# Exhibit 346

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

Jeffrey Brent, MD, PhD
Ken Kulig, MD
Edward W. Cetaruk, MD
Robert B. Palmer, PhD

26 West Dry Creek Circle, Suite 815
Littleton, CO 80120
Phone: 720-477-2500 Fax: 720-598-0409

Benjamin Hatten, MD, MPH
Nicklaus Brandehoff, MD
Alexa Camarena-Michel, MD

February 7, 2025

Re: Camp Lejeune (Raymond)

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 Kelly 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*

Edward Raymond 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. Raymond spent 9 quarters on base between November 1963 and December 1965. Although this is not the same period studied, the estimated exposure for each toxin falls within the range of modeled exposures between October 1972 and December 1985, 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. Military personnel have likewise demonstrated elevated measures of association with bladder cancer diagnosis when stationed for at least 7 quarters at Camp Lejeune from 1975-1985 (Bove 2024a). Although Mr. Raymond spent at least 7 quarters at Camp Lejeune, his estimated exposures for each compound was below the minimum for January 1975-December 1985 suggesting that the intensity of exposure is dissimilar enough that a lower exposure category is the most appropriate surrogate. 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. Raymond was on base was 600 ug/L*month ("low" exposure [>1-4600 ug/L*month]=HR 0.63). Of note, the modeled TVOC exposure for Mr. Raymond is an underestimate as it excludes DCE while Dr. Bove included DCE in his TVOC exposure estimates.

*PCE*
No PCE exposure in Hadnot Point was estimated while Mr. Raymond was on base.

*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. Raymond was on base was 579 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. Raymond was on base was 579 ug/L*month ("low" exposure=>1 to 3100 ug/L*month]). The limited number of cases meant that the measure of association could not be calculated for this subgroup.

*Vinyl Chloride*
No vinyl chloride exposure in Hadnot Point was estimated while Mr. Raymond was on base.

*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. Raymond was on base was 21 ug/L*month ("low" exposure=2 to 45 ug/L*month). The limited number of cases meant that the measure of association could not be calculated for this subgroup.

*Inhalational studies*
In two additional studies with nonmonotonic exposure response relationships, exposures were predominantly inhalational. Given that Ms. Raymond'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) (Hadkhale 2017). An Italian air pollution study of exclusively inhalational exposures also indicated an association but 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. Raymond experienced exposures at levels recognized to be hazardous to humans, demonstrating 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) and TCE exposure (ATSDR 2018).

SUBSTANTIAL EXPOSURES

As a practicing medical toxicologist, I conclude that Mr. Raymond experienced a "substantial" rather than "de minimis" exposures to TCE when considering an outcome of bladder cancer given 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 TCE in final rules published in December 2024 (EPA 2024), the toxin represents an "unreasonable threat to health". There is no safe level of exposure identified. 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). In light of the robust evidence for TCE 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. Raymond. Consequently, I conclude that Mr. Raymond experienced a "substantial" exposure not only to TCE but also to benzene in combination with TCE 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 |
|---|
| 11/22/1963-11/30/1963 |
| 12/1/1963-12/31/1963 |
| 1/1/1964-1/31/1964 |
| 2/1/1964-2/29/1964 |
| 3/1/1964-3/31/1964 |
| 4/1/1964-4/30/1964 |
| 5/1/1964-5/31/1964 |
| 6/1/1964-6/30/1964 |
| 7/1/1964-7/31/1964 |
| 8/1/1964-8/31/1964 |
| 9/1/1964-9/30/1964 |
| 10/1/1964-10/4/1964 |
| 11/25/1964-11/30/1964 |
| 12/1/1964-12/17/1964; 12/28/1964-12/31/1964 |
| 1/1/1965-1/31/1965 |
| 2/1/1965-2/28/1965 |
| 3/1/1965-3/12/1965; 3/31/1965 |
| 4/1/1965-4/30/1965 |
| 5/1/1965-5/31/1965 |
| 6/1/1965-6/30/1965 |
| 7/1/1965-7/31/1965 |
| 8/1/1965-8/31/1965 |
| 9/1/1965-9/30/1965 |
| 10/1/1965-10/31/1965 |
| 11/1/1965-11/30/1965 |
| 12/1/65 |

9 quarters

**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)

| Total Days | Exposure Location (Work/Residential) | TCE (ug/l-M) | PCE (ug/l-M) | VC (ug/l-M) | BZ (ug/l-M) | TVOC (ug/L-month) | | 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) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Sensitivity analysis | | | | | | |
| 9 | Hadnot Point | 24 | 0 | 0 | 1 | 25 | | 7 | 0 | 0 | 0 | 8 | |
| 31 | Hadnot Point | 21 | 0 | 0 | 1 | 22 | | 21 | 0 | 0 | 1 | 22 | |
| 31 | Hadnot Point | 22 | 0 | 0 | 1 | 23 | | 22 | 0 | 0 | 1 | 23 | |
| 29 | Hadnot Point | 21 | 0 | 0 | 0 | 21 | | 21 | 0 | 0 | 0 | 21 | |
| 31 | Hadnot Point | 18 | 0 | 0 | 0 | 18 | | 18 | 0 | 0 | 0 | 18 | |
| 30 | Hadnot Point | 25 | 0 | 0 | 1 | 26 | | 25 | 0 | 0 | 1 | 26 | |
| 31 | Hadnot Point | 21 | 0 | 0 | 1 | 22 | | 21 | 0 | 0 | 1 | 22 | |
| 30 | Hadnot Point | 20 | 0 | 0 | 0 | 20 | | 20 | 0 | 0 | 0 | 20 | |
| 31 | Hadnot Point | 21 | 0 | 0 | 0 | 21 | | 21 | 0 | 0 | 0 | 21 | |
| 31 | Hadnot Point | 25 | 0 | 0 | 1 | 26 | | 25 | 0 | 0 | 1 | 26 | |
| 30 | Hadnot Point | 22 | 0 | 0 | 1 | 23 | | 22 | 0 | 0 | 1 | 23 | |
| 4 | Hadnot Point | 24 | 0 | 0 | 1 | 25 | | 3 | 0 | 0 | 0 | 3 | |
| 6 | Hadnot Point | 25 | 0 | 0 | 1 | 26 | | 5 | 0 | 0 | 0 | 5 | |
| 21 | Hadnot Point | 23 | 0 | 0 | 1 | 24 | | 16 | 0 | 0 | 1 | 16 | |
| 31 | Hadnot Point | 22 | 0 | 0 | 1 | 23 | | 22 | 0 | 0 | 1 | 23 | |
| 28 | Hadnot Point | 23 | 0 | 0 | 1 | 24 | | 23 | 0 | 0 | 1 | 24 | |
| 13 | Hadnot Point | 19 | 0 | 0 | 0 | 19 | | 8 | 0 | 0 | 0 | 8 | |
| 30 | Hadnot Point | 26 | 0 | 0 | 1 | 27 | | 26 | 0 | 0 | 1 | 27 | |
| 31 | Hadnot Point | 21 | 0 | 0 | 1 | 22 | | 21 | 0 | 0 | 1 | 22 | |
| 30 | Hadnot Point | 21 | 0 | 0 | 1 | 22 | | 21 | 0 | 0 | 1 | 22 | |
| 31 | Hadnot Point | 21 | 0 | 0 | 1 | 22 | | 21 | 0 | 0 | 1 | 22 | |
| 31 | Hadnot Point | 25 | 0 | 0 | 1 | 26 | | 25 | 0 | 0 | 1 | 26 | |
| 30 | Hadnot Point | 22 | 0 | 0 | 1 | 23 | | 22 | 0 | 0 | 1 | 23 | |
| 31 | Hadnot Point | 23 | 0 | 0 | 1 | 24 | | 23 | 0 | 0 | 1 | 24 | |
| 30 | Hadnot Point | 23 | 0 | 0 | 1 | 24 | | 23 | 0 | 0 | 1 | 24 | |
| 1 | Hadnot Point | 21 | 0 | 0 | 1 | 22 | | 1 | 0 | 0 | 0 | 1 | |
| 662 | | 579 | 0 | 0 | 21 | 600 | | 483 | 0 | 0 | 17 | 492 | |
| 7.355556 | | | | | | | | | | | | | |
| aHR 1.02 | | | | | | | | | | | | | N |
| | | | | | | | | | | | | | n/a |
| aHR 1.18 | | | | | | | | | | | | | Y |
| | | | | | | HR 0.64 | | | | | | HR 0.64 | N |
| | | OR 1.68/OR 1.34 | | | | | | OR 1.68/OR 1.34 | | | | | Y |