Exhibit 342

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

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Re: Camp Lejeune (Cagiano)

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

Mark Cagiano 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 monotonically elevated measures of association with bladder cancer diagnosis when stationed for at least 7 quarters at Camp Lejeune from 1975-1985 (Bove 2024a). Mr. Cagiano spent 14 quarters on base between July 1976 and December 1987 with 11.3 quarters between 1975 and 1985, corresponding to an elevated measure of association (>10 quarters=aHR 1.20) 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. Cagiano spent 14 quarters on base between July 1976 and December 1987 with 11.3 quarters between 1975 and 1985 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. Cagiano was on base was 13744 ug/L*month ("high" exposure [>12250-64016 ug/L*month]=HR 1.20). Of note, the modeled TVOC exposure for Mr. Cagiano 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. Cagiano was on base was 510 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. Cagiano was on base was 510 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 calculations that the dose of PCE delivered to Mr. Cagiano while at Camp Lejeune ranged from 46mg to 54mg, this range 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. Cagiano was on base was 12365 ug/L*month ("high" exposure [>/=11030 ug/L*month]=OR 0.93 when compared to Camp Pendleton and OR=0.74 in an internal Camp Lejeune analysis). When partial months are accounted for in a sensitivity analysis, the estimated TCE modeled in the Hadnot Point water system while Mr. Cagiano lived on base was 10089 ug/L*month ("medium" exposure [>/=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. Cagiano was on base was 12365 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. Cagiano was on base was 701 ug/L* ("high" exposure [>500-2800 ug/L*month]=HR 0.91).

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. Cagiano was on base was 168 ug/L*month ("high" exposure [>110-601 ug/L*month]=HR 2.26).

Inhalational studies
In three additional studies with nonmonotonic exposure response relationships, exposures were predominantly inhalational. Given that Mr. Cagiano'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. Cagiano 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), and benzene (Bove 2014a). Furthermore, the exposure experienced by Mr. Cagiano 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, 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 exposure to toxins that have not been recognized to be hazardous to human health may still represent a contributory exposure for Mr. Cagiano. Consequently, I conclude that Mr. Cagiano 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	Exposure Location (Work)
7/31/76	1	Hadnot Point
8/1/1976-08/31/1976	31	Hadnot Point
9/1/1976-9/26/1976	26	Hadnot Point
10/09/1976-10/31/1976	23	Hadnot Point
11/1/1976-11/30/1976	30	Hadnot Point
12/1/1976-12/19/1976	19	Hadnot Point
12/27/1976-12/31/1976	5	Hadnot Point
1/1/1977-1/31/1977	31	Hadnot Point
2/1/1977-2/28/1977	28	Hadnot Point
3/1/1977-3/27/1977	27	Hadnot Point
4/5/1977-4/20/1977	16	Hadnot Point
11/11/1977-11/30/1977	20	Hadnot Point
12/1/1977-12/31/1977	31	Hadnot Point
1/1/1978-1/31/1978	31	Hadnot Point
2/1/1978-2/28/1978	28	Hadnot Point
3/1/1978-3/31/1978	31	Hadnot Point
4/1/1978-4/30/1978	30	Hadnot Point
5/1/1978-5/31/1978	31	Hadnot Point
6/1/1978-6/2/1978	2	Hadnot Point
7/1/1978-7/31/1978	31	Hadnot Point
8/1/1978-8/3/1978	3	Hadnot Point
8/14/1978-8/31/1978	18	Hadnot Point
9/1/1978-9/30/1978	30	Hadnot Point
10/01/1978-10/31/1978	31	Hadnot Point
11/1/1978-11/30/1978	30	Hadnot Point
12/1/1978-12/31/1978	31	Hadnot Point
1/1/1979-1/31/1979	31	Hadnot Point
2/1/1979-2/28/1979	28	Hadnot Point
3/1/1979-3/7/1979	7	Hadnot Point
11/28/1979-11/30/1979	3	Hadnot Point
12/1/1979-12/28/1979	28	Hadnot Point
1/3/1980-1/12/1980; 1/27/1980-1/31/1980	15	Hadnot Point
2/1/1980-2/29/1980	29	Hadnot Point
3/1/1980-3/31/1980	31	Hadnot Point
4/1/1980-4/30/1980	30	Hadnot Point
5/1/1980-5/26/1980	28	Hadnot Point
5/30/1987-5/30/1987	2	Hadnot Point
6/1/1987-6/30/1987	30	Hadnot Point
7/1/1987-7/31/1987	31	Hadnot Point
8/1/1987-8/31/1987	31	Hadnot Point
9/1/1987-9/30/1987	30	Hadnot Point
10/01/1987-10/31/1987	31	Hadnot Point
11/1/1987-11/30/1987	30	Hadnot Point
12/1/1987-12/31/1987	31	Hadnot Point
14	11.33333333	1061

Levels harmful to human health

Monotonic

Bove 2024a (>/= 7 quarters) aHR 1.20

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)

TCE (ug/l-M)	PCE (ug/l-M)	VC (ug/l-M)	BZ (ug/l-M)	TVOC (ug/L-month)
348	12	16	3	379
436	15	20	4	475
356	11	16	3	386
70	2	3	3	78
543	19	26	4	592
520	19	25	3	567
				0
249	9	12	4	274
346	13	17	3	379
342	13	17	2	374
218	8	11	4	241
544	22	30	4	600
513	21	28	4	566
250	10	14	4	278
348	14	19	3	384
352	15	20	3	390
231	9	13	5	258
278	12	16	4	310
333	14	19	3	369
388	17	23	3	431
475	20	28	4	527
				0
364	16	22	4	406
74	3	4	4	85
544	24	33	5	606
546	24	33	4	607
268	12	16	6	302
370	17	23	5	415
378	17	24	5	424
507	23	33	6	569
504	23	33	6	566
264	12	17	7	300
378	17	24	6	425
433	20	28	6	487
273	12	17	8	310
322	15	21	6	364
0	0	0	2	2
0	0	0	2	2
0	0	0	3	3
0	0	0	3	3
0	0	0	3	3
0	0	0	3	3
0	0	0	2	2
0	0	0	2	2
12365	510	701	168	13744

TCE (ug/l-M)	PCE (ug/l-M)	VC (ug/l-M)	BZ (ug/l-M)	TVOC (ug/L-month)
11	0	1	0	12
436	15	20	4	475
309	10	14	3	335
52	1	2	2	58
543	19	26	4	592
319	12	15	2	348
84	3	4	0	91
249	9	12	4	274
346	13	17	3	379
298	11	15	2	326
116	4	6	2	129
363	15	20	3	400
513	21	28	4	566
250	10	14	4	278
348	14	19	3	384
352	15	20	3	390
231	9	13	5	258
278	12	16	4	310
22	1	1	0	25
388	17	23	3	431
46	2	3	0	51
276	12	16	2	306
364	16	22	4	406
74	3	4	4	85
544	24	33	5	606
546	24	33	4	607
268	12	16	6	302
370	17	23	5	415
85	4	5	1	96
51	2	3	1	57
455	21	30	5	511
128	6	8	3	145
378	17	24	6	425
433	20	28	6	487
273	12	17	8	310
291	14	19	5	329
0	0	0	0	0
0	0	0	2	2
0	0	0	3	3
0	0	0	3	3
0	0	0	3	3
0	0	0	3	3
0	0	0	2	2
0	0	0	2	2
10089	416	571	138	11202

Y/N (exposure with elevated measure of association)
Y
Y
Y
Y
Y

OR 6.04

OR 6.04

HR 0.92 HR 1.24 HR 0.91 HR 2.26 HR 1.20

HR 0.92 HR 1.24 HR 0.91 HR 2.26 HR 3.33

OR 0.93/OR 0.74 OR 1.30/OR 0.99

OR 1.68/OR 1.34 OR 1.30/OR 0.99