Exhibit 495

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Re: Ronald Lee Carter
DOB: [redacted]1948; DOD: 5/9/2022
Rebuttal to report of Dr. Lisa A. Bailey

I am writing in response to your request for a response to the April 8, 2025 expert report provided by Lisa Bailey, Ph.D. that relates to Mr. Ronald Carter's cancer and its relationship to exposures incurred at Camp Lejeune. My rebuttal report does not contain a response to all of the points that Dr. Bailey makes in her report with which I disagree, nor should my rebuttal to some portions of her report and not others be viewed as agreement with the portions of her report that I do not rebut below.

Please refer to my February 7, 2025 report on Mr. Ronald Carter for a summary of my background and qualifications as well as my general causation report of December 9, 2024.

I offer the following comments, each preceded by a reference to the associated location in Dr. Bailey's report:

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(1) Page 18-20: In these pages, Dr. Bailey criticizes the non-threshold assumption with regards to level of exposure to a carcinogen and level of increased risk of cancer. Among her arguments: (A) Dr. Bailey cites the example of a chemical that is not genotoxic, but that can cause cytotoxicity (which typically requires a relatively high dose), thereby liberating oxygen radicals that can react with DNA and cause mutations and cancer; (B) Dr. Bailey argues that a non-threshold mechanism of carcinogenesis implies that any level of exposure can theoretically result in a mutation that can result in carcinogenesis, and states such a mechanism "*…is not biologically plausible, even for carcinogens that are known to react direct with DNA*". Three articles are cited in support of this assertion, i.e., Cardarelli and Ulsh (2018[2]), Golden et al. (2019[3]), and Calabrese (2023[4]).

Comment: I would agree with Dr. Bailey's point (A), i.e., that there are some chemicals that are not genotoxic but could be considered as carcinogenic based on a mechanism such as cytotoxicity and the release of oxygen radicals, a process that typically requires a non-trivial level of chemical exposure. However, as reviewed in my discussion of general causation, all 3 chemicals that I

[1] Affiliation listed for identification purposes only.
[2] Cardarelli JJ 2nd, Ulsh BA. It Is Time to Move Beyond the Linear No-Threshold Theory for Low-Dose Radiation Protection. Dose Response. 2018 Jul 1;16(3):1559325818779651. doi: 10.1177/1559325818779651. PMID: 30013457; PMCID: PMC6043938.
[3] Golden R, Bus J, Calabrese E. An examination of the linear no-threshold hypothesis of cancer risk assessment: Introduction to a series of reviews documenting the lack of biological plausibility of LNT. Chem Biol Interact. 2019 Mar 1;301:2-5. doi: 10.1016/j.cbi.2019.01.038. Epub 2019 Feb 12. PMID: 30768967.
[4] Calabrese, EJ. 2023. "Dose-response: A fundamental concept in toxicology." In Hayes' Principles and Methods of Toxicology (Seventh Edition). (Eds.: Hayes, AW; Kobets, T), CRC Press, Boca Raton, FL. p95 141. doi: 10.1201/9781003390008-4.

discussed—TCE, PCE, and benzene—have evidence of genotoxicity. With regards to point (B), the three papers cited by Dr. Bailey were written by 5 authors well-known to be supporters of the theory of hormesis, i.e., there is a threshold of exposure, for example, for radiation, below which there is either no effect or even beneficial effects. This theory is highly controversial, not backed up by the overall evidence, and not accepted by any authoritative body. As recently summarized in two reviews by 15 senior scientists based in multiple Universities, the US National Cancer Institute, and national agencies in the United States and the United Kingdom[5],[6], the epidemiological evidence, including recent systemic reviews and meta-analyses (e.g. the assessment of 21 studies by Hauptmann et al.[7]), continues to mount indicating that even very low doses of radiation (<0.1 Gy) increase the risk of cancer. In addition, in a 2021 report, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR[8]) reviewed the most recent radiobiological evidence and concluded that "There remains good justification for the use of a non-threshold model for risk inference for radiation protection purposes, given the present robust knowledge on the role of mutation and chromosomal aberrations in carcinogenesis". Overall, based on the totality of the latest scientific evidence relevant to low-dose radiation exposures, the linear no threshold (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 2015[9], the U.S. Nuclear Regulatory Commission[10] in 2021, and independent scientists in Europe[11] as recently as 2023. I agree with this position.

With regards to chemical exposures, as the sophistication of epidemiological studies continues to advance, there are several recent key epidemiological studies that shed light on the risk of cancer posed by very low levels of exposure to chemical carcinogens. (Please see next point).

(2) In pages 20-21, Dr. Bailey outlines her views on how to interpret estimates of risk developed by the U.S. Environmental Protection Agency (EPA) and Agency for Toxic Substances and Disease

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[5] Shore RE, Beck HL, Boice JD Jr, Caffrey EA, Davis S, Grogan HA, Mettler FA Jr, Preston RJ, Till JE, Wakeford R, Walsh L, Dauer LT. Recent Epidemiologic Studies and the Linear No-Threshold Model For Radiation Protection-Considerations Regarding NCRP Commentary 27. Health Phys. 2019 Feb;116(2):235-246. doi: 10.1097/HP.0000000000001015. PMID: 30585971.

[6] Simon SL, Kendall GM, Bouffler SD, Little MP. The Evidence for Excess Risk of Cancer and Non-Cancer Disease at Low Doses and Dose Rates. Radiat Res. 2022 Dec 1;198(6):615-624. doi: 10.1667/RADE-22-00132.1. PMID: 36136740; PMCID: PMC9797580.

[7] 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.

[8] United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), UNSCEAR 2020/2021 Report. Volume III. Annex C. Biological mechanisms relevant for the inference of cancer risks from low-dose and low-dose-rate radiation. New York: United Nations; 2021; E.22.IX.3, 1–238.

[9] 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.

[10] 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.

[11] Laurier D, Billarand Y, Klokov 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.

Registry (ATSDR) in relation to the task of evaluating the potential for causation between an individual's chemical exposure and health effects. Dr. Bailey makes the argument that:

"…given the conservative nature of the regulatory risk calculations, even if there is an exceedance of US EPA's risk target, that does not mean that health effects are likely to occur. Therefore, for a causation analysis, it is also useful to evaluate potential causal relationships by comparing the estimated doses for the individual to doses or exposure information from the health effect studies (animal or human) that are the basis of the toxicity criteria. These relationships are called margins of exposure (MoEs), as discussed in the next section."

Dr. Bailey goes on to define margins of exposure (MOE) as the ratio between the exposure predicted for an individual and the lowest exposure levels at which health effects have been observed (or exposure levels at which no effects have been observed, for some chemicals) in human or animal studies. Her conclusion is that:

"*If the plaintiff's exposures are well below exposures where effects have been observed in epidemiology or toxicology studies, even if there is a risk calculation greater than US EPA's targets, these results provide support that the individual exposures are not likely to be associated with the health effect of concern*."

Comment: I disagree with this conclusion. It is well-known and accepted that both epidemiological studies and toxicology studies (of animals) have practical limits with regards to statistical power. With regards to cancer, negligible risk promulgated by the World Health Organization[12] (less than 1 in 1 million), which is the same as the *de minimis* risk level typically promulgated by the U.S. Environmental Protection Agency for carcinogens of 1 in 1 million[13],[14]. As a result, 5 in a million could be considered a non-negligible risk associated with cancer that is to be avoided. From an epidemiology and statistical power perspective, assuming there is a baseline risk of cancer in a population unexposed to a carcinogen of 1 in a million, an epidemiological cohort study of 5,879,980 individuals would be required to detect a relative risk of 5 with 80% power at a statistical significance of $p<0.05$ (i.e., 2,939,990 individuals per group [exposed to carcinogen and unexposed control population). Few, if any, such cohort studies have been conducted given the enormous expense and logistics required. That is precisely why modeling is conducted using existing epidemiological data to extrapolate actual risk at doses lower than those in which "…effects have been observed in epidemiology or toxicology studies…". A similar limitation exists for toxicology studies of animals, which is why toxicology studies typically expose a relatively small group of animals (e.g., 50 in unexposed and 50 exposed at low dose, 50 at medium dose, 50 at high dose) to very high concentrations of carcinogens, and then use the data to extrapolate the effects at low concentrations.

[12] WHO. *Communicating Radiation Risks in Paediatric Imaging.* Geneva: World Health Organization 2016. ISBN 978 924 4 151034 9.
[13] US EPA. *Residual Risk—Report to Congress.* US Environmental Protection Agency, Office of Air and Radiation; Office of Air Quality Planning and Standards. Research Triangle Park:March, 1999. EPA-453/R-99-001.
[14] Castorina R, Woodruff TJ. Assessment of potential risk levels associated with U.S. Environmental Protection Agency reference values. Environ Health Perspect. 2003 Aug;111(10):1318-25. doi: 10.1289/ehp.6185. PMID: 12896853; PMCID: PMC1241613.

Finally, despite these limitations, it is important to appreciate the prospective study of cancer incidence at Camp Lejeune, where Mr. Carter worked, that were conducted by Bove et al. (2024[15]). Although not a cohort study per se with individual-level data on all subjects, the study design was able to make comparisons with the military and civilian personnel stationed at Camp Pendleton, a population with very similar demographics, and to take advantage of the associated relatively large sample sizes (military personnel: Camp Lejeune, N =154,821; Camp Pendleton, N =163,484). The investigators utilized a quantitative bias analysis procedure (QBA) to estimate the possible impacts on the adjusted hazard ratios observed of confounding from smoking and alcohol consumption (prevalence rates of which were estimated based on negative control disease analyses) and exposure misclassification bias. In their final analyses, Bove et al. found that the Camp Lejeune Marines/Navy personnel had elevated adjusted hazard ratio for all myeloid cancers (HR=1:24; 95% CI: 1.03, 1.49), acute myeloid leukemia (HR=1:38; 95% CI: 1.03, 1.85), myelodysplastic and myeloproliferative syndromes (HR=1:68; 95% CI: 1.07, 2.62). These results did not change meaningfully in the QBA analyses, and given that each of the confidence intervals of these results exclude 1, the results would be considered "statistically significant".

Thus, despite the argument made by Dr. Bailey that a threshold must exist below which exposure to a carcinogen is not likely to elevate the risk for cancer, coupled with the argument she makes that there is no direct epidemiological evidence of risk at very low levels of carcinogen exposure, such evidence now exists, including evidence pertaining specifically to Camp Lejeune.

(3) In pages 24-25, Dr. Bailey discusses her hazard assessments and toxicity criteria for TCE, PCE, benzene, and other chemicals. With respect to TCE, PCE, and benzene, Dr. Baily quotes and relies on the expert report of Dr. Julie Goodman in which she reviews the evidence pertaining to the causal relationship between these chemicals and various cancers.

Comment: I refer, instead, to my own general causation report.

(4) On page 43, Dr. Bailey critiques my specific causation report on Mr. Carter.

a. Dr. Bailey states that my evaluation "is not consistent with US EPA's risk assessment guidelines, which consider not only exposure concentrations, but also exposure frequency and duration."
Comment: This is erroneous. On page 10 of my report, in discussing specific causation with regards to Mr. Carter, I not only reviewed the exposure assessment that Dr. Reynolds conducted of Mr. Carter's exposure, but I explicitly noted that "…*Mr. Carter's status as a civilian worker at Camp Lejeune and the timing of his exposure profile very closely aligns with, and, in fact, exceeds, the exposure profile (discussed in II.G. above) of "15-years of exposure to workers on-base who lived off-base to all chemical contaminants from Hadnot Point (i.e., PCE, TCE, and benzene)" that ATSDR estimated was associated with a lifetime cancer risk of over 1 per 10,0000 for exposures between the mid-1960's to around 1982, with a peak of 2.6 per 10,000 for exposures surrounding 1970.*."

[15] Bove FJ, Greek A, Gatiba R, Kohler B, Sherman R, Shin GT, Bernstein A. Cancer Incidence among Marines and Navy Personnel and Civilian Workers Exposed to Industrial Solvents in Drinking Water at US Marine Corps Base Camp Lejeune: A Cohort Study. Environ Health Perspect. 2024 Oct;132(10):107008. doi: 10.1289/EHP14966. Epub 2024 Oct 24. PMID: 39446420; PMCID: PMC11500795.

b. Dr. Bailey criticized my comparison of the Dr. Carter's exposures with the US EPA's maximum contaminate levels (MCLs) and ATSDR's cancer risk evaluation guides (CREGs) as not a reliable risk evaluation method.

Comment: My referring to the EPA's MCLs and ATSDR's CREGs was clearly not meant to be, and was not used as a methodology for quantitating his risk. It was meant to simply contrast his exposure levels with the applicable drinking water standards. Instead, as noted above, I quoted the ATSDR risk scenario and quantitative of risk, which utilized their approach to estimating exposures for a Marine who trained and lived on base for a 3-years exposed to drinking contaminants from the Hadnot WTP, which closely matches the experience of Mr. Carter. In my opinion, the ATSDR's approach to the calculated the associated quantitative risk was rigorous.

c. Dr. Bailey relies on Dr. Goodman's expert report to conclude that the scientific evidence does not support a causal association between TCE, PCE, benzene, vinyl chloride, or 1,2 tDCE exposure and NHL.

Comment: I stand by my own expert general causation report, which concluded that TCE, PCE, and Benzene more likely than not cause NHL, both individually and collectively.

d. Dr. Bailey quotes Dr. Goodman's report to criticize the Camp Lejeune studies, specifically pointing out that there is "*high likelihood of exposure misclassification*" and noting Dr. Goodman's statement that "*Overall, most analyses do not provide evidence of associations between either working or living at Camp Lejeune or TCE, PCE, benzene, or vinyl chloride exposures at Camp Lejeune and NHL overall or any NHL subtype. Almost all risk estimates were less than or close to 1. The few reported statistically significant risk estimates were not consistently reported across analyses of the Camp Lejeune population, and all were <1*."

Comment: (A) I first point out that exposure misclassification is a common limitation in epidemiological studies. However, unless there is reason to suspect a *differential* exposure misclassification that would introduce a specific bias that increases the likelihood of finding an association (where none truly exists), non-differential (i.e., random) exposure misclassification typically results in a bias to the null, i.e., a dilution of any apparent effect. Dr. Bailey has not introduced any evidence that such a *differential* exposure misclassification existed in the Bove studies; I also point out that the Bove studies were published in top environmental health journals and went through rigorous peer review, which no doubt would have included an assessment for such a bias. (B) It is true that the Camp Lejeune studies did not find associations between exposures at Camp Lejeune and NHL that were "statistically significant". However, in the 2024 cancer incidence study, Bove et al. did find elevated adjusted hazard ratios for the NHL mantle cell and marginal zone B-cell sub-types that had confidence interval ratios ≤3 despite being based on only 27 and 43 cases, respectively. As such, these results are consistent with an increased risk, especially since the age range of the participants in the Camp Lejeune study at the end of the study's follow-up had mean (SD) and median values of only 56.3 (4.5) and 57 years, respectively. Given that the median age of those who develop NHL in the United States is around 67 years, it is clear that the lack of

statistical significance in these data cannot be construed as definitive evidence that the exposures at Camp Lejeune do not pose a risk for NHL.

This ends my rebuttal of Dr. Bailey's report.

Sincerely,

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