Exhibit 345

Toxicology Associates, Prof. LLC
Dedicated to Patient Care, Research, and Teaching in Medical Toxicology

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Re: Camp Lejeune (Laramore)

CREDENTIALS, EXPERTISE, AND EXPERIENCE

By way of introduction, I am a medical toxicologist, emergency physician, and epidemiologist. I am currently an Associate Professor at the University of Colorado School of Medicine with a primary appointment in the Section of Medical Toxicology, Department of Emergency Medicine. In addition, I am an attending physician at the Rocky Mountain Poison and Drug Center as well as a member of Toxicology Associates, Prof. LLC, a hospital based, single specialty medical group dedicated solely to medical toxicology. I am board certified in both emergency medicine and medical toxicology, and I actively practice both emergency medicine and medical toxicology. Medical toxicology is an American Board of Medical Specialties recognized sub-specialty that deals specifically with human disease associated with any potentially toxic exposure. As a medical toxicologist, I specialize in the assessment, diagnosis, and treatment of adverse effects of pharmaceuticals, other chemicals, natural toxins, envenomations, and any other potential toxicants or toxicological conditions.

I received my M.D. at the University of Texas - Southwestern Medical Center in Dallas, Texas. After completion of this degree, I entered residency in emergency medicine at Denver Health Medical Center in Denver, Colorado. Following this, I worked as faculty in the emergency medicine residency program at Denver Health Medical Center and the University of Colorado School of Medicine for a single year. Subsequently, I entered medical toxicology fellowship training at Oregon Health and Science University in Portland, Oregon. During my fellowship, I simultaneously obtained a Masters in Public Health in epidemiology and biostatistics. Upon completion of my Fellowship and M.P.H. degree, I returned to Denver in 2013 and joined the faculty at the University of Colorado School of Medicine, the Rocky Mountain Poison and Drug Center, and Toxicology Associates.

In my role at the University of Colorado School of Medicine and the Rocky Mountain Poison and Drug Center, I provide case based teaching, didactic instruction, and supervision of clinical care provided by medical and pharmacy students, residents, and medical toxicology fellows-in-training. In addition, I am actively involved in research through the University of Colorado School of Medicine and the Rocky Mountain Poison and Drug Center. Furthermore, I have an active practice primarily caring for patients through Toxicology Associates. In all these capacities, I routinely evaluate and treat patients who present with both acute and chronic toxicologic issues. I have evaluated and treated thousands of patients with toxicologic conditions during my career.

RATIONALE AND METHODS

I have previously provided reports discussing Camp Lejeune exposures as a cause of kidney and bladder cancers. Herein, I am supplementing the original report with an examination of the toxicologic significance of individual plaintiff specific exposure estimates with a focus on identifying those exposures that rise to a level "recognized to be hazardous to humans" as well as those that are "substantial" exposures. In performing this analysis, I am assuming that:

- modeling generated by the ATSDR are reasonable estimates of the monthly exposures in the Hadnot Point and Tarawa Terrace water systems at Camp Lejeune
- deposition testimony and available records accurately reflect times and locations on base for an individual patient
- estimates generated by expert Dr. Reynolds are sufficiently reliable for purposes of this analysis

For each plaintiff analyzed, I am providing a narrative discussion as well as a table of estimated cumulative exposures. If a plaintiff was only exposed via a single water system, I also performed a sensitivity analysis accounting for partial months of exposure. However, the primary analysis is consistent with the ATSDR's approach in Dr. Bove's series of published manuscripts with any time spent on base during a particular month considered a full month of exposure. In addition, if a plaintiff had exposures from both Hadnot Point and Tarawa Terrace, I either relied upon Dr Reynolds' combined exposure table as realizing the apportionment of exposure between the systems or apportioned myself based on reported intake if not provided to me by Dr. Reynolds. In these cases, a sensitivity analysis accounting for partial months was not performed as such an exercise would include an excessive degree of uncertainty making the sensitivity analysis uninformative.

EXPOSURES

Time on Base

Jimmy Laramore experienced exposures at Camp Lejeune that are recognized to be hazardous to human health when considering bladder cancer as an outcome. A minimum exposure of 1-21 quarters on base for civilian personnel at Camp Lejeune between October 1972 and December 1985 has been associated with bladder cancer diagnosis (Bove 2024a). Mr. Laramore spent 4.33 quarters on base between December 1983 and December 1984, corresponding to an elevated measure of association (1-21 quarters=aHR 1.18) with development of bladder cancer. The population is limited to civilian personnel who may have less intense exposures than military personnel. In contrast, military personnel have also demonstrated elevated measures of association with bladder cancer diagnosis when stationed for at least 7 quarters at Camp Lejeune from 1975-1985 (Bove 2024a). Exposures of 1-6 quarters did not reveal an elevated measure of association (aHR 1.02) with bladder cancer diagnosis.

TVOC

At least "medium" exposures to TVOC (>4600 ug/L*month) in Camp Lejeune water systems demonstrated a nonmonotonic association with bladder cancer (Bove 2014a). The estimated TVOC modeled in the Hadnot Point water system while Mr. Laramore was on base was 5889 ug/L*month ("medium" exposure [>4600-12250 ug/L*month]=HR 3.33). Of note, the modeled

TVOC exposure for Mr. Laramore is an underestimate as it excludes DCE while Dr. Bove included DCE in his TVOC exposure estimates.

PCE

Individual compound exposure estimates have also been associated with bladder cancer. At least "low" exposures to PCE (>0 ug/L*month) in Camp Lejeune water systems demonstrated a nonmonotonic association with bladder cancer diagnosis (ATSDR 2018). The estimated PCE modeled in the Hadnot Point water system while Mr. Laramore was on base was 280 ug/L*month ("medium" exposure [>/=36 to <711 ug/L*month]=OR 1.30 when compared to Camp Pendleton but OR=0.99 in an internal Camp Lejeune analysis). Additionally, at least "medium" exposures to PCE (>155 ug/L*month) in Camp Lejeune water systems demonstrated a nonmonotonic association with bladder cancer mortality (Bove 2014a). The estimated PCE modeled in the Hadnot Point water system while Mr. Laramore was on base was 280 ug/L*month ("medium" exposure [>155 to 380 ug/L*month]=HR 1.62). PCE exposures have also been studied with bladder cancer as an outcome in populations outside of Camp Lejeune. Massachusetts water system contamination demonstrated monotonic elevated measures of association (OR 1.16 and OR 6.04 without latency) in those exposed to "low" and "high" doses (Ashchengrau 1993). Although not explicitly defined for cases, the "low" dose category in controls ranges from any exposure (at least 0.01mg) up to the 90th percentile of relative dose delivered (27.1mg with latency and 44.1mg without latency). Given exposure expert Dr. Reynolds calculated that the dose of PCE delivered to Mr. Laramore while at Camp Lejeune ranged from 29.2mg to 54.5mg, this range of exposure corresponds to the "low" and "high" exposure groups depending on the estimation method in a water system contamination study of a non-Camp Lejeune population.

TCE

TCE in Camp Lejeune water systems demonstrated a nonmonotonic association with bladder cancer diagnosis at "low" and "medium" cumulative exposures (ATSDR 2018). The estimated TCE modeled in the Hadnot Point water system while Mr. Laramore was on base was 5889 ug/L*month ("medium" exposure [>/=110 to <11030 ug/L*month]=OR 1.68 when compared to Camp Pendleton and OR=1.34 in an internal Camp Lejeune analysis). Additionally, "medium" exposures to TCE (>3100-7700 ug/L*month) in Camp Lejeune water systems demonstrated a nonmonotonic association with bladder cancer mortality (Bove 2014a). The estimated TCE modeled in the Hadnot Point water system while Mr. Laramore was on base was 5889 ug/L*month ("medium" exposure [>3100 to 7700 ug/L*month]=HR 2.69).

Vinyl Chloride

"Medium" cumulative exposures to vinyl chloride (>205-500 ug/L*month) in Camp Lejeune water systems demonstrated an association with bladder cancer mortality (Bove 2014a). Estimated vinyl chloride modeled in the Hadnot Point water system while Mr. Laramore was on base was 509 ug/L*month if counting any period of a month as the entire month ("high" exposure [>500-2800 ug/L*month]=HR 0.91). However in a sensitivity analysis using days of the month on base to develop a proportional estimate of cumulative exposure, the estimated vinyl chloride modeled in the Hadnot Point water system was 492 ug/L*month ("medium" exposure [>205 to 500 ug/L*month]=HR 2.59).

Benzene
At least "medium" exposures to benzene (>45 ug/L*month) in Camp Lejeune water systems demonstrated a nonmonotonic association with bladder cancer mortality (Bove 2014a). The estimated benzene exposure modeled in the Hadnot Point water system while Mr. Laramore was on base was 105 ug/L*month ("medium" exposure [>45 to 110 ug/L*month]=HR 4.04).

Inhalational studies
In three additional studies with nonmonotonic exposure response relationships, exposures were predominantly inhalational. Given that Mr. Laramore's exposures represent a combination of ingestion, dermal, and inhalational routes, it is unclear whether risk estimates from these studies are directly informative. A Nordic study of largely inhalational exposures in various occupations revealed an elevated measure of association only in the highest tertiles of TCE exposure (>129.50 ppm*year=HR 1.23) and benzene exposure (>15.04 ppm*year =HR 1.16) as well as only in the middle tertile (13.60-87.55 ppm*year=HR 1.12) of PCE exposure (Hadkhale 2017). In another Nordic study, dry cleaning workers occupationally exposed to PCE demonstrated a nonmonotonic association (RR 0.91-2.39 depending on duration of exposure) with bladder cancer where a mean measurement of "at least 60-minute exposures to PCE" was 164mg/m^3 (Lynge 2006). Finally, an Italian air pollution study of exclusively inhalational exposures indicated an association only in the medium tertile of geographically segmented estimates (1.1-1.8 ug/m^3=HR 1.16) of benzene air pollution (Hadkhale 2017).

LEVELS RECOGNIZED TO BE HAZARDOUS TO HUMAN HEALTH
Mr. Laramore experienced exposures at levels recognized to be hazardous to humans and consistently demonstrate elevated measures of association with bladder cancer. This includes exposure metrics evident in the Camp Lejeune population from 1972-1985 such as time on base at Camp Lejeune (Bove 2024a), the combination of individual compounds expressed as TVOC (Bove 2014a), TCE (Bove 2014a, ATSDR 2018), PCE (Bove 2014a, ATSDR 2018), vinyl chloride (Bove 2014a), and benzene (Bove 2014a). Furthermore, the exposure experienced by Mr. Laramore is similar to a water system exposure outside of the Camp Lejeune population that demonstrated an elevated measures of association with bladder cancer for PCE (Aschengrau 1993).

SUBSTANTIAL EXPOSURES
As a practicing medical toxicologist, I conclude that the delivered dose estimates generated by exposure expert Dr. Reynolds represent "substantial" rather than "de minimis" exposures to PCE, TCE, vinyl chloride, and benzene when considering an outcome of bladder cancer. This is evident in the quantified exposures recognized to be hazardous to human health examined above. Additionally, I would consider exposures of this magnitude in a differential diagnosis for a patient presenting to my practice with suspected bladder cancer.

As noted by the EPA in banning both TCE and PCE in final rules published in December 2024 (EPA 2024), each toxin represents an "unreasonable threat to health". There is no safe level of exposure identified for either. The EPA's rationale specifically recognized TCE as a cause of kidney cancer, and TCE is presumed to act via a similar mechanism as a cause of bladder cancer. Compared to oral exposures, the EPA estimated that for an equivalent delivered dose of TCE, inhalational exposures were approximately twice as potent in causing kidney cancer (EPA 2011).

The ban for PCE was justified, at least in part, by the associations between PCE exposures and cancers (including bladder cancer). In light of the robust evidence for TCE and PCE exposure as a cause of bladder cancer, the potency of exposure in inhalational data, and the unquantified but at minimum additive impact of combined exposure to multiple toxins by various routes, even a less robust exposure to toxins that may not reach a level recognized to be hazardous to human health still represents contributory exposure for Ms. Laramore. Consequently, I conclude that Mr. Laramore experienced "substantial" exposures to TCE, PCE, vinyl chloride, and benzene at Camp Lejeune.

The opinions expressed above are to a reasonable degree of medical and scientific certainty.

Respectfully

Benjamin Hatten MD MPH

APPENDIX A: Analysis Tables

Exposure Dates	Total Days
12/10/1983-12/31/1983	22
1/1/1984-1/31/1984	31
2/1/1984-2/29/1984	29
3/1/1984-3/31/1984	31
4/1/1984-4/30/1984	30
5/1/1984-5/31/1984	31
6/1/1984-6/30/1984	30
7/1/1984-7/31/1984	31
8/1/1984-8/31/1984	31
9/1/1984-9/30/1984	30
10/1/1984-10/31/1984	31
11/1/1984-11/30/1984	30
12/1/1984-12/31/1984	31
	388

Levels harmful to human health Duration

Monotonic

Bove 2024a (>/= 7 quarters) aHR 1.02

Ashcengrau 1993 (>0 ug PCE dose delivered)

Nonmonotonic

Bove 2024a (1-21 quarters) aHR 1.18

Bove 2014a (>3100-7700ug/L*month TCE; >155-380ug/L*month PCE; >205 to 500ug/L*month vinyl chloride; >45-110ug/L*month benzene; >4600-12250ug/L*month TVOC)

ATSDR 2018 (110-<11030ug/L*month TCE; >/=36-<711ug/L*month PCE)

Exposure Location (Work)	TCE (ug/l-M)	PCE (ug/l-M)	VC (ug/l-M)	BZ (ug/l-M)	TVOC (ug/L-month)
Hadnot Point	688	34	59	9	790
Hadnot Point	427	21	36	11	495
Hadnot Point	560	27	47	8	642
Hadnot Point	587	28	50	7	672
Hadnot Point	400	18	33	12	463
Hadnot Point	491	23	42	10	566
Hadnot Point	471	22	41	7	541
Hadnot Point	507	24	45	7	583
Hadnot Point	539	26	48	8	621
Hadnot Point	443	21	39	8	511
Hadnot Point	94	3	6	8	111
Hadnot Point	639	31	59	8	737
Hadnot Point	43	2	4	2	51
	5889	280	509	105	6783
	TCE	PCE	Vinyl Chloride	Benzene	TVOC
		OR 1.16-6.04			
	HR 2.69	HR 1.62	HR 0.91	HR 4.04	HR 3.33
	OR 1.68/OR 1.34	OR 1.30/OR 0.99			

TCE (ug/l-M)	PCE (ug/l-M)	VC (ug/l-M)	BZ (ug/l-M)	TVOC (ug/L-month)	Y/N (exposure with elevated measure of association)
488	24	42	6	561	
427	15	36	11	489	
560	20	47	8	635	
587	20	50	7	664	
400	13	33	12	458	
491	16	42	10	559	
471	16	41	7	535	
507	17	45	7	576	
539	18	48	8	613	
443	15	39	8	505	
94	2	6	8	110	
639	23	59	8	729	
43	1	4	2	50	
5689	202	492	102	6486	
TCE	PCE	Vinyl Chloride	Benzene	TVOC	
					N
	OR 1.16-6.04				Y
aHR 1.18					Y
HR 2.69	HR 1.62	HR 2.59	HR 4.04	HR 3.33	Y
OR 1.68/OR 1.34	OR 1.30/OR 0.99				Y