benzene) is the extent to which they may pose risks of cancer at low levels of exposure. One of the long-running challenges to this topic is the lack of data that can be used to establish the risks posed by most carcinogens at low levels of exposure, given that epidemiological studies have limited power to detect such phenomena and experimental studies of animals have similar limitations, since the vast majority are conducted on sample sizes of 50 or fewer animals. The orthodox view for many years has been that for carcinogens that cause cancer by damaging DNA (such as a DNA-adduct mutation), there is no threshold of exposure that can be considered completely safe since even a single such exposure can theoretically initiate the carcinogenesis process (Hogberg and Järnberg, 2023).¹⁰¹ One result is that in order to derive a quantitative assessment of risk for exposure to carcinogens at levels of exposure below that which could be directly observed from either human or animal studies, it has been customary to assume a linear no-threshold (LNT)

¹⁰¹ Högberg J, Järnberg J. Approaches for the setting of occupational exposure limits (OELs) for carcinogens. *Crit Rev Toxicol*. 2023 Dec;53(3):131-167. doi: 10.1080/10408444.2023.2218887. Epub 2023 Jun 27. PMID: 37366107.

dose-response relationship between an exposure level of zero (i.e., no exposure) and the lowest observable adverse level (LOAEL) detected in either human or animal studies of a particular chemical's carcinogenesis.

This view has been challenged in on-going debates¹⁰². This is particularly true with respect to radiation exposure. For example, in radiation studies, very low doses (e.g., between 3-50 mSv) have been shown to result primarily in error-free DNA repair and elimination of aberrant cells. Cellular responses to damage, including repair and apoptosis, vary with dose and dose rate, suggesting non-linear relationships at low doses.

Nevertheless, based on the totality of the latest scientific evidence relevant to low-dose radiation exposures, including, for example, a systematic review of 26 epidemiological studies and associated meta-analysis¹⁰³ published in 2020, the LNT model remains in place and is considered reasonable and the best available approach for modeling the quantitative risk assessment for carcinogenesis that occurs through DNA damage/mutagenesis modes of action, as is the case with radiation. This is the position taken by the U.S. Environmental Protection Agency in 2015¹⁰⁴, the U.S. Nuclear Regulatory Commission¹⁰⁵ in 2021, and independent scientists in Europe¹⁰⁶ as recently as 2023. I agree with this position.

Apart from radiation, there is epidemiological evidence that is relevant specifically to the association of low-level chemical exposures with cancer that reflects the situation represented by Camp LeJeune. For example, a population-based case-control study of cancer in relation to exposure to PCE from contaminated drinking water in Massachusetts found an adjusted Odds Ratio (OR) of 5.84 (95% CI: 1.37-24.91) among those subjects whose exposure level (expressed as estimated total mass of PCE in milligrams that entered a given house as a solute in drinking water over the time period of observation) was over the 90th percentile (27.1 mg). Taking into

¹⁰² Weber W, Zanzonico P. The Controversial Linear No-Threshold Model. J Nucl Med. 2017 Jan;58(1):7-8. doi: 10.2967/jnumed.116.182667. Epub 2016 Oct 6. PMID: 27754908.

¹⁰³ Hauptmann M, Daniels RD, Cardis E, Cullings HM, Kendall G, Laurier D, Linet MS, Little MP, Lubin JH, Preston DL, Richardson DB, Stram DO, Thierry-Chef I, Schubauer-Berigan MK, Gilbert ES, Berrington de Gonzalez A. Epidemiological Studies of Low-Dose Ionizing Radiation and Cancer: Summary Bias Assessment and Meta-Analysis. J Natl Cancer Inst Monogr. 2020 Jul 1;2020(56):188-200. doi: 10.1093/jncimonographs/lgaa010. Erratum in: J Natl Cancer Inst Monogr. 2023 May 4;2023(61):e1. doi: 10.1093/jncimonographs/lgac027. PMID: 32657347; PMCID: PMC8454205.

¹⁰⁴ U.S. EPA. Comment to the Secretary of the U.S. Nuclear Regulatory Commission on the petitions for rulemaking filed with the U.S. NRC concerning Linear No-Threshold Model and Standards for Protection against Radiation. Docket ID NRC-2015-0057. Available at: <https://www.regulations.gov/document/NRC-2015-0057-0436>; accessed on December 2, 2024.

¹⁰⁵ NRC 2021 Linear no-threshold model and standards for protection against radiation. A proposed rule by the Nuclear Regulatory Commission on 08/17/2021 Federal Register vol 86. Available at: www.federalregister.gov/documents/2021/08/17/2021-17475/linear-no-threshold-model-and-standards-for-protection-against-radiation; accessed on December 2, 2024.

¹⁰⁶ Laurier D, Billarand Y, Klovov D, Leuraud K. The scientific basis for the use of the linear no-threshold (LNT) model at low doses and dose rates in radiological protection. J Radiol Prot. 2023 Jun 29;43(2). doi: 10.1088/1361-6498/acdfd7. PMID: 37339605.

account 5 years of latency, the adjusted OR was 8.33 (95% CI: 1.53-45.29) among those subjects whose exposure level was over the 90th percentile (44.1 mg). As another example, in an ecological study involving 75 towns in New Jersey¹⁰⁷, a comparison of towns with the highest TCE exposures (>5 µg/L) v. towns without detectable TCE demonstrated an age-adjusted rate ratio for NHL of 1.36 (95% CI: 1.08-1.70).

Finally, the series of epidemiological studies conducted by Bove and colleagues of Camp Lejeune itself provides direct evidence addressing the low-level chemical exposures of Camp Lejeune residents to TCE, PCE, and benzene and associations with risk of cancer. As noted in the most recent iteration of the study that was peer-reviewed and published in 2024, the drinking water served by Camp Lejeune's Tarawa Terrace treatment plant was contaminated by an off-base dry cleaner, with PCE concentrations in water samples measured between 1980 and 1985 having a maximum concentration of 215 µg/L. In addition, the drinking water served by Camp Lejeune's Hadnot Point treatment plant was contaminated by on-base sources of solvents, with TCE and PCE concentrations in water samples having a maximum concentration of 1,400 µg/L and 100 µg/L, respectively, along with benzene. Historical dose reconstruction modeling by the Agency for Toxic Substances and Disease Registry (ATSDR) found that contamination of drinking water had begun in the mid-1950's and largely terminated in 1985 when the contaminated supply wells were shut down.

As in their earlier studies of Camp Lejeune, the authors of the 2024 study sought to determine if being stationed or employed at Camp Lejeune between 1975 and 95 (Marines/Navy personnel) or between October 1972 and December 1985 (civilian workers) increased the risk of specific causes of death in comparison to being stationed or employed at Camp Pendleton, with the analysis examining data on specific causes of death beginning in 1979 and, in this most recent analysis, updated to 2018. The authors calculated standardized mortality ratios (which, by comparing death rates with the general population, were acknowledged to be susceptible to the Healthy Worker effect bias). In addition, the authors compared the death rates of individuals who had been at Camp Lejeune (n=159,128 Marines/Navy personnel, n=7,332 civilian workers) with individuals who had been at Camp Pendleton (n=168,406 Marines/Navy personnel, n=6,677 civilian workers), which conferred the epidemiological design benefit of comparing populations that differed in terms of exposures (the drinking water at Camp Pendleton had not been contaminated) but were similar in terms of demographic and socioeconomic factors, pre-enlistment screening and fitness requirements, training activities, and types of military and civilian employee occupations, which minimized influence in relation to biases and unmeasured confounders (thereby minimizing the Healthy Worker effect bias). Various strategies were employed to examine for and control for any residual biases and confounding.

In summary, based on proportional hazards regression models, the investigators found that compared to the general population, Camp Lejeune Marines/Navy personnel had hazard ratios adjusted for sex, race, and five-year age groups that were about the same for all cause

¹⁰⁷ Cohn P, Klotz J, Bove F, Berkowitz M, Fagliano J. Drinking Water Contamination and the Incidence of Leukemia and Non-Hodgkin's Lymphoma. *Environ Health Perspect.* 1994 Jun;102(6-7):556-61. doi: 10.1289/ehp.94102556. PMID: 9679115; PMCID: PMC1569761.

mortality (0.99; 95% CI: 0.97, 1.01), but elevated for all malignant cancers (1.07; 95% CI: 1.02, 1.12). In the comparisons between Camp Lejeune v. Camp Pendleton Marines/Navy personnel, the adjusted hazard ratio for all causes was about the same (0.99; 95% CI: 0.97, 1.01), with an adjusted hazard for all cancer malignancies elevated at 1.06 (95% CI: 1.0, 1.11).

Although almost none of the elevated risk ratios associated with specific cancers were statistically significant, the risk for cancers associated with Camp Lejeune exposures is likely to increase over time, since, as the authors pointed out in the discussion section of their paper, almost all of the Marines/Navy personnel (who comprise 96% of the Camp Lejeune study population) was aged <65 years by the end of follow-up, with many more cancers likely to develop in the ensuing years. Nevertheless, in terms of overall risk, the statistically elevated risks for cancer among the Camp Lejeune Marines/Navy personnel, observed both in relation to the general population (expressed as the standardized mortality ratio) as well as in relation to the Camp Pendleton Marines/Navy personnel (expressed as the hazard ratio), provides support for the general proposition that low-level community exposures to chemicals such as TCE, PCE, and benzene, constitute a risk for cancer causation.

V. The Combined Effect of Exposure to TCE, PCE, Benzene, and NHL

As far as I'm aware, there are no epidemiological, toxicological, or mechanistic studies that specifically assess the risk of NHL in individuals who are exposed to both TCE and benzene (i.e., together, but not in mixtures with other chemicals). When considering the carcinogenic potential of simultaneous exposure to two known carcinogens, one may anticipate that the two carcinogens may increase risk of cancer in an additive fashion – which is typically the default assumption when regulators assess chemicals that act through a common mode of action.¹⁰⁸ It is theoretically possible, as has been demonstrated by the example of asbestos and smoking in the causation of lung cancer,^{109,110} that two chemicals may interact in some way to increase the risk of NHL beyond what would be expected by summing the individual risks (i.e., a supra-additive or multiplicative risk, also known as “synergy”); on the other hand, it is also possible that two chemicals may interact in some way that results in an overall risk of NHL that is less than the sum of the individual risks, perhaps by mutual interference.

The lack of research and peer-reviewed studies that provides substantive evidence regarding the risks posed by “mixtures” of carcinogens has been acknowledged for a long time,

¹⁰⁸ Goodson WH 3rd, Lowe L, Carpenter DO, et al. Assessing the carcinogenic potential of low-dose exposures to chemical mixtures in the environment: the challenge ahead. *Carcinogenesis*. 2015 Jun;36 Suppl 1(Suppl 1):S254-96. doi: 10.1093/carcin/bgv039. Erratum in: *Carcinogenesis*. 2016 Mar;37(3):344. PMID: 26106142; PMCID: PMC4480130.

¹⁰⁹ Markowitz SB, Levin SM, Miller A, Morabia A. Asbestos, asbestosis, smoking, and lung cancer. New findings from the North American insulator cohort. *Am J Respir Crit Care Med*. 2013 Jul 1;188(1):90-6. doi: 10.1164/rccm.201302-0257OC. PMID: 23590275.

¹¹⁰ Klebe S, Leigh J, Henderson DW, Nurminen M. Asbestos, Smoking and Lung Cancer: An Update. *Int J Environ Res Public Health*. 2019 Dec 30;17(1):258. doi: 10.3390/ijerph17010258. PMID: 31905913; PMCID: PMC6982078.

with recent initiatives begun to advance a common framework for research in the laboratory¹¹¹ as well as in epidemiological studies.¹¹²

In the case of TCE, PCE, and benzene, given the lack of direct evidence of their combined effects, what can be appreciated based on chemistry and mechanism of action, first, is that there is no reason to anticipate that the combination would somehow result in a reduced risk of cancer based on mutual interference. Second, as discussed above, the mechanisms of action by which both chemicals are likely to cause cancer have substantial overlap. All three chemicals, including their metabolites (products of their metabolism in the human body), have been shown to be genotoxic (causing damage to DNA) and/or to cause chromosomal aberrations (causing DNA chromosomes to malform). Both DNA damage and chromosomal aberrations are well known risk factors for cancers. Both TCE and benzene are also known to be immunosuppressive (i.e., they suppress the immune system, the function of which includes surveillance for and destroying cancerous cells). Having a weakened immune system is well known to be a risk factor for cancer, including, specifically, NHL. As such, in my opinion, it is reasonable to apply the regulatory approach to carcinogens with a common mode of action (as discussed above) and conclude that the combined risk of simultaneous exposure to TCE, PCE, and benzene is more likely than not to be at least additive.

This concludes my evaluation of general causation with respect to exposure to TCE, PCE, benzene, and NHL. I reserve the right to update or amend the opinions contained herein based on new or additional evidence not currently available.

Respectfully,



Howard Hu, M.D., M.P.H., Sc.D.

December 9, 2024

¹¹¹ Rider CV, McHale CM, Webster TF, Lowe L, Goodson WH 3rd, La Merrill MA, Rice G, Zeise L, Zhang L, Smith MT. Using the Key Characteristics of Carcinogens to Develop Research on Chemical Mixtures and Cancer. *Environ Health Perspect.* 2021 Mar;129(3):35003. doi: 10.1289/EHP8525. Epub 2021 Mar 30. PMID: 33784186; PMCID: PMC8009606.

¹¹² Savitz DA, Hattersley AM. Evaluating Chemical Mixtures in Epidemiological Studies to Inform Regulatory Decisions. *Environ Health Perspect.* 2023 Apr;131(4):45001. doi: 10.1289/EHP11899. Epub 2023 Apr 6. PMID: 37022726; PMCID: PMC10078806.

Appendix A

CURRICULUM VITAE

Updated on November 24, 2024

NAME: Howard Hu
 PRIMARY AFFILIATION: Keck School of Medicine, University of Southern California
 SECONDARY AFFILIATIONS: School of Public Health, University of Michigan
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CV: Howard Hu, M.D., M.P.H., Sc.D.

EDUCATION:

9/1973-6/1976	Biology	B.Sc.	Brown University
9/1977-6/1982	Medicine	M.D.	Albert Einstein College of Medicine
9/1979-6/1980 (degree in 6/1982*)		M.P.H. (Occ Hlth)	Harvard School of Public Health
9/1985-6/1986	Epidemiology	M.S.	Harvard School of Public Health
7/1986-6/1990	Epidemiology	Sc.D.	Harvard School of Public Health

* Awarding of the Harvard M.P.H. to medical students is delayed until the M.D. degree is conferred

POSTDOCTORAL TRAINING:

Research Fellowships

7/1987-6/1988 Occupational Health Research Fellow, Dept. of Environmental Health
Harvard School of Public Health

Internship and Residencies

7/1982-6/1983	Intern in Medicine	Boston City Hospital
7/1983-6/1984	Junior Assistant Resident, Internal Medicine	Boston City Hospital
7/1984-6/1985	Senior Assistant Resident, Internal Medicine	Boston City Hospital
7/1985-6/1987	Resident, Occupational Medicine	Harvard School of Public Health

CERTIFICATION AND LICENSURE:

1984	Massachusetts Medical License Registration
1985	American Board of Internal Medicine, Diplomate
1987	American Board of Preventive Medicine, Diplomate (Occupational Medicine)
2006	Michigan Medical License Registration
2013	College of Physicians & Surgeons of Ontario
2018	Washington State Medical License Registration
2021	California State Medical License Registration

ACADEMIC APPOINTMENTS:

9/1988-6/1992	Instructor in Medicine Department of Medicine, Harvard Medical School
9/1988-6/2006	Associate Physician (Clinical and Research), Channing Laboratory, Department of Medicine, Brigham & Women's Hospital
9/1990-6/1994	Assistant Professor of Occupational Medicine Department of Environmental Health, Harvard School of Public Health
7/1992-6/1997	Assistant Professor of Medicine

CV: Howard Hu, M.D., M.P.H., Sc.D.

Department of Medicine, Harvard Medical School
 7/1994-6/2002 Associate Professor of Occupational Medicine
 Department of Environmental Health, Harvard School of Public Health
 7/1997-8/2006 Associate Professor of Medicine
 Department of Medicine, Harvard Medical School
 7/2002-8/2006 Professor of Occupational and Environmental Medicine (tenured)
 Department of Environmental Health, Harvard School of Public Health
 9/2006-6/2012 Chair and Professor of Environmental Health Sciences (tenured), Department of
 Environmental Health Sciences, University of Michigan School of Public Health
 9/2006-8/2009 Adjunct Professor of Occupational and Environmental Medicine
 Department of Environmental Health, Harvard School of Public Health
 9/2006-6/2012 Research Associate Physician, Channing Laboratory, Department of
 Medicine, Brigham & Women's Hospital
 5/2007-2012 Professor of Epidemiology, University of Michigan School of Public Health
 5/2007-2012 Professor of Internal Medicine, University of Michigan Medical School
 1/2009-2012 NSF International Endowed Department Chair, University of Michigan School of
 Public Health, Department of Environmental Health Sciences
 7/2012-2018 Professor of Environmental Health, Epidemiology and Global Health (tenured)
 Dalla Lana School of Public Health, University of Toronto, Toronto, Ontario,
 Canada (on sabbatical/administrative leave, 2017-2018)
 7/2012-2018 Professor, School of Medicine, University of Toronto, Toronto, Ontario, Canada
 7/2012- Adjunct Professor, Department of Environmental Health Sciences, University of
 Michigan School of Public Health
 7/2012-2013 Director, Dalla Lana School of Public Health, University of Toronto, Toronto,
 Ontario, Canada
 7/2013-6/2018 Founding Dean, Dalla Lana School of Public Health, a Faculty of the University
 of Toronto, Toronto, Ontario, Canada
 7/2018- Affiliate Professor (started as a Visiting Scholar, transitioned in 2018),
 Department of Occupational and Environmental Health Sciences, University of
 Washington School of Public Health, Seattle, WA
 7/2020- Professor (tenured) and Flora L. Thornton Endowed Chair (2020-2024),
 Department of Population and Public Health Sciences (previously, Department of
 Preventive Medicine), Keck School of Medicine, University of Southern
 California, Los Angeles, CA
 7/2024 Sabbatical (1 year, through 6/2025)

ADMINISTRATIVE APPOINTMENTS:

7/1991-6/2006 (Founding) Director, Metals Epidemiology Research Group, Channing Laboratory,
 Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, and
 Department of Environmental Health, Harvard School of Public Health
 7/1992-6/1995 Director, Commission to Investigate the Health and Environmental Effects of Nuclear
 Weapons Production, International Physicians for the Prevention of Nuclear War
 7/1996-6/2006 Director, Residency Program in Occupational and Environmental Medicine, Harvard
 School of Public Health

CV: Howard Hu, M.D., M.P.H., Sc.D.

7/1996-8/2006 Director, Occupational and Environmental Medicine Core, National Institute for Occupational Safety and Health Educational Resource Center at the Harvard School of Public Health

7/1998-6/2004 (Founding) Medical Editor, Environmental Health Perspectives (official journal of NIEHS)

7/2000-8/2006 Associate Director, the Harvard NIEHS Environmental Sciences Center, Harvard School of Public Health

7/2004-6/2009 (Founding) Principal Investigator and Director, Harvard Center for Children's Environmental Health and Disease Prevention Research (co-PI and co-Director after 9/1/08)

9/2006-6/2012 Chair, Department of Environmental Health Sciences, University of Michigan School of Public Health

9/2006-2012 Director, Occupational Epidemiology Core, NIOSH Education and Research Center, University of Michigan

9/2006-2012 Co-Director, Michigan-Harvard/Harvard-Michigan Metals Epidemiology Research Group

7/2009-2011 Director, NIA T32 Training Grant in Aging and Public Health, University of Michigan School of Public Health

1/2010-2012 Chair, Faculty Steering Committee on Global Health, University of Michigan School of Public Health

4/2011-2012 (Founding PI) and Director, University of Michigan NIEHS P30 Core Center.

7/2012-2013 Director and Professor, Dalla Lana School of Public Health, University of Toronto, Toronto, Ontario, Canada

7/2013-6/2018 Founding Dean (2013-2017) and Professor, Dalla Lana School of Public Health, a Faculty of the University of Toronto, Toronto, Ontario, Canada

7/2020-6/2024 Flora L. Thornton Endowed Department Chair (2020-2024) and Professor, Department of Population and Public Health (previously, the Department of Preventive Medicine until July 1, 2021), Keck School of Medicine, University of Southern California, Los Angeles, CA

CLINICAL APPOINTMENTS:

7/1985-6/1987 Attending Physician, Emergency Department, Whidden Memorial Hospital

7/1985-6/1988 Assistant Visiting Physician, Department of Medicine, Boston City Hospital

1/1985-6/2006 Consultant in Occupational and Environmental Medicine, Center for Occupational and Environmental Medicine, Northeast Specialty Hospital (formerly known as the Olympus Specialty Hospital, the Massachusetts Respiratory Hospital, and Norfolk County Hospital).

3/1987-9/1987 Attending Physician, Occupational Health Program, University Hospital/Boston University Medical Center

7/1988-9/2006 Associate Physician, Brigham and Women's Hospital

7/1990-6/1995 Occupational/Environmental Medicine Consultant, Brigham and Women's Hospital Employee Health Services

7/2007-2012 Associate Physician, Division of General Medicine, Department of Medicine, University of Michigan Health System

CV: Howard Hu, M.D., M.P.H., Sc.D.

1/2019-2020 Staff Physician, RotaClinic-Lake City, Seattle, WA
 2021 (pending) Associate Physician, Keck Medical Center

OTHER ACADEMIC POSITIONS and MAJOR VISITING APPOINTMENTS:

7/1987-6/1990 Visiting Physician, South Cove Health Center, Boston (Chinatown)
 7/1996-8/2006 Associate, Center for Health and the Global Environment, Harvard Medical School
 2/1997 Alice Hamilton Visiting Professor, Division of Occupational and Environmental
 Medicine, Department of Medicine, University of California at San Francisco
 11/2000- Visiting Scientist, Sri Ramachandra Medical College and Research Institute
 7/2010- Senior Consultant, Tianjin Centers for Disease Control and Prevention, Tianjin,
 China
 10/2012- Visiting Professor, Shanghai Key Laboratory of Children's Environmental Health,
 Xinhua Hospital, Shanghai Jiao-Tung University, China
 7/2013-6/2016 Visiting Professor, Shanghai Jiao Tong School of Medicine, China
 5/2015- Affiliate Scientist to the Li Ka Shing Knowledge Institute, St. Michael's Hospital,
 Toronto, Canada

MAJOR RESEARCH INTERESTS:

1. Environmental and molecular epidemiologic research related to heavy metals, potential endocrine disruptors, other neurotoxicants, carcinogens, and their impact on adverse health outcomes.
2. Gene-environment interactions; epigenetic dysregulation
3. Fetal/early life exposures and long-term effects
4. Aging-environment interactions
5. Health disparities
6. Health and human rights
7. Health, climate change, sustainability, and the global environment
8. "Big Data" for population health
9. Attitudes, behaviors, the immune response to infection and vaccines, and susceptibilities related to COVID19.

GRANTS (as PI, Co-PI, or primary mentor only):

Past Funding:

1980 (summer) Montefiore Hospital, Bronx NY, PI; \$2,000 (approx)
 A study of rural and occupational health in Tulua, Colombia, South America
 1982 (summer) Albert Einstein College of Medicine, PI; \$3,000 (approx)
 A study of occupational/environmental health in Shanghai, China
 7/1987-6/1989 NIEHS Center Grant ES00002 Pilot Project, PI; \$12,000
 The Long-term Renal and Neurologic Effects of Childhood Plumbism
 7/1989-6/1990 NIEHS subcontract 7083-1, PI; \$50,000 (approx)

CV: Howard Hu, M.D., M.P.H., Sc.D.

The Use of X-Ray Fluorescence to Measure Lead Burden and Childhood Lead Exposure

7/1990-6/1992 Agency for Toxic Substances and Disease Registry, PI; \$150,000 (approx)
"Clinical Environmental/ Occupational Medicine Research Fellowship Award",

7/1990-6/1991 NIEHS Center Grant ES00002 Pilot Project, PI; \$12,000

The Metabolic Effects of Pregnancy and Lactation on Lead Burden

7/1990-6/1991 Harvard School of Public Health Basic, PI

Research Support Grant; \$10,000

K-X-Ray Fluorescence Measured Lead Burden

10/1991-11/1991 NIOSH Special Grants, PI; \$50,000 (approx)

The Carpenters Lead Project

4/1991-3/1996 NIEHS/R01, PI; \$2,200,000 (approx)

The Epidemiology of Lead, Diet and Blood Pressure

7/1991-6/1996 NIEHS/R01 supplement, PI; \$240,000 (approx)

The Epidemiology of Lead, Diet and Blood

Pressure--Research Supplement for Minority Investigator

7/1992-6/1995 NIEHS/R01 (Office of Research on Women), PI; \$200,000 (approx)

Lead and Hypertension in Women

7/1993-6/1996 NIEHS/subcontract, PI; \$150,000 (approx)

Exposure to Neurotoxins as Risk Factors for Amyotrophic Lateral Sclerosis

7/1995-6/1998 State of Washington, Department of Labor, PI; \$350,000 (approx)

SPECT Imaging of the Brain in Patients with Multiple Chemical Sensitivity Syndrome and Controls

7/1996-6/1997 NIEHS Center Grant ES00002 Pilot Project, PI; \$15,000

Electrocardiographic abnormalities in association with low-level lead exposure among middle-aged to elderly men: the Normative Aging Study

4/1995-3/2000 NIEHS Project PI (Program Project PI: Richard Monson); \$1,800,000 (approx)

Lead Exposure, Accumulation in Bone, and Reproductive Toxicity Among Men and Women In Mexico

4/1995-3/2000 NIEHS Project PI (Program Project PI: Richard Monson); \$1,900,000 (approx)

Lead Exposure, Accumulation in Bone, and Cognitive Toxicity Among Elderly Men and Women

6/1997-5/2002 NIEHS/R01 ES05257 PI; \$2,312,274

Lead Biomarkers, Aging, and Chronic Disease

7/1997-6/1999 NIEHS Center Grant ES00002 Pilot Project, PI; \$10,000

The effect of genetic polymorphisms of metallothionein-IIA on mRNA levels in middle-aged to elderly men: the Normative Aging Study

7/1998-6/2003 NIEHS/R01 PI (with no-cost extension; 5R01ES007821); \$2,291,833

Lead Dose Biomarkers, Reproduction, and Infant Outcomes

7/1999-6/2000 NIEHS Center Grant ES00002 Pilot Project, co-PI; \$14,000

Magnetic Resonance Spectroscopy in the Evaluation of Lead Neurotoxicity: the Normative Aging Study

7/2000-6/2001 MAVERIC (Massachusetts Area Veterans Epidemiology Resource and Institute Center) Pilot Project PI (with Dr. Robert Wright, co-PI); \$10,000

The Use of Magnetic Resonance Spectroscopy in Lead Poisoning

CV: Howard Hu, M.D., M.P.H., Sc.D.

CV: Howard Hu

7/2000-6/2001 NIOSH Center Grant Pilot Project, PI (with Dr. Robert Wright, co-PI); \$12,000
Interaction between ApoE Genotype and Lead Exposure in the Development of Cognitive Impairment

7/2002-6/2004 The Rasmussen Foundation/Health Care Without Harm; \$50,000
Medical Use of Phthalate Containing Products in the Neonatal Intensive Care Unit and Biomarkers of Neonatal Phthalate Metabolites

7/2002-6/2003 NIEHS Center Grant Pilot Project, PI; \$8,000
Vitamin D Receptor Gene and Bone Lead in Reproduction

3/2004-2/2005 The Critelli Family Foundation; \$10,000
Review of Environmental Cadmium Exposure and Toxicity

4/2000-3/2007 NIEHS Project Leader (Program Project PI: Richard Monson; 5P01ES05947); \$2,472,677; Controlled Trial in Pregnancy of Dietary Supplements for the Suppression of Bone Resorption and Mobilization of Lead into Plasma (no cost extension)

4/2000-3/2007 NIEHS Project co-Leader (Program Project PI: Richard Monson; 5P01ES05947); \$1,210,000 (approx); A Community-Based Study of Lead Exposure Pathways, Biomarkers of Dose, Health Effects, and Phytoremediation Strategies at the Tar Creek Superfund Site (no cost extension)

4/2002-9/2007 NIEHS/R01 PI (5R01ES010798); \$3,011,295
Gene-Metal Interactions and Parkinson's Disease

10/2003-9/2007 NCMHI/P20 Project Leader (MD000501-01; Hughes Harris, PI); \$828,781 (Project)
"FAMU and Harvard Center for Health and Health Care Disparities"

8/2003-7/2008 NIEHS/R01 PI (2R01ES05257-11A2); \$3,357,424 (became co-PI in 2007 after move to University of Michigan)
Lead-Gene Interactions and Cognition

6/2004-3/2009 NIEHS/P01 PI (5 P01ES012874-01); \$6,662,670 (became co-PI in 2006 after move to University of Michigan)
Metals Mixtures and Children's Health (Center for Children's Environmental Health and Disease Prevention Research)

7/2002-12/2009 NIH/R03 PI (1R03TW005914; no cost ext through 2008); \$192,000 (approx)
Lead, Genes, and Cognition in Children in Chennai, India

9/2006-7/2011 NIEHS/R01 PI (R01ES0007821); \$3,116,831
Fetal Origins of Neurobehavior: Lead and Cholesterol Metabolism Interactions

7/2006-6/2011 NIEHS/R01 co-PI (R01ES013744; PI Wright), \$3,200,000
Stress, Lead, Iron Deficiency and Neurodevelopment

7/2006-6/2011 NIEHS/R01 co-PI (R01ES014930; PI Wright), \$2,800,000
Metal Mixtures and Neurodevelopment

2/2008-2/2010 Michigan Institute for Clinical and Health Research (MICHR; home of the UM CTSA; UL1RR024986) Pilot Project PI; \$26,000 (no cost extension)
Epigenetics of Early Life Events and Environmental Toxicants

4/2009-4/2010 Michigan Alzheimer's Disease Research Center Pilot Project PI, \$25,000
Environment, Epigenetics and Alzheimer's Disease (no cost extension)

12/2009-12/2010 University of Michigan Center for Global Health Pilot Project PI, \$25,000

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Climate Variability and Impacts on Mortality and Morbidity in Chennai, India:
A Pilot Project Stemming from the 2009 U.S.-India Workshop on Climate Change
and Public Health, Goa India (no cost extension)

9/2009-9/2010 Michigan Institute for Clinical and Health Research (MICHHR; home of the UM
CTSA; UL1RR024986) Pilot Project PI; \$26,000 (no cost extension)
Epigenetics and Epigenomics in the Etiology of Alzheimer Disease
7/2008-6/2011 NIA/T32 PI (T32AG027708); \$450,000
Interdisciplinary Training Program in Aging and Public Health

4/2010-3/2015 NIEHS P42 Superfund Co-Inv, Project 2, Co-investigator (P42ES017198; PI:
Alshawabkeh, Project 2 Leader: Meeker) Puerto Rico Testsite For Exploring
Contaminant Threats, \$12,000,000

4/1/2011-6/2015 NIEHS Core Environmental Health Sciences Center, Founding PI and Director
(until 2012; now consultant; P30 ES017885), \$ 4,620,100;
“Lifestage Exposures and Adult Disease”

4/2010-3/2014 NIEHS/EPA P20 Co-PI and Clin Health Specialist (P20 ES018171; PI Peterson)
Formative Children’s Environmental Health and Disease Prevention Center,
\$1,959,960; “Perinatal Exposures, Epigenetics, Child Obesity & Sexual Maturation”

7/1/2013-6/30/2014 CIHR, Canadian Institute for Health Services and Policy Research; Planning
Grants-Priority Announcement:Partnerships for Health System Improvement; PI, \$24,992
“The Surviving Opioid Overdose with Naloxone (SOON) Project and Roundtable”

07/1/11-06/30/16 NIEHS K01 ES019909 (co-mentor; PI: Somers)
“Immune dysfunction associated with early life heavy metal exposure”

4/1/12-3/30/17 NIEHS R01ES013744 (consultant; PI: Wright; Mt Sinai School of Medicine)
“Stress-Lead Interactions and Child Development”

7/1/2012-7/1/2017 European Commission (EC), Funded under FP7-Health, Project 304925, co-
Investigator; PI, epidemiologic studies, \$6,000,000 E
“A novel micronutrient-based strategy to prevent hearing impairments: test and road to
market for age-related hearing loss and preservation of residual hearing”

6/1/2012-7/1/2020, 1R01ES021446, PI, \$4,140,000 (parent + supplement awards);
“Prenatal and Childhood Exposure to Fluoride and Neurodevelopment”

5/15/2015-5/15/2020 Health Canada; PI, \$200,000 (Phase 1); \$1,400,000 (proposed Phase
2) “A Community-based First Nation Study of Cancer and the Environment in Northern
Ontario”

5/1/2021-4/30/2022 Environmental Pollutant Risk Factors for Worse COVID-19 Related Clinical
Outcomes, PI, \$49,999; the Southern California Environmental Health Sciences Center
Pilot Project Program, University of Southern California, Los Angeles, CA

3/8/2021-3/8/2022 The USC SARS-CoV-2 Vaccination Campaign Research Initiative: Uptake,
Markers and Determinants of Effectiveness, Subsequent Behaviors, PI, \$1,200,000; The
Keck COVID-19 Research Fund in the Keck School of Medicine; USC Office of the
Provost; USC Office of Research.

11/1/2020-10/30/2022 The Population Health COVID-19 Pandemic Research Center, co-PI,
\$320,000, The Keck Medicine COVID-19 Research Initiative; the Keck Foundation

3/24/2020-2/28/2022 Pilot Project Proposal for Rapid Response Funding, University of Michigan

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- NIEHS P30 Core Sciences Center (co-PI); \$6,250, “Environmental Cadmium and Influenza-related Mortality in NHANES: An Environment-Infectious Disease Interaction Study with Implications for Strategies for Reducing COVID-19-related Morbidity and Mortality”
- 9/1/16-8/31/22 NIH 5R01ES026033-02, (Consultant/Co-investigator; PI: Arora at Mt. Sinai School of Medicine) \$648,000 “Novel Biomarker to Identify Critical Windows of Susceptibility to Metal Mixture”
- 12/31/2020-6/30/2023 The Los Angeles Pandemic Surveillance Cohort Initiative, PI, \$ 1,997,934; The Los Angeles County Public Health Department, PH-003903-W2/U.S. Centers for Disease Control and Prevention Cooperative Agreement US0CK000498.
- 4/1/13-3/31/23 NIEHS/EPA P01ES022844 (co-inv; PI: Peterson at the University of Michigan) “Lifecourse Exposures & Diet: Epigenetics, Maturation & Metabolic Syndrome.”

Current Funding

- 8/22/23-10/31/25 Research and Innovation (R&I) Collaborative Research Planning Award Office of Research, University of Southern California, MPI, \$72,074; “Building a Faculty Team and Platform for Research on Health and Health Policy that Improves Population Health in a Low Resource Setting in India Known for Innovation: the State of Meghalaya”
- 7/1/16-6/30/26 CIHR (co-PI; Director; PI: Jeffrey Brook at the Dalla Lana School of Public Health) \$4,700,000 CNDN “CANadian Urban Environmental (CANUE) Health Research Consortium”
- 9/1/17-6/30/27 NIH R24ES028502 (Consultant/Co-investigator; PI: Peterson at the University of Michigan, “E3GEN: Multigenerational Effects of Toxicant Exposures on Life Course Health and Neurocognitive Outcomes in the ELEMENT Birth Cohorts”; \$2,009,022

Applications Under Review

- 1/1/2025-12/31/2025 Reckitt Global Hygiene Institute Foundation, PI, \$199,997 “Climate Change and Precipitation Extremes in India: The Diarrhea Outbreaks, Sanitation and Hygiene Interventions (DOSHI) Project”
- 1/1/2025-12/31/2025 Pilot Project Proposal, Southern California Environmental Health Sciences Center NIEHS P30 Pilot Project Program, co-I (PI: T. Islam) “Effects of Climate Change on Respiratory Diseases in Meghalaya, India”
- 7/1/2025-6/30/2027 Novo Nordisk Fonden Foundation, PI, \$1,015,254 (Application No. 0093393) “Remedying the Effects of Climate Change on Respiratory and Diarrheal diseases (RECoRD): A Scalable Project in Rural India”

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7/1/2025-6/30/2030 NIH 1 RM1 NS143787-01, PI, \$12,280,616

“The Multiethnic Translational Research on Neuro-PASC (METRON) Project)

HONORS AND AWARDS:

1978-1982 National Health Service Corps Scholarship

1985-1988 National Research Service Award

1990-1992 Agency for Toxic Substances and Disease Registry Clinical Environmental Medicine Award

1994 Will Solimene Award of Excellence, American Medical Writers Association, for: Chivian E, McCally M, Hu H, Haines H, eds. *Critical Condition: Human Health and the Environment*. Cambridge: The MIT Press, 1993.

1997 Alice Hamilton Lecturer, University of California at San Francisco

1998 First Prize for Best Infant Nutrition Research, Instituto Danone, Mexico (for González-Cossío T, Peterson KE, Sanín L, Fishbein SE, Palazuelos E, Aro A, Hernández-Avila M, Hu H. “Decrease in birth weight in relation to maternal bone lead burden.” Published in *Pediatrics*)

1999 National Institute for Environmental Health Sciences “Progress and Achievement of the Year Award”, 1998-1999

1999 True Memorial Lecturer, Maine Medical Center, Portland ME.

2000-2001 Faculty Sabbatical Award, Harvard School of Public Health

2000-2001 Senior Fulbright Scholar in India

2001 Hoopes Prize, Faculty Mentorship (for Senior Thesis of Charles Lin, “More than Black and White: Lead Poisoning as an Environmental Justice Issue in Boston”)

2003 Best Paper in Preventive Medicine by a Medical Student (for Senior Thesis of Vanitha Janakiraman; Janakiraman V, Hu H, Mercado-Garcia A, Hernandez-Avila M. A randomized crossover trial of nocturnal calcium supplements to suppress bone resorption during pregnancy. *Am J Prev Med* 2003;24:260-4.). American College of Preventive Medicine, Ulrich and Ruth Frank Foundation for International Health.

2004 Das Travel Grant Award, The South Asia Initiative, Harvard University (for Travel in India)

2005 Adolph G. Kammer Merit in Authorship Award, the American College of Occupational and Environmental Medicine (for Rhodes D, Spiro A, Aro A, Hu H "Relationship of Bone and Blood Lead Levels to Psychiatric Symptoms: The Normative Aging Study", Published in the *Journal of Occupational and Environmental Medicine*)

2006 Teacher of the Year Award, Occupational/Environmental Medicine Residents, Harvard School of Public Health

2006 Harriett Hardy Award, the New England College of Occupational and Environmental Medicine

2009 Linus Pauling Award for Lifetime Achievements, American College for the Advancement of Medicine

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- 2011 Award for Excellence, American Public Health Association
- 2015 John R. Goldsmith Award for Outstanding Contributions to Environmental Epidemiology, International Society for Environmental Epidemiology
- 2016 Election to Fellowship, Canadian Academy of Health Sciences

MEMBERSHIPS IN PROFESSIONAL SOCIETIES

Memberships

- 1981- American Public Health Association (APHA)
- 1982-2006 Massachusetts Coalition for Occupational Safety and Health
- 1983-1989 American College of Physicians
- 1985- Physicians for Social Responsibility
- 1987- Physicians for Human Rights
- 1990- International Society for Environmental Epidemiology (ISEE)
- 1990-2000 American Association for the Advancement of Science
- 1990-2006 Association of Occupational and Environmental Clinics (AOEC)
- 1991- International Physicians for the Prevention of Nuclear War (IPPNW)
- 1994-1996 Society for Occupational and Environmental Health (SOEH)
- 2000-2012 American College of Occupational and Environmental Medicine (ACOEM)
- 2009-2012 Society of Toxicology
- 2012-2018 Canadian Public Health Association (CPHA)
- 2020- Washington State Medical Association
- 2020- International Society for Children's Health and the Environment
- 2023- California Academy of Preventive Medicine

Committee Assignments

- 1981-1982 Program Committee, Occupational Safety and Health Section, APHA
- 1987-1988 Program Committee, Asian-American Caucus, APHA
- 1992-1998 Membership Committee, ISEE
- 1995-1998 Quality Assurance Committee, AOEC
- 1997-1998 Program Committee, 1998 Superfund Basic Research Program, Annual National Meeting
- 2001-2006 Program Committee, New England College of Occupational and Environmental Medicine Annual Meetings

EDITORIAL POSITIONS AND BOARDS:

- 1977-1982 Einstein Community Health Newsletter
- 1988-1992 Bookreview Co-Editor, Section on Occupational Safety and Health, Am Public Health Assoc.

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1993- Journal of Health and Human Rights
 1998- Environmental Health Perspectives (Founding Medical Editor, 1998-2004; Associated Editor, 2004-)
 2004- American Journal of Industrial Medicine
 2007-2009 Faculty of 1000 Medicine
 2017- Current Environmental Health Reports
 2017- Faculty of 1000 Medicine

PEER REVIEW SERVICE

American Journal of Clinical Nutrition
 American Journal of Epidemiology
 American Journal of Industrial Medicine
 Archives of Environmental and Occupational Health
 Biomed Central
 Circulation
 Environmental Epidemiology
 Environmental Health
 Environmental Health Perspectives
 Environment International
 Environmental Research
 Epidemiology
 Indian Journal of Medical Research
 Journal of Health and Human Rights
 Journal of the American Medical Association
 Kidney International
 Lancet
 New England Journal of Medicine
 Pediatrics
 PLOS One
 Science of the Total Environment

TEACHING:

1. LOCAL CONTRIBUTIONS (at the Harvard School of Public Health, 1985-2006)

1985- “Toxicology of the Kidney and Urinary Tract”
 Guest Lecturer for TOX204a,b
 1988- “Occupational Health”
 Guest Lecturer for EH201a,b
 1989-1992 “Lead Toxicology”

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- 1990- Guest Lecturer for TOX204a,b
Grand Rounds in Occupational/Environmental Medicine
- Director
- 1990-2000 Introduction to Occupational and Environmental Medicine (EH232c,d)
Course director, lecturer
- 1990- "The Epidemiology of Lead Exposure, Dose, and Toxicity"
Guest Lecturer for EPE215c,d and EPE215t
- 1990- "Solvent toxicity"
Fundamentals of Industrial Hygiene, Continuing Education Department
- 1992 "Current Research on Lead", Metals Epidemiology Research Group Seminar
Presenter
- 1992 "Lead Poisoning Without a Known Source in a Hyperthyroid Patient"
Case discussant, Grand Rounds in Occupational and Environmental Medicine
- 1992- "Biological Markers of Lead Dose"
Guest Lecturer, EHE280c,d
- 1994- "Screening for Lead Toxicity"
Guest lecturer, EPI227d
- 1994- "Lead Exposure and Biological Monitoring"
Guest Lecturer, ID263b
- 1994- "Case Study: Lead"
Guest Lecturer and Case Discussant, EH202d
- 1996- Introduction to Environmental Health (EH201b)
Course director and lecturer
- 1997- Human Health and Global Environment Change (EH278a,b)
Course Co-developer, Co-director, and lecturer

Hospital courses and Invited Teaching Presentations (Harvard-affiliated Hospitals)

- 1990 Guest Lecturer on Occupational Medicine
Residency Program, Department of Medicine, Brigham and Women's Hospital
- 1994 Speaker, Grand Rounds; "Is Lead a Ticking Time Bomb?"
Department of Obstetrics and Gynecology, Brigham and Women's Hospital
- 1994 Speaker, Grand Rounds; "Is Lead a Ticking Time Bomb?"
Department of Medicine, Brockton V.A. Hospital
- 1994 Speaker, Symposium on Preventive Medicine and Clinical Epidemiology,; "Is Lead a Ticking Time Bomb"; Brigham and Women's Hospital
- 1995 Discussant, "Multiple Chemical Sensitivity", Occupational/Environmental Medicine
Grand Rounds, Occupational Health Program, Harvard School of Public Health
- 1996 Guest lecturer, "Lead Toxicity as a Paradigm for a Regional and Global Health Hazard", Environmental Health Student Group, Holmes Society, Harvard Medical School
- 1997 Speaker, "Mobilization of maternal bone lead as a hazard to the fetus", Grand Rounds, Dept. of Neonatology, Beth Israel Hospital, Boston, MA
- 2000 Guest lecturer, "Update on Lead Toxicity Research", Program in Pediatric

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- 2000 Toxicology, Children's Hospital
Discussant, "Adult Lead Toxicity", Weekly Case Round, Department of Medicine, Brigham and Women's Hospital, Boston.
- 2000 Lecturer, "Update on Lead Toxicity, Hypertension, and Chronic Renal Failure", Renal Rounds, Division of Nephrology, Department of Medicine, Brigham and Women's Hospital, Boston.
- 2002 Lecturer, "Maternal Bone Lead as a Threat to Fetal Development", Program in Neonatology, Beth Israel-Deaconess Hospital, Boston, MA

Doctoral student committees

Chair and member:

- | | |
|-----------------------|---|
| Dr. Rokho Kim | Dr.P.H. Occupational Health and Epidemiology, '96 |
| Dr. Yawen Cheng | Sc.D. Epidemiology, '98 |
| Dr. Sharon Tsaih | Sc.D. Epidemiology, '99 |
| Dr. Hung Yi Chuang | Sc.D. Occupational Health, '99 |
| Dr. Adrienne Ettinger | Sc.D. Environmental Health, '03 |
| Dr. Florence Wang | Sc.D. Environmental Health, '05 |
| Dr. Sung K. Park | Sc.D. Environmental Health, '05 |
| Dr. Pradeep Rajan, | Sc.D. Occupational Health, '06 |

Member/Advisor:

- | | |
|---------------------|---|
| Dr. How Ran Guo | Sc.D. Occupational Health, '94 |
| Dr. Joshua Cohen | Sc.D. Health Policy and Management, '94 |
| Dr. Jane Hoppin | Sc.D. Environmental Health, '95 |
| Dr. Salma Elreedy | Sc.D. Environmental Health, '97 |
| Dr. Mary Jean Brown | Sc.D. Maternal and Child Health, '00 |
| Dr. Brisa Sanchez | Sc.D. Biostatistics, '06 |
| Dr. Ami Zota | Sc.D. Environmental Health, '07 |
| Dr. Ananya Roy | Sc.D. Environmental Health, '08 |
| Dr. Elissa Wilker | Sc.D. Environmental Health, '09 |

Post-doctoral fellow mentor:

Dr. Marinelle Payton (Channing Lab), Dr. Susan Korrick (Channing Lab), Dr. Rokho Kim (Channing Lab), Dr. Viji Potula (HSPH Research Fellow), Dr. Barbara Nowak (Visiting Scientist from Silesian University School of Medicine, Poland), Dr. Robert Wright (Channing Lab), Dr. Ming Tsuang Wu (HSPH Research Fellow), Dr. Yawen Cheng (Channing Lab), Dr. Geeta Mathur (neonatology fellow at the Brigham and Women's Hospital), Dr. Sri Hari Bojja (HSPH Research Fellow), Dr. Hae-Kwan Cheong (Visiting Scientist from Dongguk University School of Medicine, S. Korea), Dr. Sahar Elmarsafawy (HSPH Research Fellow), Dr. Jing Lu (Visiting Scientist from the Chinese Academy of Preventive Medicine), Dr. Dieter Affeln (Occ/Env Med Fellow), Dr. Ahmed Gomaa (Occ/Env Med Fellow), Dr. Chris Leffler (Occ/Env Med Fellow), Dr. Ronald Dykeman (Occ/Env Med Fellow), Dr. Uma Dhanabalan (Occ/Env Med Fellow), Dr. Hsien-Wen Hsu (Occ/Env Med Fellow), Dr. Betty Ann Cohen (Occ/Env Med Fellow), Dr. Arvin Chin (Occ/Env Med Fellow),
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Dr. Daniel Rhodes (Occ/Env Med Fellow), Dr. Richard Wittman (Occ/Env Med Fellow), Dr. Sun-Dong Lee (Visiting Scientist from Sangji University, Korea), Dr. Ronald Green (Occ/Env Med Fellow), Dr. Erma Lawson (Environmental Health Fellow), Dr. Marc Weisskopf (Environmental Health Fellow), Dr. Bridget Bagert (Occ/Env Med Fellow), Dr. John Jarrell (Visiting Scientist from University of Calgary), Dr. Jennifer Weuve (Environmental Health Fellow), Dr. Karen Chou (Visiting Scientist from Michigan State), Dr. Nitin Jain (Channing Laboratory Fellow), Dr. Adrienne Ettinger (Children's Center Scientist), Dr. Sam Myers (Fellow in Alternative and Complementary Medicine), Dr. Marcelo Targino (Occ/Env Med Fellow), Dr. Manish Arora (Post-doctoral fellow from University of Sydney), Dr. Huiling Nie (Post-doctoral fellow from McMaster University).

Other faculty mentorship:

Elizabeth Rubinstein (HMS Summer research), Alicia Marier (HMS Summer research), Vanitha Janakiraman (HMS Summer research), Young-Sook Lim (Harvard College Summer research), Charles Lin (Harvard College Senior thesis research), Ed Hsieh (Harvard College Summer research), Naveen Thomas (Emory University Medical School Senior thesis research), Shreekrishna Akilesh (Harvard Dental School summer research), Christine Pace (HMS Summer research)

Advisory and supervisory responsibilities

1985-1987	Attending Physician, outpatient general medicine clinic, Boston City Hospital; weekly precepting for housestaff and medical students
1990-2006	Preceptor, Residency in Occupational and Environmental Medicine, Harvard School of Public Health at the Mass Respiratory Hospital
1990-2006	Advisor to general M.P.H. students, Harvard School of Public Health.

2. LOCAL CONTRIBUTIONS (at the University of Michigan, 2006-2012)

2006-	<u>Principles of Environmental Health (EHS-500)</u> Course director and lecturer
2006-	<u>Environmental Epidemiology (EHS-608)</u> Guest lecturer on birth cohorts and environmental epidemiology
2006-	<u>Occupational and Environmental Disease (EHS-501)</u> Guest lecturer on metals exposure and health effects; Course Director (2009-)
2007-	<u>Metals Exposure, Biomarkers and Toxicity: A Multi-disciplinary Environmental Epidemiology Approach (EHS-698 reading course)</u> Course director and lecturer
2008-2009,	<u>Topics in Environmental Health Sciences (EHS-688)</u>
2010-2011	Course director and lecturer
2009	<u>Occupational and Environmental Disease (EHS-501)</u> Course director and lecturer
2009-	<u>On-line (Long-distance Foundations in Public Health Certificate Program): Principles of Environmental Health (EHS-500-801)</u> Course director and lecturer
2009	Introduction to Public Health (HMP-200)

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- 2009- Guest lecturer on environmental health
Seminars in Aging and Public Health (EPID 813)
 Course director and lecturer
- 2011 Seminar on Public Health in China (HMP-xxx)
 Guest lecturer on “Environmental Health in China”

Post-doctoral fellow mentor:

Dr. Sung Kyun Park (Environmental Health Sciences Fellow, now Research Assistant Professor), Dr. Brisa Sanchez (Biostats Research Assistant Professor, now Assistant Professor), Dr. Richard Pilsner (Robert Wood Johnson Health & Society Fellow), Dr. Aimin Zhang (Environmental Health Sciences Fellow, Toxicology Training Grant), Dr. Ananya Roy (Environmental Health Sciences Fellow), Dr. David Cantonwine (Reproductive Sciences Fellow).

Doctoral Student Advisor (principal)

- | | |
|--------------------|---|
| David Cantonwine | Ph.D. Environmental Health Sciences (2009) |
| Myriam Afeiche | Ph.D. Environmental Health Sciences (co-mentor with Karen Peterson; 2010) |
| Yoon-Hyeong Choi | Ph.D. Environmental Health Sciences (co-mentor with Sung Kyun Park; 2011) |
| Katie F. Bush | Ph.D. Environmental Health Sciences (co-mentor with Marie O’Neill; 2011) |
| Kelly Bakulski | Ph.D. Environmental Health Sciences (2012) |
| Gamola Fortenberry | Ph.D. Environmental Health Sciences (co-mentor with John Meeker; 2013) |
| Siying Huang | Ph.D. Environmental Health Sciences (2013) |
| Deena Thomas | Ph.D. Environmental Health Sciences (2014) |
| Rebecca Tutino | Ph.D. Environmental Health Sciences (2015) |
| Zishaan Farooqui | Ph.D. MD-PhD Medical Scientist Training Program (2015) |

Masters Student Thesis Advisor

Bradley Lampe (OEE), Troy Meissner (OEE), Pheba Alexander (OEE), Brian Davis (OEE & HBHE), Aaron Leftwich (OJOC program), Suengwon Lee (Nutrition), Allen Zhong (OEE), Graham Newman (OEE), Jacqueline Barkoski (OEE)

Undergraduate Thesis Advisor

Lauren Schwartz (Neuroscience, LSA)

3. LOCAL CONTRIBUTIONS (at the University of Toronto, 2012-2017)

- 2012 Determinants of Community Health (Faculty of Medicine)
 Guest lecturer on ‘The Future of Medicine & Public Health in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World’.
- 2012- CHL5004H Introduction to Public Health

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- Guest lecturer on “The Future of Public Health (and Your Role !) in a Hot, Flat, Crowded...and Diverse, Aging, Stratified, Urbanized, Polluted, Thirsty, Hungry, Debt-Ridden World”. “What is Public Health?”, “Climate Change and Health”
- 2012- CHL 5912F Industrial Toxicology.
Guest lecturer on the “Toxicology of Metals”.
- 2013-2014 Department of Family & Community Medicine “Building Blocks” (short course for International post-graduate primary care trainees); Guest lecturer on “Public Health & Primary Care”
- 2013- CHL5701H Doctoral Seminar, Collaborative Doctoral Program in Global Health
Guest lecturer on “The Challenges of Environmental Health in a Rapidly-Changing World, from the Molecular to the Global”.
- 2014 JCR1000 “Interdisciplinary Approach to Global Challenges”
Guest lecturer on “Global Environmental Health”
- 2014- PHS100H1 “Grand Opportunities in Global Health”; Guest lecturer on “Urban Environments”
- 2015 Public Health & Preventive Medicine Residency Rounds “Physicians, Climate, and other Global Environmental Changes: Our Role”
- 2016 CHL5004H Introduction to Public Health, Course Co-Director (with Professor Erica DiRuggiero)
- 2016 CHL 7001H F6 Environmental-Molecular Epidemiology, Course Co-Moderator (with Professor Morteza Bashash)
- 2016 CHL5701H Doctoral Seminar, Collaborative Doctoral Program in Global Health, Course Co-Director (with Professors Erica DiRuggiero and Abdallah Daar)
- 2016 Joint Seminar, “The Impact on Intelligence, Behaviour, and Society of Lead Exposure: A Case Study of a Global Pollutant and On-going Research”; Collaborative Program in Neurosciences and Collaborative Global Health Doctoral Program, University of Toronto
- 2016 CHL5420H “Global Health Research Methods”
Guest lecturer on “The Early Life Exposures in Mexico to Environmental Toxicants Project (ELEMENT): A Global Health Collaboration Case Study”

Masters student research advisor
Maele Marchand

Doctoral student advisor
Adele Carty

Doctoral student thesis committee member
Laura Bogaert

Doctoral student thesis examination committee member
Claudie CY Wong (doctoral student in epidemiology, Jockey School of Public Health and Primary Care, Chinese University of Hong Kong)
Zilong Zhang (doctoral student in epidemiology, Jockey School of Public Health and

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Primary Care, Chinese University of Hong Kong)

Post-doctoral fellow mentor:

Siying Huang, Ph.D.; Morteza Bashash, Ph.D.; Roman Pabayo, Sc.D. (Harvard School of Public Health); Tripler Pell, M.D., M.P.H.

4. LOCAL CONTRIBUTIONS (at the University of Washington, 2017-2020)5

Doctoral student thesis research mentor

Megan Suter

Doctoral student special projects advisor

Rachel Shaffer

Joey Frostad

Rebecca De Buen

5. LOCAL CONTRIBUTIONS (at the University of Southern California, 2020-present)

2020- PM 502 Foundations of Public Health

Guest lecturer on “Global Health and the Global Burden of Disease Study”

2020- PM 601 Basic Theory and Strategies in Prevention

Guest lecturer on “Sociocultural Theories: Health Disparities and Environmental Justice”

2021- Health Justice and Systems of Care curriculum, Keck School of Medicine Year 01

Guest lecturer on “Introduction to Public Health (in a Hot, Crowded, Diverse, Aging, Inequitable, Urbanized, Polluted, Thirsty, Hungry, Debt-Ridden World)”

Medical student research advisor

Kelly Burk (2022-2023; now a Neurology Resident)

6. NIH K-grant mentorship:

Robert Wright, M.D., M.P.H. (K-23 ES000381, “*Neurochemical and Genetic Markers of Lead Toxicity*”), 2000-2005; Dr. Wright is now Professor of Pediatrics, Mt. Sinai School of Medicine

Marc Weisskopf, Ph.D. (K-01 ES012653, “*New Biomarkers of Neurotoxicity*”), 2004-2009; Dr.

Weisskopf is now Professor (tenured) of Occup Health, Harvard Sch Public Health

Sung Kyun Park, Sc.D. (K-01 ES016587; “*Environment, Novel Aging Outcomes, and Genetics*”),

2009-2014; Dr. Park is now Associate Prof (tenured), Department of Epidemiology, University of Michigan Sch Public Health

Emily Somers, Ph.D. (K-01 ES019909; “*Immune Dysfunction Associated with Early Life Heavy Metals Exposure*”), 2011-2016; Dr. Somers is now Associate Prof, Division of Rheumatology,

Department of Internal Medicine, University of Michigan Medical School

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Ashley Malin, Ph.D. (K-99/R00 ES031676; “*Childhood fluoride exposure and sleep patterns*”, 2021-2026; Dr. Malin is now an Assistant Professor, Department of Epidemiology, University of Florida School of Public Health.

COMMITTEE, ORGANIZATIONAL, AND VOLUNTEER SERVICE

National/International

1978-1982 Taskforce on Occupational and Environmental Health, Co-coordinator, Am Med Stu Assoc

1989 Ad Hoc Study Committee, National Institute for Environmental Health Sciences Council

1989-2006 Association of Occupational and Environmental Medicine Clinics (AOEC)-- (through the Northeast Specialty Hospital Center for Occupational and Environmental Medicine)

1989-1990 Member, Relative Risk Reduction Strategies Committee, Science Advisory Board, U.S. Environmental Protection Agency

1989-1992 Member, Board of Directors, Physicians for Human Rights, Boston, MA

1991 National Institutes of Health, General Clinical Research Center Program, Site Visit Team

1992-2019 Member, National Advisory Committee, Physicians for Human Rights, Boston, MA

1992 Special Study Section member (R3/S1/B3), National Institutes of Health

1994 Ad Hoc Reviewer, National Institutes of Health, General Dental Research Center Program

1994 Advisory Board, Institute for Energy and Environmental Research

1994-1996 Associate, Project on Global Environmental Change and Health, Physicians for Social Responsibility

1995 Ad Hoc Reviewer, National Institutes of Health, Diagnostic Radiology Study Section

1996- Member, Editorial Board, Health and Human Rights—an International Journal

1995-1998 Advisory Committee, Consortium for Environmental Education in Medicine, Cambridge, MA.

1996-1997 Reviewer, Agency for Toxic Substances and Disease Registry

1997-1998 Program Committee, Annual Mtg, NIEHS Superfund Basic Research Group Centers

1998-2013 (Founding) Medical Editor (1998-2004); Associated Medical Editor (2004-), Environmental Health Perspectives (official journal of NIEHS)

2001 Ad Hoc Reviewer, National Institutes of Health, R-13 applications

2002-2006 External Advisory Committee, Program Project on Lead and Osteoporosis, University of Rochester

2003-2005 Member, Ad-Hoc Expert Panel to Form Medical Management Guidelines for Lead-Exposed Adults, (supported by NIOSH and AOEC)

2003-2009 Member, Working Group on Lead and Pregnancy, Advisory Committee on Childhood Lead Poisoning Prevention, U.S. Centers for Disease Control and Prevention

2004 Ad Hoc Reviewer, National Institutes of Health, K-23 applications

2004 Ad Hoc Reviewer, Draft of “Immunization Safety Review: Vaccines and Autism” Immunization Safety Review Committee, Institute of Medicine, National Academies of

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- Science
- 2004 Finalist (one of 8), Search for Director, National Institute for Environmental Health Sciences, U.S. National Institutes of Health
- 2005 Member, Strategic Planning Conference, National Institute for Environmental Health Sciences, Research Triangle Park, NC
- 2006 Ad Hoc Reviewer, Draft of "Preterm Birth: Causes, Consequence, and Prevention" Committee on Understanding Premature Birth and Assuring Health Outcomes, Institute of Medicine, National Academies of Science
- 2006 Member, External Advisory Committee, NIEHS Center, University of Rochester
- 2007 Member, Ad Hoc Study Section, Special Emphasis Panel/Scientific Review Group 2007/05 ZES1 JAB-C (DI) (NIEHS Discover Centers)
- 2007-2010 Member, Board on Population Health and Public Health Practice, Institute of Medicine, National Academies, Washington DC.
- 2007 Member, Ad Hoc Review Panel, Centers of Excellence Program, Swedish Council for Working Life and Social Research.
- 2007-2008 Member, Search Committee for Director of Extramural Research, NIEHS
- 2007 Special Consultant, Ad Hoc Study Section, Special Emphasis Panel/Scientific Review Group 2008/01 ZAR1 CHW-G (NIAMS Arthritis Centers)
- 2008 Report Reviewer, Draft National Research Council Report, "The National Children's Study Research Plan: A Review", National Academies
- 2008 Report Reviewer, Draft National Research Council Report, "Gulf War and Health: Updated Literature Review of Depleted Uranium", Institute of Medicine, National Academies
- 2008-2009 Data Safety Monitoring Board, "d-Penicillamine Chelation in lead-poisoned Children—A Phase II/III Trial" (R01FD003361; PI: Michael Shannon)
- 2008 Subcommittee to review Draft Report on Bisphenol A, Science Board, Food and Drug Administration
- 2008 Planning Committee, International Symposium on the Environmental and Health Consequences of Metal Mining and Smelting
- 2008-2009 Co-Chair, Planning Committee, "Climate Change Impacts on Public Health in India", Workshop that took place in Goa, India in Aug-Sept 2009 co-sponsored by UM Center for Global Health, the US Centers for Disease Control and Prevention and the Indian Council for Medical Research
- 2008 Finalist (one of 2), Search for Director, National Institute for Environmental Health Sciences, U.S. National Institutes of Health
- 2009-2012 Member, Board on Environmental Studies and Toxicology, National Research Council
- 2009 Reviewer, NIH Challenge Grants, Special Emphasis Panel/Scientific Review Group 2009/10 ZRG1 GGG-F
- 2009-2010 External Member, Academic Program Review Site Visit Committee, Department of Environmental and Occupational Health Sciences, University of Washington School of Public Health
- 2010-2012 Member, External Advisory Committee, University of Rochester NIEHS P30 Core Center

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- 2010 Member, Ad-hoc review committee, National Health Research Institutes of Taiwan, Special Emphasis Panel—NHRI-Kaoshiung Medical College Program Project on “: “Gene Environment Interaction in the Genesis of Asthma and Allergic Diseases”
- 2010-2012 Member, Advisory Board, Institute of Public Health, Florida Agricultural & Mechanical University, Tallahassee, FL
- 2011 Reviewer, NIEHS Career Development Awards, Special Emphasis Panel/Scientific Review Group 2011/05 ZES1 LKB-J (K9)
- 2011-2016 Member, NIEHS National Advisory Environmental Health Sciences Council
- 2012 Member, Editorial Board, Journal of Alzheimer’s Disease
- 2015 Member and External Reviewer, School of Population and Public Health Review Committee, University of British Columbia, Vancouver, B.C.
- 2016-2021 Chair, Board of Directors, Canadian Urban Environmental Health Research Consortium, (National Consortium based out of the Dalla Lana School of Public Health)
- 2017-2023 Member, Energy Research Committee, Health Effects Institute, Boston, MA
- 2017-2018 Executive Co-Chair, Workshop on the Global Burden of Disease-Pollution and Health Initiative, March 1-2, 2018, Institute for Health Metrics and Evaluation, Seattle, WA
- 2017- Executive Co-Leader, Global Burden of Disease-Pollution and Health Initiative
- 2019-2022 Member, Research Advisory Committee, Centre of Environmental Health, The Public Health Foundation of India and the Tata Institute of Social Sciences, New Delhi, India
- 2019 Reviewer, draft report on trace metals levels in pregnancy women, Agency for Toxic Substances and Disease Registry, Centers for Disease Control and Prevention, Atlanta
- 2019 Reviewer, draft report on Concentration-Response Functions between Lead Exposure and Adverse Health Outcomes for Use in Benefits Analysis: Cardiovascular-Disease Related Mortality”, EPA National Center for Environmental Economics Office of Policy
- 2019- Member, Advisory Council, Physicians for Human Rights, New York, NY
- 2019 Reviewer, Special Emphasis Panel/Scientific Review Group 2020/01 ZES1 LAT-S (K9) Applications, Center for Scientific Review, U.S. National Institutes of Health
- 2019-2020 Member, Board of Advisors Taskforce, Marilyn Brachman Hoffman Foundation, Dallas, TX
- 2020-2021 Member, External Advisory Committee, New York University/NIEHS Environmental Health Core Sciences Center, New York, NY
- 2020 Member, NIEHS DR2 Work Group SARS-CoV-2/COVID-19 Environmental Health Research Needs Panel.
- 2020- Chair (2020-2024), member, Scientific Advisory Board, the Marilyn Brachman Hoffman Foundation, Dallas, TX
- 2020 Member, The Lancet Commission on Pollution and Health: Update
- 2021-2024 Member, Clean Air Scientific Advisory Committee (CASAC) Lead Review Panel, U.S. Environmental Protection Agency
- 2022 Member, Review Panel, RFA-RM-21-025: NIH Faculty Institutional Recruitment for Sustainable Transformation (FIRST) Program: FIRST Cohort. Office of the NIH Director, Office of Strategic Coordination.
- 2022- Member, Board of Directors, Pure Earth, New York, NY.
- 2022- Co-Chair, International Research Council, The Public Health Foundation of India, Delhi,

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- India
- 2023- Member, Board of Directors, Wellness Equity Alliance, Los Angeles, CA
- 2023- Member, Lead Working Group, Pahle Foundation, Delhi, India
- 2024- Member, Workgroup on Prevention of Lead Exposure in Adults of the Lead Exposure and Prevention Advisory Committee (LEPAC), National Center for Environmental Health/Agency for Toxic Substances and Disease Registry

Regional

- 1988-1990 Health Facilities Appeals Board, Member, Dept. Public Health, Comm. Of Mass.
 - 1988-2006 Advisory Board, Massachusetts Department of Public Health, Sentinel Event Notification System for Occupational Risks (SENSOR) Project
 - 1989-1995 Advisory Board, Massachusetts Division of Occupational Hygiene, Lead Registry Project
 - 1990-1992 Board of Directors, Member, Health Care for All, Boston, Massachusetts
 - 1993-1995 Faculty Council, Member, Harvard School of Public Health
 - 1995-2006 Faculty Advisory Committee, Public Health Practice Program, Harvard School of Public Health
 - 1996-2006 Advisory Board, Boston VA Environmental Hazards Center, Boston
 - 1997-2001 Faculty Steering Committee, Center for Children's Health, Harvard School of Public Health
 - 1996-2006 Senior Epidemiology Consultant, Massachusetts Veterans Epidemiology Research and Information Center, Boston.
 - 1996-2006 Associate, Center for Health and the Global Environment, Harvard Medical School
 - 1997-2002 Faculty Advisory Committee on Continuing Professional Education, Harvard School of Public Health
 - 1998-2006 Faculty Steering Committee, Masters of Public Health program, Harvard School of Public Health
 - 2001-2003 Board of Directors, New England College of Occupational and Environmental Medicine
 - 2001-2006 Associate Director, Harvard NIEHS Environmental Sciences Center, Harvard School of Public Health
 - 2001-2006 Senior Advisory Council Member, Lowell Center for Sustainable Production, University of Massachusetts, Lowell, MA
 - 2003-2006 Member, Human Subjects Committee, Harvard School of Public Health
 - 2003-2006 Advisory Committee, Occupational Health Services Research Program, Harvard School of Public Health
 - 2006 Study Section Review Committee, Pilot Project Program, Graham Environmental Sustainability Institute, School of Natural Resources and Environment, University of Michigan
 - 2006-2007 Chair, Planning Committee, Health Sector, May 8-10, 2007 National Summit on Coping with Climate Change, University of Michigan
 - 2007-2009 Member, Advisory Committee, SPH Practice Committee, University of Michigan
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School of Public Health

2007-2012 Member, Residency Advisory Committee, General Preventive Medicine Residency, University of Michigan School of Public Health

2008-2009 Member, Steering Committee, NIA T32 Training Grant on Aging Research (PI: Mary Haan), University of Michigan School of Public Health

2008-2013 Member, Advisory Committee, Outstanding New Environmental Scientist Awardee (Marie O'Neill), NIEHS

2008-2009 Member, Search Committee for Director of the Risk Science Center, University of Michigan School of Public Health

2009 Co-Chair, Planning Committee, Workshop on Predicting and Preventing Climate Change Impacts on Public Health, Goa, India (Collaboration with the UM Center for Global Health, the US Centers for Disease Control and Prevention, and the Indian Council for Medical Research)

2009-2011 Director and PI, NIA T32 Training Grant on Aging Research, University of Michigan School of Public Health

2009-2010 Member, Planning Committee, University Research Corridor (U of M, Michigan State, Wayne State) symposium on environmental health sciences in January 2010

2009-2012 Faculty Associate, Center for Global Health, University of Michigan

2009-2012 Member, Internal Advisory Board, Cancer Epidemiology Education in Special Populations Program, University of Michigan School of Public Health

2009-2011 Chair, Steering Committee on Global Health, University of Michigan School of Public Health

2010-2012 Member, Executive Committee, Graham Environmental Sustainability Institute, University Of Michigan

2010-2012 Member, Committee on Diversity, University of Michigan School of Public Health

2012-2017 Chair, Executive Committee, Dalla Lana School of Public Health, University of Toronto

2012-2017 Chair, Tenure Committee, Dalla Lana School of Public Health, University of Toronto

2012-2017 Chair, Decanal Promotions Committee, Dalla Lana School of Public Health, University of Toronto

2012-2017 Chair, Executive Advisory Committee, Institute for Global Health Equity & Innovation, Dalla Lana School of Public Health, University of Toronto

2013-2015 Interim Director, Institute for Global Health Equity & Innovation, Dalla Lana School of Public Health, University of Toronto

2013-2014 Co-Chair, Research Committee, Dalla Lana School of Public Health, University of Toronto

2014-2017 Chair, Executive Advisory Committee, Institute for Health Policy Management and Evaluation, University of Toronto

2014 Chair, Ad-hoc Committee to create an Institute for Indigenous Health (based on a \$10 million endowment gift made to DLSPH), Dalla Lana School of Public Health, University of Toronto; Chair, Executive Advisory Committee beginning 2015

2015-2017 Chair, Executive Advisory Committee, Joint Centre for Bioethics, University of Toronto

2015-2018 Chair (2015-2017); Member (2017-2018), Taskforce on Environmental Health, Ministry of Health and Longterm Care, Province of Ontario

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- 2016-2017 Chair, Executive Advisory Committee, Centre for Critical Qualitative Health Research, University of Toronto
- 2017-2018 Executive Co-Chair, Workshop on the Global Burden of Disease-Pollution and Health Initiative (a collaboration between the Global Alliance on Health and Pollution and the Institute for Health Metrics), Seattle, WA
- 2020-2024 Chair, Executive Committee, Department of Population and Public Health Sciences, Keck School of Medicine, University of Southern California, Los Angeles, CA
- 2020-2023 Co-Leader, Population Health COVID-19 Pandemic Research Center, Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, CA
- 2020-2023 Member, Public Health Advisory Committee, Office of the Provost, University of Southern California, Los Angeles, CA
- 2020- Member, Presidential Working Group on Sustainability, University of Southern California, Los Angeles, CA
- 2022-2023 Member, Search Committee for the Chair of the Department of Medicine, Keck School of Medicine, University of Southern California, Los Angeles, CA
- 2022- Co-Leader, Sustainability and Healthcare Research Initiative, University of Southern California, Los Angeles, CA
- 2023-2024 Co-Organizer and Co-host, the USC Symposium of Global Health, Satellite Session on March 7, 2024 to the Annual Meeting of the Consortium of Universities for Global Health, Los Angeles, CA
- 2023- Director, Dhablania and Kim Family Global Medicine and Health Fellowship, Keck School of Medicine, University of Southern California, Los Angeles, CA

Hospital

- 1982-1985 Occupational Safety and Health Committee, Member, Boston City Hospital, Boston
- 1983-1984 House Officers Association, Treasurer, Boston City Hospital
- 1984-1985 House Officers Association, Co-President, Boston City Hospital

OTHER PUBLIC SERVICE

- 1987 Member, Fact-finding tour on "The Health Effects of Massive Exposure to Tear Gas", Seoul, South Korea, July 11-18 (Sponsored by Physicians for Human Rights, American College of Physicians)
- 1988 Member, Fact-finding tour on "Chemical Weapons and the Iraqi Kurdish refugees", Turkey Oct 6-16 (Sponsored by Physician for Human Rights and the MacArthur Foundation)
- 1990 Leader, Fact-finding tour on "Health and Human Rights in Burma (Myanmar)", Thailand-Burma Dec. 26-Jan 6 (Sponsored by Physician for Human Rights and the MacArthur Foundation)

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- 2009 Consultant and senior advisor, Fact-finding tour on “Mining and Potential Exposures and Health Effects in Guatemala”, August 2009 (Sponsored by Physicians for Human Rights)

CONSULTING POSITIONS

- 1987-1989 Consultant, "In-Vivo Total Body Lead Analysis by X-Ray Fluorescence", NIH/SBIR Grant 2R44ES03918-02
- 1988-1989 Consultant, "Boston Area Health Coalition Demonstration Project", DHHS/MP000003-A1
- 1993-1995 Consultant, Employee Health Services, Brigham and Women's Hospital
- 1994 Consultant, Public Welfare Foundation, Washington, DC (review of Environmental Programs)
- 1997-2006 Consultant, Pediatric Environmental Health Center, Children's Hospital, Boston, MA
- 2000 Consultant, Doris Duke Foundation, New York, NY (review of potential Environment and Medicine programs)
- 2009-2010 Consultant and Member, Academic Program Review Site Visit Committee, Department of Environmental and Occupational Health Sciences, University of Washington School of Public Health, Seattle, WA
- 2011 Consultant, JPB Foundation, New York, NY (review of Environmental Health programs)
- 2014-2016 Advisor, Hearing Health Sciences, Ann Arbor MI and Amsterdam, Netherlands
- 2020 Consultant on Environment, Pollution and Health, United Nations Environment Programme, Nairobi, Kenya

VISITING PROFESSORSHIPS

- 1997 Alice Hamilton Visiting Professor, University of California at San Francisco
- 2000-2001 Visiting Professor, Sri Ramachandra Medical College & Research Institute, Chennai, India
- 2004 Visiting Professor, Department of Environmental Medicine, University of Rochester
- 2013 Visiting Professor, Shanghai Key Laboratory, Shanghai Jiao-Tung University

SEMINARS AND EXTRAMURAL INVITED PRESENTATIONS (last 15 years, since 2009; prior presentations upon request)

- 2009 Speaker, “Evidence for Lead as an Environmental Stressor of Alzheimer's Disease and the Role of Epigenetics”, Symposium Panel, Annual Meeting of the Society for Toxicology, Baltimore, MD
- 2009 Keynote Speaker, “Lead, Late-Life and Early Life Effects, and the Emerging Field of Environmental Epigenetics: Looking Ahead”, Annual Meeting of the American College for the Advancement of Medicine, San Diego, CA

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- 2009 Speaker, "Lead Toxicity and Mechanistically-Oriented Molecular Epidemiology: Targeting the Epigenetics of Alzheimer's Disease", Seminar Series, Institute for Environmental Health Sciences, Wayne State University, Detroit, MI
- 2009 Speaker, "Climate Change Impacts on Health in the Developing World", Research Discussion Series, University of Michigan Center for Global Health
- 2009 Speaker, "Autism, Aggressive Behavior, Anxiety, and Alzheimer's: are Environmental Toxicants Playing a Major Etiologic Role?", Department of Psychology, University of Michigan
- 2009 Speaker, "Early Life Exposures and Endocrine Disruption: Evidence from Molecular Epidemiology", Pediatric Endocrine Seminar, University of Michigan Medical School
- 2009 Distinguished Speaker, "Lead Toxicity: Twenty Years of Research On The Poison That Keeps on Poisoning" 10th Anniversary of the Department of Microbiology and Environmental Toxicology, University of California at Santa Cruz
- 2010 Speaker, "The Centers for Disease Control and Prevention & the Environmental Protection Agency: Potential Funding Opportunities for Regional Collaboration in Michigan", University Research Corridor Symposium on Environmental Health, Detroit, MI.
- 2010 Speaker, "The Future of Public Health", University of Washington School of Public Health
- 2010 Speaker, "The Environment Meets the Epigenome: Is This Where Autoimmunity Begins?" Symposium on Autoimmunity and Epigenetics, University of Michigan
- 2010 Keynote Speaker, "A New Twist to an Old Story: The Evidence for Early Life Lead Exposure as a Risk Factor for Alzheimer's Disease through Epigenetic Programming", NIEHS Environmental Health Sciences Center and Toxicology Training Program Retreat, University of Rochester, NY
- 2010 Speaker, "Lead Toxicity: Twenty Years of Research on The Poison That Keeps on Poisoning" and "Environmental Health Sciences at the University of Michigan", Tianjin Centers for Disease Control, Tianjin, China
- 2010 Speaker, "Pediatric Lead Toxicity", Xinhua Hospital and the Shanghai Jiao-Tung Medical University Department of Pediatrics, Shanghai, China
- 2010 Speaker, "Environmental Health Sciences at the University of Michigan", Fudan University, Shanghai, China
- 2010 Speaker, "Alzheimer's Disease, Epigenetics and the Environment", Symposium Update, Alzheimer's Disease Association, Ann Arbor, MI
- 2010 Speaker, "Environmental Justice, Progress (and the Lack Thereof) and the Role of Research", Roundtable on Environmental Health Sciences, Research and Medicine, Institute of Medicine, National Academies, Washington DC.
- 2010 Speaker, "White Coats, Population Science and Poison Gas: A Life Spent at the Intersection of Academic Medicine, Global Health & Human Rights", Robert Wood Johnson Clinical Fellows Program, University of Michigan Medical School, Ann Arbor, MI
- 2011 Speaker, "The Three Most Difficult Challenges to Molecular Epidemiologic Research on Gene-Environment Interactions: Lead Toxicity as a Case Study." Department of Human Genetics, University of Michigan Medical School, Ann Arbor, MI
- 2011 Speaker, "The Integration of Data on Environmental Carcinogens with Population and Genetic Resources", "Opportunities & Challenges for Translational Research on Cancer

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- Prevention”, Translational Cancer Prevention & Biomarkers Workshop, Mazamdur-Shaw Cancer Center, Bangalore, India.
- 2011 Speaker, “Success in the Academy”, Faculty Panel, Students of Color of Rackham, Rackham Graduate School, University of Michigan
- 2011 Speaker, “White Coats, Population Science and Poison Gas: Fact-Finding Missions by Health Professionals for Human Rights”, Sujal Parikh Memorial Symposium, University of Michigan Medical School.
- 2011 Speaker, “The Analysis of Biomarker Data to Ascertain the Contribution of Environmental Exposures to the Etiology of Disease: Lead Exposure and Toxicity as a Case Study”, Department of Computational Medicine and Bioinformatics, University of Michigan Medical School.
- 2012 Speaker, “Research and Analysis Linking Upstream and Downstream Disparities Work”, Webinar hosted by the Health & Environmental Funders Network, Bethesda, MD, with 52 Foundations related Health.
- 2012 Keynote Speaker, “The Future of Public Health & Medicine in a Crowded, Diverse, Stratified, Hot, Urbanized, Polluted, Thirsty, Hungry and Debt-Ridden World”. E.J. Van Lier Memorial Convocation and Health Sciences Center Research Day, West Virginia University, Morgantown, West Virginia
- 2012 Plenary Speaker, “Transgenerational Impacts of Pollutants on Offspring: Recent Insights and Case Studies”, Connaught Global Challenge International Symposium, University of Toronto.
- 2012 Speaker, “Environmental Impacts on Aging (+ an update on the Dalla Lana School of Public Health)”, Community Medicine Rounds, University of Toronto
- 2012 Speaker, “The Environment & Public Health in a Research-Intensive University: Opportunities for Scholarship in a Crowded, Diverse, Stratified, Hot, Urbanized, Polluted, Thirsty, Hungry and Debt-Ridden World”, School for the Environment, University of Toronto
- 2012 Speaker, “Big Public Health Challenges (& Opportunities) in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World”, External Advisory Meeting, Public Health Ontario, Toronto
- 2012 Speaker, “Canadian Public Health Schools (in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World): The View from Toronto, External Advisory Board Meeting, Institute for Population and Public Health, Canadian Institutes for Health Research, Toronto
- 2012 Speaker, “Sustainable Development and Health: The Global Mining Industry”, Canadian Society for International Health Annual Meeting, Ottawa
- 2012 Speaker, “Big Public Health Challenges (& Opportunities) in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World”, Xinhua Hospital/Shanghai Jiao-Tung University, Shanghai, China.
- 2012 Speaker, “The Impact of Population-Wide Lead Exposure and Gene-Lead Interactions on Chronic Disease”, Genetic Grand Rounds, Sick Kids Hospital, Toronto.
- 2012 Speaker, “Looking behind the curtain: Lead Toxicity as a Case Study of Methodologic Challenges in Gene-Environment Interactions Research”, Strategic Training in Advanced

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- Genetic Epidemiology (STAGE), Dalla Lana School of Public Health, University of Toronto.
- 2012 Keynote speaker: “Public Health—the Next Frontier in Health Professions Education”. Council of Health Sciences annual retreat, University of Toronto.
- 2013 Speaker, “White Coats, Population Science and Poison Gas: Lessons from a Life Spent at the Intersection of Academic Medicine, Global Health & Human Rights”, Joint Center for Bioethics, University of Toronto
- 2013 Speaker, “Gauging environmental impact on the development of chronic inflammation”, Connaught Global Challenge Workshop, University of Toronto.
- 2013 Speaker, “The Future of Public Health & Medicine in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World”, Grand Rounds, Department of Medicine, University of Toronto.
- 2013 Speaker, “Metals, Mega-trends, and Me: Reflections on Research and the Vision for the Dalla Lana SPH”, Occupational and Environmental Medicine Grand Rounds, St. Michael’s Hospital, Toronto, ON.
- 2013 Speaker, “Air pollution and Cardiovascular Disease: Health Impacts, Mechanisms, and Research Opportunities”, University of Toronto & FMUSP-InCor Symposium on Cardiology, Sao Paulo, Brazil.
- 2013 Speaker: “Lead Exposure’s Impact on Health and Policy: A History of Neglect and Missed Opportunities”, Public Health Policy Rounds, CIHR Strategic Training Program in Public Health Policy, University of Toronto.
- 2013 Speaker: “Lead Toxicity: The Long Tail of Health Impacts (and On-going Research Opportunities!) From an Historical Environmental Air Pollutant”, Southern Ontario Centre for Air Pollution and Aerosol Research, University of Toronto.
- 2013 Speaker: “Water and Sanitation”, Water, Sanitation and Hygiene (WASH) Canada, Toronto, Ontario, Canada
- 2014 Speaker: “Conflict and Public Health”, Ontario Medical Association, Toronto, Canada
- 2014 Panelist: “Judging Evidence: Finding a Place for Variation in an Evidence-Based World”, Health Quality Ontario, Toronto, Canada
- 2014 Speaker: “The Grand Convergence: Creating Health in a Globalized World”, Special meeting of the Canadian Chamber of Commerce in Shanghai
- 2014 Speaker: “The Grand Convergence: Creating Health in a Globalized World”, Jockey School of Public Health and Primary Care, Chinese University of Hong Kong, Hong Kong, China
- 2015 Speaker: “The Grand Convergence: Creating Health in a Globalized World”, School of Public Health and the ASEAN Institute, Mahidol University, Bangkok, Thailand
- 2015 Speaker: “Gene-environment Interactions and the Role of Big Data in Environmental Health” Seminar series, School of the Environment, University of Toronto, Toronto, Canada
- 2015 Speaker: “Global Health Security”, Ill with Illness—Economic, Social & Security Barriers to the Provision of Global Health, Munk School of Global Affairs, University of Toronto, Toronto, Canada
- 2015 Speaker: “The Dalla Lana School of Public Health: Big Ideas and Initiatives for Creating Health in a Globalized World”, Speaker Series, University of Toronto Alumni of Toronto.
- 2015 Speaker: “Unique Scientific Opportunities for the Precision Medicine Initiative National Research Cohort: Exposomics, Data Linkage, and Global Collaborations”. Working group on

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- President Obama's Precision Medicine Initiative (Chaired by Francis Collins, Director, NIH)
- 2015 Speaker: "What is the Role of Schools of Public Health in the 21st Century?" 50th Anniversary Celebration of the Department of Epidemiology, Biostatistics and Occupational Health, McGill University, Montreal, Quebec.
- 2015 Welcoming Address: "Global Public Health and Mental Health", Going Global for Mental Health conference, Centre for Addictions and Mental Health/Department of Psychiatry/Dalla Lana School of Public Health, Toronto, ON
- 2015 John Goldsmith Memorial Lecture: "Big Data, Environmental (and Social) Epidemiology, Power and Politics", Opening Plenary Session, International Society for Environmental Epidemiology Annual Meeting, Sao Paulo, Brazil
- 2015 Inaugural Speaker: "The Future of Public Health and Medicine in a Crowded and Complex World", Global Health Leadership Series, PSG Medical School & the Shanti Ashram Foundation, Coimbatore, Tamil Nadu, India
- 2016 Speaker "The Future of Public Health & Medicine in a Crowded, Diverse, Aging, Stratified, Urbanized, Polluted, Hot, Thirsty, Hungry, Debt-Ridden World", Indian Institutes of Public Health—Hyderabad, Hyderabad, India
- 2016 Speaker: "Integration of Public Health & Health Care: The Unmet Agenda for a Truly Sustainable Health System", Board of Directors Retreat, Toronto East General Hospital, Toronto
- 2016 Plenary speaker: "Health Promotion, Prevention and Health Protection: Innovative Initiatives", 6th Asia-Pacific Conference on Public Health | 1st ASEAN Health Promotion Conference Bangkok, August
- 2016 Speaker: "Big Data, Environmental (and Social) Epidemiology, Power and Politics", Mount Sinai School of Medicine, New York, NY
- 2016 Plenary Speaker: "The Impact of Environmental Toxicants on Health: Recent Epidemiologic Approaches & Advances", International College of Integrative Medicine Annual Meeting, Toronto, ON
- 2016 Plenary Speaker: "Big Data and Implications for Environmental Health", 15th Anniversary Conference, Jockey Club School of Public Health & Primary Care, Chinese University of Hong Kong, Hong Kong
- 2016 Plenary Speaker: "Innovations in Assessing Lead Poisoning and Child Health: Policy & Clinical Implications", Chinese University of Hong Kong-Fudan-Oxford International Symposium on Health Impacts of Environmental Exposures", Hong Kong
- 2016 Speaker: "Addressing a Changing Environment (and Impacts on Health, AKA Can India Survive Modernization?)", Indian Institutes of Technology Alumni, Canada, International Conference 2016, Toronto.
- 2016 Plenary Speaker, "Hidradenitis Suppurativa: Research Directions from a Population Health Perspective", Symposium on Hidradenitis Suppurativa Advances, Toronto.
- 2016 Plenary Speaker, "Children's Environmental Health", The 2016 Annual National Conference on Children's Healthcare, Shanghai, China
- 2016 Special Guest Speaker, "Big Data, Environmental (and Social) Epidemiology, Power and Politics", Shanghai Municipal Center for Disease Control, Shanghai, China
- 2016 Lecturer, "Lead and Human Health: Recent Research and Associated Lessons for Science & CV: Howard Hu, M.D., M.P.H., Sc.D.

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- Policy”, Fudan University School of Public Health, Shanghai, China
- 2017 Lecturer, “The Impact of Environmental Toxicants on Health: Recent Epidemiologic Approaches & Advances”, Saw Swee Hock School of Public Health, National University of Singapore, Singapore
- 2017 Lecturer, “The Future of Academic Public Health”, Saw Swee Hock School of Public Health, National University of Singapore, Singapore
- 2017 Lecturer, “Recent Advances in Understanding, Preventing, and Reversing the Impact of Environmental Factors on Health”, Society of Chinese Bioscientists in America, Li Ka Shing Knowledge Institute, St. Michael’s Hospital, Toronto, ON
- 2017 Lecturer, “Environmental Epidemiology in the Era of Exposomics, Lifecourse Epidemiology, Big Data and Big Science”, Department of Environmental Health, Harvard School of Public Health, Boston, MA
- 2017 Speaker, “The Role of a Re-emergent Canadian School of Public Health in a Hot, Hungry, Polluted, Aging, Polarized World Prone to Pandemics, Chronic Disease, and Unsustainable Health Systems”, Royal Canadian Institute for Science, Toronto, ON
- 2017 Speaker, “The Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) Birth Cohort Study: Current Research on Fluoride and Neurodevelopment”, Seminar Series in Environmental Epidemiology, University of Washington School of Public Health, Seattle, WA
- 2017 Plenary Speaker: “New realities arising from the extractive industries and agri-business: the Pollution and health perspective,” Hong Kong Summit of Global Health Leaders. University of Hong Kong, Hong Kong
- 2018 Plenary Speaker: “The GBD-Pollution and Health Initiative: Challenges & Opportunities”, Workshop on the Global Burden of Disease-Pollution and Health Initiative, Institute for Health Metrics, University of Washington, Seattle, WA
- 2018 Guest Lecturer: “Partnerships, Local Responsiveness, National and Global Impacts”, University of Iowa College of Public Health, Iowa City, IA
- 2018 Plenary Speaker: “Current Research on Fluoride and Neurodevelopment: The Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) Birth Cohort Study”, Annual meeting of the International Academy of Oral Medicine and Toxicology, Denver, CO
- 2018 Speaker, “Recent Epidemiologic Research on Lead Toxicity: New Surprises regarding an Old Global Pollutant”, Department of Environmental and Occupational Health Sciences Seminar Series, University of Washington School of Public Health, Seattle, WA
- 2018 Speaker: “The Early Life Exposures in Mexico to Environmental Toxicants (ELEMENT) Birth Cohort Study: Current Research on Fluoride and Neurodevelopment”, Symposium on Fluoride research, Annual meeting of the International Society for Environmental Epidemiology/International Society for Exposure Science, Ottawa, ON
- 2018 Panelist, “The Fluoridation Decision: Considering the Evidence for Benefits, Possible Risks as well as Ethical World Views”, Annual meeting of the International Society for Environmental Epidemiology/International Society for Exposure Science, Ottawa, ON
- 2018 Invited speaker: “Grand Opportunities”, The UC-Irvine School of Population Health and the Samueli College of Health Sciences, Irvine, CA
- 2018 Invited speaker, “The Global Burden of Disease-Pollution and Health Initiative”, Office of
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- The Director and the Global Environmental Health Program, U.S. National Institute for Environmental Health Sciences, Research Triangle Park, NC
- 2019 Invited speaker, “Evaluating, treating and managing disabilities of patients with chemical intolerance”, Symposium on Chemical Intolerance—A Way Forward, Marilyn Brachman Hoffman Foundation and the Hoffman Program on Chemicals and Health at the Harvard T.H. Chan School of Public Health, Dallas, TX
- 2019 Invited Lecturer: “The Global Burden of Disease-Pollution and Health Initiative”, Center for Population Health Sciences, Stanford University, Palo Alto, CA
- 2019 Invited Lecturer: “Lead and Fluoride: Old and New Toxicant Issues and the Global Burden of Disease”, British Columbia Centre for Disease Control, Vancouver, BC, Canada
- 2019 Invited Lecturer: “Lead and Fluoride: Old and New Toxicant Issues and the Global Burden of Disease”, University of California, Davis, CA, USA
- 2019 Invited speaker, “A Framework for Adding Environmental Exposure-Outcome Pairs to the Global Burden of Disease: The Global Burden of Disease-Pollution and Health Initiative”, 2019 Annual Meeting of the International Society for Environmental Epidemiology, Utrecht, Netherlands
- 2019 Invited speaker, “The Global Burden of Disease – Pollution and Health Initiative: Impacts on Human Capital”, Air Pollution, Health and Human Capital Nexus in Chinese Cities Scoping Meeting, Institute of Urban Environments, Chinese Academy of Sciences, Xiamen, China
- 2019 Invited Speaker, “Toxic Chemicals, Human Health, and Human Rights”, A Human Right to Health: Pathways and Responses, Seattle University Law School, Seattle, WA
- 2020 Invited Lecturer: “The Herbert Wertheim School of Public Health at UC San Diego: Grand Opportunities.” University of California at San Diego, San Diego, CA
- 2020 Invited speaker and panelist, “Health Effects, Historical and Contemporary Use of Tear Gas and Other Riot Control Agents”, Environmental Exposure Grand Rounds, Minnesota Department of Health, Health Partners, University of Minnesota School of Public Health, Hennepin Regional Poison Center. (Webinar)
- 2020 Plenary symposium speaker: “The Pollution, Climate and Global Burden of Disease Initiative: The Challenge of Estimating Exposures in Countries with Little or No Data”, Annual meeting (virtual) of the International Society for Exposure Science
- 2020 Invited Speaker, “The Pollution, Climate and Global Burden of Disease Initiative”, the Centre for Air Pollution, Energy and Health Research (CAR), University of Sydney, Australia (Webinar)
- 2020 Invited speaker, “The ELEMENT birth cohort study, and the Global Burden of Disease-Pollution, Climate and Health Initiative: Two Opportunities for New Collaborations”, The NIEHS P30 Southern California Environmental Health Sciences Center, University of Southern California (Webinar)
- 2020 Presenter, “Sustainability and Population Health: Ideas for an Agenda at USC, Presidential Working Group on Sustainability, University of Southern California
- 2020 Invited speaker, “The Global Burden of Disease—Pollution, Climate and Health Initiative: A Focus on the Potential Role of Spatial Sciences”, the Spatial Sciences Institute, University of Southern California

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- 2020 Invited speaker, “Environment Exposures, Epigenetics, Epidemiology, Etiology, and Cancer: Which “E” is the Weak Link?” Norris Comprehensive Cancer Center, University of Southern California.
 - 2021 Presenter, “Update on the Department of Preventive Medicine”, Basic Science and Clinical Chairs Council, Keck School of Medicine, University of Southern California
 - 2021 Invited Speaker, “The changing nature of population health management: The Population Health Perspective”, Annual State of Reform Southern California Health Policy Conference, San Diego, CA
 - 2021 Invited Speaker, “Update and Work on COVID-19, Health Inequities, and Social Justice”, the 31st meeting of the California Public Health/Prevention Medical Leadership Forum.
 - 2021 Invited Speaker, “The Department of Population and Public Health Sciences: Update and Our Work on Fast- and Slow-Moving Population Health Crises”. Keck School of Medicine’s 2021 Alumni Day CME program.
 - 2022 Invited Speaker, “Lead Exposure and Non-communicable Diseases”, The Impact of Lead Pollution on NCDs Webinar, co-sponsored by the Global Alliance on Health and Pollution, and the World Federation of Public Health Associations.
 - 2022 Invited Speaker, “Environmental Risk Factors for Diabetes and Obesity: Endocrine-Disrupting Chemicals and the Built Environment”, USC Diabetes and Obesity Research Institute’s 9th Annual Research Symposium, Los Angeles, CA.
 - 2022 Invited Speaker, “Long-lived Endocrine Disrupting Chemicals: Update on the Epidemiology”, California Coastal Chloro-Contamination Conference. UC Santa Barbara, Santa Barbara, CA
 - 2022 Invited Speaker, “The Lancet Commission on Pollution and Health: Progress Update”, New York University School of Global Public Health, virtual briefing hosted by the Global Alliance on Health and Pollution
 - 2022 Invited Speaker, “Hot Spots of Toxic Pollution in Kenya, Senegal and Tanzania (2016-2020): Data from the Toxic Sites Identification Program conducted by Pure Earth. International Society for Environmental Epidemiology Africa regional meeting (virtual).
 - 2023 Presenter, “Long-term effects of prenatal fluoride and lead exposure on educational delay in Mexico”, Annual Meeting of the International Society for Environmental Epidemiology, Kaohsiung, Taiwan
 - 2023 Invited Speaker, “Sustainability and Our Profession: Decarbonizing the Healthcare Industry”, Grand Rounds, Kaohsiung Medical University, Kaohsiung, Taiwan
 - 2023 Invited Speaker, “The USC Department of Population and Public Health Science”, Department of Occupational & Environmental Health, National Taiwan University, Taipei, Taiwan.
 - 2023 Invited Speaker, “The impact of lead on non-communicable diseases: Why we should be concerned”. Bloomberg Philanthropies, New York, NY
 - 2023 Invited Speaker, “Brain Health and Aging-Habits, Prevention and Innovative Treatment”, USC Keck School of Medicine Alumni & Reunion event, Los Angeles, CA
 - 2024 Invited Speaker, “Sustainability and Our Profession: Decarbonizing the Healthcare Industry”, Mid-Year Event, Patient Safety Movement, Irvine, CA
 - 2024 Invited Speaker, “A Ministerial Dialogue: Preventing Maternal and Child Exposure to Toxic
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- Lead, 77th session, World Health Assembly, United Nations, Geneva, Switzerland
- 2024 Invited Keynote Speaker, “The Global Burden of Disease (and the Stark Associated Inequities) from Pollution and Climate Change: A Snapshot of Insights and Progress on Solutions”, Los Angeles Global Health Conference, UCLA.
- 2024 Invited Speaker, “Early lead exposure and its lasting impacts on cognitive development and health outcomes”, Addressing Lead Poisoning in South Asia: Impact, Challenges and Policy Solutions; Sanrachna Foundation, Advanced Study Institute of Asia at SGT University, Gurugram, India
- 2024 Invited Speaker, “Lead Exposure and Its Lasting Impacts on Children’s Brain Development and Adult Cardiovascular Disease”, International Convening on Lead Poisoning in India The Pahle Foundation, New Delhi, India
- 2024 Invited Speaker, “The Impact of Lead Exposure on Children’s Brain Development and Adult Cardiovascular Disease, and the Indian Context”, Roundtable, the World Bank, Delhi, India
- 2024 Invited Speaker, “Lead in Bone and Blood: An Update on Biomarkers & Their Significance”, Assessing Lead Impacts: Effects on People and Society, Montefiore Medical Center, A New York State Regional Lead Resource Center, Bronx, NY
- 2024 Invited Speaker, “Fluoride and Neurodevelopment: the Evidence, and the Tension between Dental Public Health and Environmental Health”, Department of Occupational and Environmental Health, Colorado School of Public Health, Denver, CO

INVENTIONS/PATENTS: n/a

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Abstracts of Work (Upon request)

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Appendix B

Howard Hu, M.D., M.P.H., Sc.D.

Consultant in Occupational and Environmental Medicine, Internal Medicine, and Epidemiology¹

Depositions, Trials, in US federal, state, or county court cases, the last 5½ years (2019-present, as of November 26, 2024)

DESCRIPTION: In each of these cases, Dr. Hu served as either a consultant in occupational and environmental medicine & epidemiology who evaluated a particular individual or individuals and rendered an expert opinion on general causation and/or specific causation, and/or as a consultant in occupational and environmental medicine & epidemiology who reviewed the literature on a particular issue and rendered a scientific opinion on general causation.

DATE	TYPE	CASE	CLIENT
5/16/19	Deposition	A.O.A., et al. v. Doe Run Resources Corporation, et al., Case No. 4:11-CV-00044-CDP	Schlichter, Bogard & Denton, LLP, St. Louis, MO
9/16/19-9/17/19	Deposition	Don Strong et al., v. Republic Services, Inc., et al.	Humphrey, Farrington & McClain, P.C., Independence, MO
9/24/19	Deposition (non-retained fact witness)	Food & Water Watch, Inc., et al, v. United States Environmental Protection Agency (US EPA), et al.	(testified on the work and results of my epidemiologic research team on the potential neurodevelopmental impacts of fluoride, in response to subpoena from the U.S. EPA., arranged by Waters Kraus Paul, P.C., Segundo, CA)
10/9/19	Deposition	Pamela Butler, et al. v. Mallinckrodt, Inc., et al.	Humphrey, Farrington & McClain, P.C., Independence, MO
2/8/20	Trial	USA v. Gary Spengler, M.D.	Oberheiden & McMurrey, Dallas, TX 75240
6/8/20	Trial (non-retained fact witness)	Food and Water Watch v US EPA	(see above)
8/6/20	Deposition	A.O.A. et al. v. Doe Run Resources Corp.	Schlichter, Bogard & Denton, LLP
8/31/20 and 9/1/20	Deposition	Marc Czapla and Jill Czapla v. Republic Services, Inc et al.	Humphrey, Farrington & McClain, P.C., Independence, MO
10/12/20 and 11/5/20	Deposition	Flint Water Cases, Civil Action No. 5:16-cv-10444-JEL- MKM	Weitz & Luxenberg P.C. 220 Lake Drive East, Suite 210 Cherry Hill, NJ
6/8/22 and 6/9/22	Deposition	Teresa Fornek v. Sterigenics, LLC et al.	Smith LaCien, LLP, Chicago, IL

¹ Current academic position, as of July 1, 2020: Professor and the Flora L. Thornton Chair of the Department of Population and Public Health Sciences, Keck School of Medicine, University of Southern California, Los Angeles, CA.

7/1/22	Deposition	Susan Kamuda v. Sterigenics, LLC et al.	Salvi, Schostok & Pritchard P.C., Chicago, IL
7/21/22	Deposition	Heather Schumacher v. Sterigenics LLC et al.	Romanucci & Blandin, LLC, Chicago IL
9/9/22	Deposition	Teresa Fornek v. Sterigenics LLC, et al.	Smith LaCien, LLP, Chicago, IL
10/27/22	Trial	Teresa Fornek v. Sterigenics LLC, et al.	Smith LaCien, LLP, Chicago, IL
11/21/22	Deposition	Flint Water Cases, Civil Action No. 5:16-cv-10444-JEL- MKM	Weitz & Luxenberg P.C. 220 Lake Drive East, Suite 210 Cherry Hill, NJ
2/21/23	Deposition	Kevin Wright v. UNOCAL, UNION OIL, et al.; CASE NO.: 21CV00925	Erin L. Powers, Trial Attorney, 548 Market St PMB 66906, San Francisco, CA 94104
3/29/23	Deposition	Bryan Dick-Ipsen v. Tri-Supply Co., et al.; Cook County, IL; Case No: 2018 L 011367	Jeffrey J. Lowe; Carey Danis & Lowe; 8235 Forsyth Suite 1100, St. Louis, MO 63105
5/16/23	Trial	Kevin Wright v. UNOCAL, UNION OIL, et al.; CASE NO.: 21CV00925	Erin L. Powers, Trial Attorney, 548 Market St PMB 66906, San Francisco, CA 94104
10/13/23	Deposition (non-retained fact witness)	Food & Water Watch, et al. v. U.S. EPA, et al.	See above
1/31/24	Trial (non-retained fact witness)	Food & Water Watch, et al. v. U.S. EPA, et al.	See above
7/3/24	Deposition	FTCA Flint Water Cases, Civil Action No.: 4:17-cv-11218 (consolidated)	Law Offices of Deborah A. LaBelle, 221 North Main Street Suite 300, Ann Arbor, MI 48104

Appendix C

*Howard Hu, M.D., M.P.H., Sc.D.
Occupational/Environmental Medicine, Internal Medicine, and Epidemiology
Professor of Preventive Medicine (Department Chair, 2020-2024)*
Department of Population and Public Health Sciences
Keck School of Medicine, University of Southern California*

*Consultant Address: 3363 Monterosa Drive, Altadena CA, 91001, USA
Consultant Email: howardhu2225@gmail.com*

Consulting rates, as of July 1, 2024

Pre-deposition and pre-trial work (reviewing documents, analyzing data, preparing reports, communications, etc.)

\$700 US/hr

Deposition testimony

\$1,200 US/hr

Trial testimony (at relevant location)

\$10,000 US/day + travel expenses

Travel: \$200 US/hr (door to door)

NOTE: Payment to be submitted as a check by mail or wire transfer to a U.S. bank account

* For identification and affiliation purposes only.