

Exhibit 343



Toxicology Associates, Prof. LLC

Dedicated to Patient Care, Research, and Teaching in Medical Toxicology

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Re: Camp Lejeune (Criswell)

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

Mike Criswell experienced exposures at Camp Lejeune that are recognized to be hazardous to human health when considering bladder cancer as an outcome. Military personnel have demonstrated elevated measures of association with bladder cancer diagnosis only when stationed for at least 7 quarters at Camp Lejeune from 1975-1985 (Bove 2024a). Mr. Criswell spent 9.3 quarters on base between July 1975 and March 1977, corresponding to a monotonically elevated measure of association (7-10 quarters=aHR 1.18) with development of bladder cancer. 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. Criswell spent 9.3 quarters on base between July 1975 and March 1977, indicating an elevated measure of association (1-21 quarters=aHR 1.18) with development of bladder cancer. Of note, the population in this study is limited to civilian personnel who may have less intense exposures than military personnel.

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. Criswell was on base was 10358

ug/L*month (“medium” exposure [$>4600-12250$ ug/L*month]=HR 3.33). Of note, the modeled TVOC exposure for Mr. Criswell is an underestimate as it excludes DCE while Dr. Bove included DCE in his TVOC exposure estimates.

PCE

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. Criswell was on base was 1406 ug/L*month (“high” exposure [≥ 711 ug/L*month]=OR 2.07 when compared to Camp Pendleton and OR=1.54 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. Criswell was on base was 1406 ug/L*month (“high” exposure [>380 to 8585 ug/L*month]=HR 1.24). 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. Criswell while at Camp Lejeune ranged from 120.6mg to 168.0mg, this degree of exposure corresponds to the “high” exposure group with an elevated measure of association (OR 6.04) 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. Criswell was on base was 8404 ug/L*month (“medium” exposure [$\geq 110-11030$ 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. Criswell was on base was 8404 ug/L*month (“high” exposure [>7700 ug/L*month]=HR 0.92).

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. Criswell was on base was 463 ug/L* (“medium” exposure ($>205-500$ 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. Criswell was on base was 85 ug/L*month (“medium” exposure [>45 -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. Criswell’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 (Hadjkhale 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 $164\text{mg}/\text{m}^3$ (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 (Hadjkhale 2017).

LEVELS RECOGNIZED TO BE HAZARDOUS TO HUMAN HEALTH

Mr. Criswell 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), PCE (Bove 2014a, ATSDR 2018), TCE (ATSDR 2018), vinyl chloride (Bove 2014a) and benzene (Bove 2014a). Furthermore, the exposure experienced by Mr. Criswell 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 Mr. Criswell. Consequently, I conclude that Mr. Criswell 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

A handwritten signature in black ink, appearing to be 'B. Hatten', written in a cursive style.

Benjamin Hatten MD MPH

APPENDIX A: Analysis Tables

Hadnot Point
TCE
PCE
VC
BZ
Terawa Terrace
TCE
PCE (TechFlowMP Model)
PCE (MT3DMS Model)
VC
BZ
Totals HP & TT
TCE
PCE (TechFlowMP Model)
PCE (MT3DMS Model)
VC
BZ
TVOC

Levels harmful to human health

Monotonic

Bove 2024a (>= 7 quarters)

Ashcengrau 1993 (>0 ug PCE dose delivered)

Nonmonotonic

Bove 2024a (1-21 quarters)

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)

	Chart 1: 1L	Chart 2: ATSDR	Chart 3: Deposition/FM
Cumulative ug/l-M	Cumulative consumption (total ug= days*concentration per L)	Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions)
8,357	120,644	818,290	1,018,536
258	3,517	24,781	30,543
380	5,374	36,900	45,784
85	1,252	8,251	10,350
47	907	3,885	5,576
1,148	22,351	95,791	137,489
1,593	30,986	132,797	190,604
83	1,615	6,922	9,935
-	-	-	-
8,404	121,551	822,175	1,024,112
1,406	25,869	120,572	168,032
1,851	34,503	157,578	221,147
463	6,990	43,822	55,719
85	1,252	8,251	10,350
10,358	155,661	994,820	1,258,213

**Y/N
(exposure
with
elevated
measure
of
associati
on)**

Duration	TCE	PCE	VC	Benzene	TVOC	Y/N
9.3 quarters						Y
aHR 1.18		OR 6.04				Y
						Y
aHR 1.18						Y
	HR 0.92	HR 1.24	HR 2.59	HR 4.04	HR 3.33	Y
	OR 1.68/OR 1.34	OR 2.07/OR 1.54				Y