EXHIBIT 33

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UNITED STATES DISTRICT COURT
EASTERN DISTRICT OF LOUISIANA

KATHLEEN MOLLER

Plaintiff,

vs.

MARTIAN SALES, INC; JOPEN, LLC, A TEXAS LIMITED LIABILITY COMPANY; JOHNSON FOODS, LLC, A WYOMING LIMITED LIABILITY COMPANY; LP IND., LLC, A WYOMING LIMITED LIABILITY COMPANY; CAG HOLDINGS, LLC, A WYOMING LIMITED LIABILITY COMPANY; RMH HOLDINGS, LLC, A WYOMING LIMITED LIABILITY COMPANY; OLISTICA, AN UNINCORPORATED ASSOCIATION; ABC INSURANCE COMPANY AND JOHN DOES 1-4.

Defendants.

CIVIL ACTION NO. 24-0228-WBV-DPC

DENNIS J. PAUSTENBACH, Ph.D., DABT, CIH EXPERT REPORT

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EXPERT REPORT OF DENNIS J. PAUSTENBACH, Ph.D., DABT, CIH

1. I have been asked by Defendants to review materials pertaining to, and to formulate opinions, regarding Ms. Moller's kratom use.

2. My time spent in the preparation of this opinion letter and reviewing documents to formulate my opinions, as well as for any deposition or trial testimony I may be called upon to give, will be billed at a rate of $725 per hour. My opinions in this matter are expressed to a reasonable degree of scientific certainty and are based on information available to me on the date of this report. I have provided a list of documents that were reviewed in forming my opinions. This report may be supplemented if new information becomes available.

3. My opinions below are based on my background, education, training, experience, and the information available to me as of the date of this report. I reserve the right to amend or supplement my opinions to the extent that new information becomes available.

4. My opinions are also based on information that is related to this case, which includes a review of case materials provided to me by counsel, documents in the open scientific literature, and the opinions of some other experts.

5. A copy of my current curriculum vitae is attached as Appendix A. My list of References and Documents Considered is attached as Appendix B. My list of prior testimony for the last four years is set out in Appendix C.

EXPERIENCE

6. I am a board-certified toxicologist and certified industrial hygienist with nearly 40 years of experience in occupational health, risk assessment, toxicology, and environmental engineering. My experience includes investigating the health effects of exposure to, as well as the remediation of, carcinogenic and noncarcinogenic chemicals, including dioxins, furans, benzenes, polychlorinated biphenyls (PCBs), petroleum products, methyl tertiary-butyl ether (MTBE), chlorinated hydrocarbons, glycol ethers, beryllium, lead, 1,3-butadiene, methylene chloride, tetrachloroethylene (PCE), perfluorooctanoic acid (PFOA), chromium, MCHM, airborne biologic agents, kratom and kava, nicotine and asbestos, to name a few. I have published about 300 peer-

reviewed articles and written more than 50 book chapters in these fields. I have also given many lectures at universities on these issues and have conducted or supervised the conduct of more than 700 risk assessments related to persons exposed to various chemicals and radionuclides, occupational exposure to industrial chemicals, contaminated foods and pharmaceuticals, as well as contaminated soils, sediment, air, water and consumer products.

7. I have been the editor for three editions of the most popular textbook in the field of health risk assessment which is entitled "Human and Ecological Risk Assessment: Theory and Practice" (2024).

8. I have specialized in the areas of industrial and environmental toxicology, occupational health, historical state-of-knowledge regarding environmental issues, as well as ecological and human risk assessment. I am board certified in industrial hygiene (American Board of Industrial Hygiene [ABIH]), in toxicology (Diplomate of the American Board of Toxicology [DABT] and Fellow of the Academy of Toxicological Sciences [FACTS]), and in safety (Certified Safety Professional [CSP]). I have served as a graduate instructor/adjunct professor in industrial hygiene in the School of Pharmacy at Purdue University for four years; teaching all the courses in the curriculum. In that role, I taught more than 300 students about the theory and practice of this profession. This program in industrial hygiene, which I am credited for having founded, quickly became the largest program of its kind in the country.

9. I have also passed the theory portion of the professional engineering exam. I have served on many scientific advisory boards to government agencies in the U.S. and elsewhere; including being an advisor to the Administrator of the EPA and the head of the Centers for Disease Control (CDC). I have been an adjunct professor at five or more different universities. My professional education, training, and background are consistent with the topics and areas of scientific study about which I will testify.

10. I have studied issues associated with the hazards of ingested and inhaled consumer products and pharmaceuticals for many years. Specifically, in 2009, my team and I conducted a state-of-the-art evaluation of worldwide regulatory initiatives that evaluated possible health hazard

posed by trace levels of various trace chemicals in personal care products and drinking water. More recently, between 2018 and 2022 several products were identified by FDA as being contaminated with various forms of nitrosamines; we conducted health risk assessments on several of them, most notably n-nitrosodimethylamine (NDMA) and n-nitrosodiethylamine (NDEA). In March 2022, I presented a paper entitled "Current Thoughts on Risk Assessment Methodologies Used to Assess the Carcinogenic Risk of N-Nitrosodimethylamine (NDMA) and N- Nitrosodiethylamine (NDEA)" at the Society of Toxicology Annual Meeting in San Diego, California. In 2024, I published "Risk characterization of N-nitrosodimethylamine in pharmaceuticals" in the journal Food and Chemical Toxicology (2024).

11. I have conducted risk assessments of herbal, food, and vitamin supplements. In 2021, I conducted an evaluation of the toxicology of each component of an over-the-counter herbal supplement to determine the risk of acute liver failure. I have also evaluated risks associated with contamination of vitamin and food supplements with lead, arsenic, and vitamin A. Additionally, from 2021 to present, my team and I have examined potential risks associated with the contamination of a beverage with an agent that allegedly caused acute viral hepatitis in some consumers. Over the years, I have conducted risk assessments for an array of contaminated food products, with examples including frozen processed meats, kale, spices, candies and baked goods.

12. My colleagues and I recently published a review of the literature on kratom, mitragynine, and 7-hydroxymitragynine (Heywood et al. 2024). This included a comprehensive analysis of *in vitro*, *in vivo*, and kratom product chemistry data, along with human clinical data, case study/series, and epidemiological evidence.

13. We also presented two papers at the 2025 national College of Problems of Drug Dependence (CPDD) conference in New Orleans entitled "A Systematic Evaluation of the Literature Related to Alleged Kratom-Induced Seizures" and "What Are the Acute Adverse Health Effects of Kratom, if Any: An Analysis of Case Reports."

14. Given the above, the study of the ingestion and inhalation risks of kratom and its alkaloids is a logical extension of my previous training and experience.

MATERIALS REVIEWED IN FORMULATING OPINIONS

15. My opinions are provided in Sections IV and V of this report. My opinions are based on my professional qualifications, work experience, and knowledge of industrial hygiene, toxicology, risk assessment, and related fields. My opinions also are based on information that is related to this case. In the process of preparing this report, my colleagues or I have reviewed and relied upon the references and case-specific documents listed in Appendix B.

16. In addition to these materials, I have reviewed published papers and textbooks on the toxicology and epidemiology of kratom, mitragynine, and other indole alkaloids available in the open literature and facts or data reasonably relied upon by persons in my field.

CASE-SPECIFIC INFORMATION

General Case Summary

17. Ms. Harmony Anne Moller (Ms. H. Moller) was born on February 18, 1986, and passed away on February 6, 2023, at the age of 36 (Obituary: pg. 1; Coroner's Report: pg. 2, 6). It was reported that Ms. H. Moller's cause of death was "[a]bnormally high left coronary ostia takeoff exacerbated by mitragynine use" (Coroner's Report: pg. 12; Death Certificate). According to a Patient Care Record, the primary impression for Ms. H. Moller's death was cardiac arrest (St. Tammany FD EMS Records: pg. 1). According to the Third Amended Complaint, "… ingestion of Kratom" was allegedly secondary to the cause of death (Third Amended Complaint, paragraph 33).

18. According to the Third Amended Complaint, Ms. H. Moller "… died as the result of the ingestion of OPMS brand Kratom which she purchased in Mandeville Louisiana" (Third Amended Complaint paragraph 30).

Timeline of the Incident on February 6, 2023

- **1:15 p.m.**: Ms. Kathleen Moller (Ms. K. Moller), the decedent's mother, saw Ms. H. Moller conscious (Incident Report: pg. 6). She reported that Ms. H. Moller previously told her that she "… was not feeling well and throwing up" (Incident Report: pg. 9).

- **1:30 p.m.**: Ms. K. Moller discovered Ms. H. Moller laying on her side on the floor of her bedroom. Ms. K. Moller reported that she started CPR until paramedics were on the scene (Incident Report: pg. 6). Ms. K. Moller noted that Ms. H. Moller's skin "… showed blueish discoloration" and that her eyes "… weren't pinpoint," and that Ms. H. Moller did not have a pulse (Incident Report: pg. 13). Ms. K. Moller reported that she "… heard gurgling from Harmony while she conducted chest compressions" (Incident Report: pg. 13).
- **2:11 p.m.**: Deputies dispatched to an unresponsive female (Incident Report: pg. 5).
- **2:58 p.m.**: Ms. H. Moller was pronounced deceased.

Ms. Harmony Anne Moller's Relevant Medical & Records

19. According to the answers to the interrogatories, Ms. H. Moller "… lived for many years abroad and her medical providers [were] unknown to Plaintiff at th[e] time" (Int. #4). According to the answers to the interrogatories, Ms. H. Moller "… occasionally drank alcohol and/or used marijuana" (Int. #8). According to the answers to the interrogatories, Ms. H. Moller "… did not share much [medication or medical] information …" and she "… picked up a prescription for Depo-Provera injection on May 16, 2022" (Int. #9).

20. According to the answers to the interrogatories, Ms. K. Moller had "[g]rief, depression, anxiety, loss of enjoyment of life, [and] loss of consortium" (Int. #12). According to the answers to the interrogatories, Ms. H. Moller "… died from ingestion of OPMS brand Kratom, which she purchased at the Ra Shop in Mandeville, Louisiana" (Int. #21).

Autopsy and Toxicology Screening

21. According to the postmortem forensic medical examination report, Ms. H. Moller had pulmonary edema and congestion, chronic passive congestion of the liver, and mild nephrosclerosis (Coroner's Report: pg. 12). However, according to organ donor records, Ms. H. Moller's cause of death was due to "[c]hronic [c]ongestion of [l]iver …[p]ending [t]ox …" (Coroner's Report: pg. 30). It was reported that Ms. H. Moller also had cardiomegaly with mild biventricular hypertrophy (Coroner's Report: pg. 12).

22. According to the toxicology report, Ms. H. Moller's femoral blood was qualitatively positive for caffeine and naloxone (Coroner's Report: pg. 16). According to the toxicology report, quantitative values for pharmaceuticals were provided including Delta 9-Carboxy THC (10 ng/mL), Delta-9 THC (8.6 ng/mL), Diphenhydramine (64 ng/mL), Promethazine (18 ng/mL), and Mitragynine (150 ng/mL) (Coroner's Report: pg. 16). According to the toxicology report, quantitative values for endogenous and dietary compounds were provided including creatine (0.864 mg/dL), sodium (142 mmol/L), potassium (7.47 mmol/L), chloride (118 mmol/L), glucose (18.2 mmol/L), and urea nitrogen (17.2 mmol/L) (Coroner's Report: pg. 16).

23. According to a Comprehensive Drug Analysis conducted by UCSF Clinical Toxicology and Environmental Biomonitoring Laboratory, confirmed drugs included mitragynine (158 ng/mL), 7-OH mitragynine (0.8 ng/mL), diphenhydramine, Naloxone, and Promethazine (Coroner's Report: pg. 21). It was reported that the value for 7-OH mitragynine "… was below the lower limit of quantification of 3 ng/mL and equal to the limit of detection for the analysis" (Coroner's Report: pg. 21).

Scene Description

24. It was reported that on top of the bedroom dresser was "… drug paraphernalia, one red lighter, one suspected marijuana grinder, one nuEra cannabis container containing suspected marijuana residue, and four empty prescription bottles" (Incident Report: pg. 14). It was noted that the empty prescription bottles included Acyclovir 400 mg (filled 9/26/2022), Methylphenidate HCL ER 27 mg (filled 4/12/2022), Hydrocod/Acetam-5-325 mg (filled 12/1/2015), and Gabapentin 300 mg (filled 12/8/2021) (Incident Report: pg. 14). It was indicated that Acyclovir and Hydrocod/Acetam-5-325 were prescribed to Ms. K. Moller; however, the bottle containing Methyphenidate HCL ER was prescribed to Trenton A. McNab and the bottle containing Gabapentin had its prescription label torn (Incident Report: pg. 14).

25. It was reported that the first bedroom drawer contained "… an empty liquor bottle, one additional empty prescription bottle [for] Amoxicillin 500 mg: prescribed to Harmony Moller (filled 2/10/22), one black case containing suspected marijuana residue, and one suspected item of

drug paraphernalia. The second drawer contained multiple articles of clothing and five empty bottles of Jameson Irish Whiskey. The third drawer contained multiple additional articles of clothing" (Incident Report: pg. 14).

26. It was reported that "[a]fter relocating to the hallway, an open door was observed left of the stairwell. The opened door led to what appeared to be a storage room. Two twin doors were located across the room, which led to a balcony. On the right side of the storage room shared a connected bathroom with the decedent's bedroom. Inside the bathroom, a Rouse's bag was located. A search of the bag was conducted, which contained over-the-counter drugs and other items" (Incident Report: pg. 14) which included: one full bottle of CVS brand Daytime Cold & Flu Relief, one bottle of Apetropics chewable CBD, Lion's Mane, and Cordyceps gummies, one bottle of DG Health immediate release Mucus Relief 400 mg, one bottle of DG Health Naproxen Sodium 220 mg, one bottle of Natrol High Caffeine Energy support tablets 200 mg, one opened sealed bag of Maeng Da brand Kratom of 30 capsules (18 g), one opened sealed bag of Maeng Da brand Kratom 4 oz powder, and one unopened bottle of Knob Creek Whiskey (Incident Report: pg. 14).

27. According to the photos provided of the kratom products found in Ms. H. Moller's bag, she had one bag containing 30 capsules (18 grams) of Green Vein Maeng Da O.P.M.S Silver Kratom (MOLLER_000023). According to the packaging, the product contained 600 mg mitragyna speciosa, 7.8 mg mitragynine (1.3% mitragynine content), and less than 0.27 mg 7-OH-mitragynine (<0.045% 7-OH-mitragynine content) per capsule (MOLLER000024). It was noted that the whole package contained a total of 234 mg mitragynine and less than 8.1 mg 7-OH-mitragynine (MOLLER000024).

28. The directions on the packaging stated to "[c]onsult your healthcare professional to determine if this product is right for you, and if so, how to use it properly" (MOLLER000024). The package included a disclaimer that stated "By using this product you accept full responsibility for any adverse events of health complications that may arise from its use. Manufacturers/Re-sellers assume no responsibility or liability for the use or misuse of this product"

(MOLLER000024). In addition, the package included a warning that stated "This product contains botanical kratom leaf capsules. Only for use as a botanical specimen. Mitragyna Speciosa is an unapproved dietary ingredient. The manufacturers/re-sellers of this product, therefore, cannot advise on its use. Ingesting Mitragyna Speciosa can be dangerous. Consult your physician about potential interactions, other possible complications, and precautionary measures before considering this product. By opening this package you accept full responsibility for the use of the product including but not limited to any adverse events or health complications. Inform your physician of the alkaloid content, labeled on the package. Manufacturers/Re-sellers assume no responsibility for the use or misuse of this product" (MOLLER000024)

29. In addition, a photo of one bag of Green Vein Maeng Da Silver O.P.M.S. Kratom 4 oz powder found in Ms. H. Moller's bag was provided (MOLLER_000025). According to the packaging, the product contained 13 mg mitragynine (1.3% mitragynine content) and less than 0.45 mg 7-OH-mitragynine (<0.045% 7-OH-mitragynine content) per gram of powder (MOLLER000026). It was noted that the whole package contained a total of 1474.2 mg mitragynine and less than 51 mg 7-OH-mitragynine (MOLLER000026).

30. The directions on the packaging stated to "[c]onsult your healthcare professional to determine if this product is right for you, and if so, how to use it properly" (MOLLER000026). The package included a disclaimer that stated "By using this product you accept full responsibility for any adverse events of health complications that may arise from its use. Manufacturers/Re-sellers assume no responsibility or liability for the use or misuse of this product" (MOLLER000026). In addition, the package included a warning that stated "This product contains botanical kratom leaf powder. Only for use as a botanical specimen. Mitragyna Speciosa is an unapproved dietary ingredient. The manufacturers/re-sellers of this product, therefore, cannot advise on its use. Ingesting Mitragyna Speciosa can be dangerous. Consult your physician about potential interactions, other possible complications, and precautionary measures before considering this product. By opening this package you accept full responsibility for the use of the product including but not limited to any adverse events or health complications. Inform your

physician of the alkaloid content, labeled on the package. Manufacturers/Re-sellers assume no responsibility for the use or misuse of this product" (MOLLER000026).

Expert Reports

Expert Report of Christopher Williams, PhD (dated November 17, 2025)

31. Dr. Willams stated that Ms. H. Moller's "… cause of death was officially documented as 'abnormally high left coronary ostia takeoff exacerbated by mitragynine use'. It [was] [his] opinion that the mechanisms of Kratom alkaloid activity support this conclusion" (Williams: pg. 2).

32. Dr. Williams reported that "… Kratom contains alkaloids which rival the potency of clinically available opioids like morphine in experimental models" (Williams: pg. 2). He added that:

> "… cardiotoxicity has been clearly linked to opioid usage including Kratom, which includes QT prolongation. Experimental models have shown that MG inhibits delayed rectifying K+ currents, similar to methadone, potentially leading to cardiac arrythmias. It is important to note that blockade of K+ signaling has been implicated in sudden cardiac death related to toxic opioid exposure" (Williams: pg. 3).

33. Dr. Williams stated that, according to a 2023 FAERS study:

> "… MG was associated with a greater incidence of ventricular arrhythmia/cardiac arrest than any other drug tested… In 39% of those cases, MG exposure was indicated in the absence of concurrent QTc prolonging drugs, thereby suggesting that MG was the primary driver of cardiotoxicity" (Williams: pg. 2-3).

34. Dr. Williams opined that kratom derived products:

> "… can lead to unintentional cases of polypharmacy, where drugs with overlapping off-target effects can result in significant toxicity. In the case of Harmony Moller, Kratom may well impact K+ currents in the heart and exacerbate the typically moderate effects of antihistamines like diphenhydramine or promethazine. Of note, the level of the diphenhydramine (64 ng/ml) and promethazine (18 ng/ml) are in accordance with

expected levels in therapeutic drug usage, suggesting that these drugs alone were not used at a level that should precipitate cardiorespiratory toxicity" (Williams: pg. 4).

Expert Report of Josef Thundiyil, M.D. (dated November 13, 2025)

35. Dr. Thundiyil stated that he agreed that "… mitragynine use contributed to and caused Harmony Moller's death" and that "… Harmony Moller's death was an adverse cardiorespiratory event caused by kratom" (Thundiyil: pg. 2-3). He added that "[t]he substances present in Harmony Moller's system at time of death are insufficient to cause [her] death in the absence of mitragynine" (Thundiyil: pg. 3).

36. Dr. Thundiyil reported that cardiac arrythmias were a documented symptom of kratom use and that kratom related deaths involve clinical findings consistent with cardiorespiratory arrest (Thundiyil: pg. 9). He added that:

> "[k]ratom also acts as a stimulant. Stimulants like kratom have the potential to cause enlargement of the heart. An enlarged heart is a heart that is electrically susceptible to arrythmias. The stimulant effects of kratom increase the likelihood of electrical instability and sudden cardiac death. The stimulant effect of kratom has been associated to occur at lower doses" (Thundiyil: pg. 9-10).

37. Dr. Thundiyil stated that Ms. H. Moller's conditions, including pulmonary edema and congestion, chronic passive congestion of the liver, mild nephrosclerosis, high origin of left coronary artery, and cardiomegaly, "… may have been co-morbidities for [her] but were not the primary cause of death. The primary cause of death was a cardiorespiratory event caused by kratom" (Thundiyil: pg. 10). He stated that "the concentration of mitragynine (150 ng/ml) found in Ms. H. Moller's blood was sufficient to cause adverse events" (Thundiyil: pg. 10).

38. Dr. Thundiyil stated that "… those selling the kratom frequently do not provide warnings or safe instructions for use and dosing" (Thundiyil: pg. 9).

Expert Report of Ronald H. Wharton, MD, FACC, FASE (dated November 4, 2025)

39. Dr. Wharton reported that:

> "[t]here are multiple reports in the medical literature that associate the use of kratom products with the development of ventricular tachycardia and ventricular fibrillation (VT/VF), which cause the heart to stop beating. This leads to the immediate cessation of circulation and death within a few minutes unless treated with defibrillation. [His] opinion [was] based on exclusion, as a cardiorespiratory death, such as cardiac arrhythmia, cannot be confirmed by autopsy or postmortem toxicology" (Wharton: pg. 2).

40. Dr. Wharton explained that adverse cardiac events such as VT/VF can be caused by exogenous substances and stated that a review of opioid-associated arrhythmia in North America published in 2023 "… demonstrated a 'disproportionate' association of 'life threatening arrhythmias in North America' with mitragynine" (Wharton: pg. 2). He noted that the "… literature discussing kratom consumption and its effects suggests a significant linkage between the main alkaloid in Mitragyna speciosa (kratom) use, ventricular arrhythmias (ventricular tachycardia, ventricular fibrillation) and cardiorespiratory adverse events" (Wharton: pg. 2-3).

41. Dr. Wharton reported that the "… high ostia take-off of the left coronary artery is a benign finding which was not a cause of [Ms. H. Moller's] sudden death" (Wharton: pg. 3). He stated that "… more likely than not, Harmony Moller died from a cardiorespiratory adverse event induced by consumption of kratom" (Wharton: pg. 3).

Expert Report of Evelyn Cadman (dated November 17, 2025)

42. Ms. Cadman stated that the "… supply, marketing, distribution, and sale of OPMS Kratom fails to comply with basic industry and regulatory standards of care for supplements, and in particular New Dietary Ingredients" (Cadman: pg. 6). She added that the kratom in this case was misbranded and that "[d]efendants violated the standards that prohibit the marketing, distribution, and sale of unapproved and new drugs" (Cadman: pg. 6-7).

43. Ms. Cadman stated that "… none of the named defendants ever verified that the OPMS kratom products manufactured and sold by them were reasonably safe for human consumption" (Cadman: pg. 8). She reported that "… OPMS kratom consumers, including Harmony Moller, were not properly warned of the serious risks from Kratom, including the risks

of seizures, cardiorespiratory adverse effects, death, dependence and addiction, and drug-drug interactions (Cadman: pg. 13).

Expert Report of Ryan J. Clark (dated November 18, 2025)

44. Mr. Clark stated that:

"1. Defendants' failure to produce internal financial information and income tax returns is unusual and provides no basis for verifying their financial representations.

2. Mr. Gabbay's and Jopen's financial control and alleged payments on behalf of Martian Sales lack appropriate accounting and documentation and indicate commingling between the two entities.

3. Jopen's financial contributions to the American Kratom Association further illustrate and irregular business practice and irregular accounting practices.

4. Mr. Gabbay and/or Jopen exercises significant control over Martian Sales and other various aspects of the kratom business, despite having no equity ownership in Martian Sales, which is highly unusual.

5. The financial practices and intercompany conduct between Jopen and Martian Sales are consistent with factors commonly identified in forensic accounting investigations as indicators of a lack of corporate separateness" (Clark: pg. 6).

Expert Report of Sarah D. Kowitt, PhD (dated November 12, 2025)

45. Dr. Kowitt stated that the "… warnings on the kratom packages discovered near Ms. Moller were not adequate" (Kowitt: pg. 2). She reported that the "… Defendant's warnings did not warn about risks associated with product use," "… did not provide clear instructions on how to avoid risks associated with product use," "… did not use language that was clear or understandable," and "… did not optimally capture or hold attention" (Kowitt: pg. 2-4).

46. Dr. Kowitt stated that "… the manufacturers in this case knew or should have known that users could be exposed to a risk of significant injury or death under normal conditions of use. The warnings provided did not reasonably inform an ordinary user of the nature and extent of the risk of death or serious health risks" (Kowitt: pg. 5).

OVERVIEW OF MY OPINIONS

Opinion 1 - People in numerous countries have used kratom (Mitragyna speciosa) safely for hundreds (if not thousands) of years. The body of scientific literature on kratom and its alkaloids continues to evolve, but historic and recent data indicate that beneficial effects associated with kratom use have a robust pharmacologic basis. It is my opinion that few to no adverse effects are expected in typical users when kratom is used alone and that adverse outcomes in dual- and polysubstance users can be attributed to other compounds, not kratom or its alkaloids.

Opinion 2 - The risk assessment process is a methodology that is widely accepted and used by many scientific bodies and governmental agencies around the world. It is my opinion that it is an appropriate method to use for characterizing the health risks potentially associated with use of kratom products (especially when comparing potential health risks associated with kratom use to those associated with use of opioids to yield similar effects).

Opinion 3 - The health effects of kratom can be attributed to more than 50 indole alkaloids present in the plant. Of these, mitragynine and 7-hydroxymitragynine are the most biologically significant. It is my opinion that alkaloid concentrations vary across kratom product types, but data from the peer-reviewed literature indicates that these typical concentrations contained in teas or capsules, taken in a reasonable manner, would not increase the risk of acute or chronic adverse health effects among typical users.

Opinion 4 - Animal studies and studies of humans are defined as *in vivo* analyses. Kratom, mitragynine, 7-hydroxymitragynine, and other associated alkaloids have been studied *in vivo* for over 100 years. It is my opinion that these studies provide support for two primary conclusions. First, kratom and its alkaloids can yield dose-dependent stimulant or pain relief effects under typical human use and may aid with opioid withdrawal. Second, kratom alkaloids can be classified as very low to moderately toxic in animals. It is my opinion that since these alkaloids are only a small percentage of kratom products by weight, few to no adverse health effects are expected in kratom users, even among higher volume consumers. No chronic adverse

effects have been reported *in vivo* following repeated kratom administration at relatively high doses to test animals or humans.

Opinion 5 - Studies of isolated cells, termed *in vitro* analyses, help provide information on potential organ-specific effects and the toxicological mechanisms driving health outcomes following exposure to a given compound. *In vitro* data indicate that kratom alkaloids are primarily metabolized by Cytochrome P450 (CYP) isoforms CYP3A4 and CYP2D6 and that physiological effects are driven by alkaloid activity at opioid, adrenergic, serotonergic, and dopaminergic receptors. Kratom's pain relief (analgesic) and euphoric effects can be explained by kratom alkaloid binding at opioid receptors (ORs). However, it is my opinion that the activity of mitragynine, 7-hydroxymitragynine, and other kratom alkaloids at ORs is weak relative to that of numerous drugs (e.g., morphine, fentanyl) and endogenous endorphins (e.g., β-endorphin, enkephalins, dynorphins). However, depending on the dose, 7-hydroxymitragynine can cause morphine like effects if ingested in a relatively pure state (i.e., as a synthetic isolate).

Opinion 6 - Data on the effects of kratom use in humans includes epidemiological evidence, as well as information from case reports and case series. It is my opinion that human studies, although limited, indicate that kratom use is associated with potential benefits including stimulant effects, pain relief, and as a substitute or withdrawal aid for opioids at doses yielding few to no adverse behavioral or physiological effects. Case data indicate that kratom's alkaloids produce few to no acute adverse health effects when kratom is used alone, but that mitragynine and 7-hydroxymitragynine have been reported along with other compounds in the post-mortem blood and tissues of dual- and polysubstance users.

Opinion 7 - Based on the records regarding Ms. Moller's passing, it is likely that she experienced an adverse cardiovascular event, which led to her death. Kratom has not been found to increase the risk of heart attacks in humans. It is my opinion that, should it be determined that Ms. Moller experienced a heart attack or comparable cardiovascular event which resulted in her death, kratom would not have been the cause.

Opinion 8 – It is possible to assess the health risks of using specific kratom products by considering various exposure scenarios and by combining multiple lines of evidence from available scientific literature. Based on this information, it is my opinion that estimated whole-body and compartment-specific alkaloid doses for typical kratom users are 3-fold to 50,000-fold below those expected to increase the risk of adverse health effects in animals, depending on the types of products used. This is a conservative estimate, as animals are often more sensitive to potentially adverse effects of chemical exposures than humans. It is my opinion that, given the available literature, typical doses of kratom, such as the ones that Ms. Moller potentially ingested, would not increase her risk of death. Additionally, her postmortem blood concentration of mitragynine of 150 ng/mL is comparable to blood concentrations observed in human volunteers following kratom doses that are documented to be well-tolerated in humans with no adverse effects.

BASES FOR OPINIONS

Basis of Opinion 1

M. Speciosa Traditional Use

47. *Mitragyna speciosa*, commonly known as kratom, is a tree native to Southeast Asia, specifically, Thailand, Indonesia, Malaysia, Borneo, Myanmar, and Vietnam. *M. speciosa* can reach up to 25 meters tall at maturity and is noted for having dark green leaves with green, white, or red veins (Cinosi et al. 2015). It conventionally grows in swampy areas, in areas inundated by water, and alongside riverbanks (Grundmann 2017; Ahmad et al. 2022). Depending on the country, kratom is referred to by its local name: ketum or biak (Malaysia), keton or kadamba (Indonesia), benisa (Myanmar), mambog or lugub (Philippines), ithang, thom, kratom (Thailand), or giam (Vietnam) (Gong et al. 2012; Cinosi et al. 2015; Grundmann 2017; Eastlack et al. 2020).

48. Kratom leaves were historically used in rural communities by farmers, laborers, or fishermen in Southeast Asia as a stimulant to reduce or prevent fatigue stemming from hard labor by chewing the leaves raw or drying them for later consumption (Cinosi et al. 2015; Grundmann 2017; Singh et al. 2017). Although less common in Southeast Asia, it is reported that the dried

leaves of kratom can also be smoked to achieve a relaxing effect (Grundmann 2017). Medicinally, kratom was traditionally used to treat symptoms of substance abuse disorders (including opium withdrawal), headaches, toothaches, diarrhea, cough, fever, stroke symptoms, sleeplessness, typhoid, cholesterol issues, stimulate appetite, pain, cough, hypertension, diabetes, anxiety, as a wound poultice, and as a deworming agent (Gong et al. 2012; Grundmann 2017; Eastlack et al. 2020; Ahmad et al. 2022). Very few of these benefits have been studied in clinical settings.

49. Apart from medicinal uses, Kratom was also utilized for centuries for social purposes and to perform cultural and religious ceremonies in Southeast Asia (Cinosi et al. 2015; Singh et al. 2017). It was reported that kratom was used as a substitute for alcohol in predominately Muslim communities in Malaysia and South Thailand (Singh et al. 2017; Charoenratana et al. 2021). In Thailand, kratom has been recognized as a culturally significant plant as of the 19th century (Charoenratana et al. 2021).

50. Plants in the genus, *Mitragyna,* that are native to Africa, historically were used to treat various disease, such as malaria, rheumatism, cardiovascular disease, amenorrhea, inflammation, headaches, bronchopulmonary disease, and diabetes mellitus (Ahmad et al. 2022). The first description of kratom as *Mitragyna* by a western scientist came from Dutch botanist Pieter Willem Korthals, who travelled through Southeast Asia with the Dutch East India Company Commission for Natural Science from 1830 to 1837 (Ahmad et al. 2022). Mr. Korthal eponymously described the now commercially significant cultivar of kratom, *Mitragyna speciosa Korth,* though several other species are also classified within the genus.

M. Speciosa Globally

51. Brown et al. (2017) conducted a Google trends search for the term "kratom" and reported that the number of searches had increased steadily since 2004, and the United States, Canada, Austria, and Germany held the greatest number of queries. Garcia-Romeu et al. (2020) conducted a "… cross section, anonymous online survey …" between January and December 2017 to characterize the demographic trends among kratom users predominantly in the U.S. (97%). The authors reported that among 2,798 self-reported kratom users with a mean age of 40, 90% were

white and 61% were women (Garcia-Romeu et al. 2020). This means that the kratom user population is both older, less racially diverse, and more female than, for instance, groups using illicit drugs.

52. Singh et al. (2017) reported that in Malaysia, kratom has been banned under the Poisons Act since 1952, and in Thailand it has been criminalized since 1943. In 1979, the Thai government lessened the punitive actions related to kratom, and 2018 lifted the ban of use, production, and possession for medicinal use (Singh et al. 2017; Veltri and Grundmann 2019). In 2021, Thailand passed an update to the Narcotics Act (No. 8) B.E. 2564 (Office of the Narcotics Control Board 2021) allowing for possession and consumption of kratom and eliminating kratom-related offenses and in 2022, Thailand passed the Kratom Plant Act B. E. 2565 (Office of the Narcotics Control Board 2022) regulating the trade, sale, consumption, study, and advertising of kratom in Thailand. Currently, kratom use and possession is controlled in several European countries including, Denmark, Finland, Lithuania, Poland, Romania, Sweden, and the UK (Singh et al. 2016; Singh et al. 2017). Despite its legal status at the time, a 2012 cross-sectional study found that Kratom consumption was prevalent in Thailand and Malaysia, with 88% of respondents reporting consuming Kratom daily, most often through tea (Ahmad and Aziz 2012).

M. Speciosa in the United States

53. Multiple studies have reported that kratom was first introduced in the United States in the 1980s or 1990s and gained popularity in the U.S. in the last several decades (Henningfield et al. 2018; Kruegel and Grundmann 2018; Veltri and Grundmann 2019). It was reported that the introduction of kratom to the United States could have been fueled by the increase in migration from Southeast Asia to the U.S. at that time (Henningfield et al. 2018; Veltri and Grundmann 2019).

54. Grundmann (2017) presented a synopsis on kratom that estimated the number of users in the U.S. and reported that it ranged from three to five million based on membership numbers obtained from the American Kratom Association. A more recent study conducted by Swogger et al. (2022) found that up to 10 - 16 million people in the United States used kratom.

Researchers and various health agencies estimated current kratom use prevalence at 1.3-6.1% (increased from past-year use prevalences of 0.6% and 0.8% in 2019 and 2021, respectfully) (Palamar 2021; Schimmel et al. 2021; Substance Abuse and Mental Health Services Administration 2022; Swogger et al. 2022).

55. Bath et al. (2020) reported on a cross-sectional, anonymous United States national online survey that collected 10,000 responses in October 2016 related to demographics, health, and kratom use patterns. Their results showed that 72% of kratom users were 31 years and older, 54% were married or partnered, 57% were male, 57% employed, and 89% self-described as white non-Hispanic (Bath et al. 2020).

Regulations Concerning Kratom in the United States

56. The FDA has not significantly evaluated any kratom processors or distributors in the U.S., nor has it studied the validity of the associated effects (including alleviation of opioid withdrawal symptoms, pain treatment, and fatigue). No medications (over-the-counter or prescribed) which containing kratom or its active alkaloids have been approved by the Agency (U.S. Food and Drug Administration (FDA) 2024).

57. The FDA considers kratom as: (U.S. Food and Drug Administration (FDA) 2024).

> "a new dietary ingredient for which there is inadequate information to provide reasonable assurance that such ingredient does not present a significant unreasonable risk of illness or injury and, therefore, dietary supplements that are or contain kratom are adulterated under section 402(f)(1)(B) of the Food, Drug, and Cosmetic Act (FD&C)" (U.S. Food and Drug Administration (FDA) 2024)

58. In 2016, the DEA moved to regulate kratom as a Schedule 1 compound under the Controlled Substances Act (U.S. Drug Enforcement Administration (DEA) 2016; Commons 2018; Henningfield et al. 2018; Corkery et al. 2019). The Agency received comments from thousands of individuals and stakeholders against the classification and in support of kratom's continue legality; these comments cited the benefits experienced by kratom users, including eased withdrawal from addictive substances and other ailments (Henningfield et al. 2018; Corkery et al. 2019). The DEA

subsequently did not classify kratom as a Schedule 1 substance, and it remains unregulated at the federal level as of 2024.

State-Level Regulations in the United States

59. As of early 2024, 22 states and the District of Columbia regulate kratom, mitragynine, and/or 7-hydroxymitragynine in "some manner"; kratom components are Schedule I controlled substances in Alabama, Arkansas, Indiana, Rhode Island, Wisconsin, and Washington D.C. and "regulated drugs" in Vermont (Legislative Analysis and Public Policy Association (LAPPA) 2024). In 16 of these states, the possession, sale, and/or manufacture of kratom products is regulated, all of which include age restrictions at either 18 or 21, and 10 of these states regulate kratom labelling on some level (Legislative Analysis and Public Policy Association (LAPPA) 2024). Nevada legalized the sale of kratom in 2019 to individuals over the age of 18 years old (NRS 597.998). Louisiana began to regulate kratom as a controlled substance in August of 2025. It should be noted that this occurred after Ms. Moller's passing.

60. The rest of the U.S. (28 states) does not regulate kratom or consider it or its components a controlled substances (Legislative Analysis and Public Policy Association (LAPPA) 2024). However, thirty-three states have proposed new legislation regarding either the regulation of kratom products and/or the scheduling of kratom components in 2023 and 2024 (Legislative Analysis and Public Policy Association (LAPPA) 2024).

Why People Use Kratom

61. Users of kratom in the United States note similar motivations for use to Southeast Asian users, including to self-treat pain, as an opioid substitute, and for mood disorders (Eggleston et al. 2019; Smith et al. 2022). Kratom consumption is reported in combination with use of other compounds such as cannabis, vaped nicotine, and in some instances buprenorphine (Smith et al. 2022). (Smith et al. 2022) surveyed kratom users in the U.S. to determine kratom-use initiation, sourcing, motivations, preference, and stigma. The authors reported that the average age of use initiation was 30 years old, and of 129 individuals (51.9% female, white 71.9%), kratom was ranked as a preferred substance by 48.5% (Smith et al. 2022). While the authors reported the

strongest product association with past-year kratom use was vaped nicotine, they also concluded that more research would clarify the potential benefits of using the whole leaves and "… select alkaloids with less risk when compared to traditional full opioid agonists" (Henningfield et al. 2018; Grundmann et al. 2021; Smith et al. 2022). Collectively, these data indicate that many United States kratom users are utilizing these products as an alternative to high-risk compounds linked to diverse adverse health effects. This means that, among certain user groups and at certain doses, the availability of kratom may contribute net benefits to public health.

Kratom Products

Traditional Preparations

62. In Southeast Asia, people consume kratom by smoking or chewing the plant's leaves and via brewing a tea to drink (Singh et al. 2016). Leaves are usually chewed multiple times throughout the day depending on the needs of users (Singh et al. 2017). People in Thailand commonly chew the kratom leaves while people in Malaysia more frequently consume kratom as a juice (Singh et al. 2016). Kratom juice is prepared by boiling "older" kratom leaves for a maximum of 4 hours. Once brewed the juice is left to cool before being packed in bags and sold (Singh et al. 2017)

63. Relative to more recreational uses, in Asia, some users reportedly prepare homemade cocktails containing three ingredients including kratom, a caffeine-containing syrup, and codeine or diphenhydramine; the drink is reported to elicit alcohol-like effects (Tanguay and International Drug Policy Consortium 2011; Hassan et al. 2013). In other instances, sugar and sweet beverages are sometimes added to kratom teas to mask the bitter taste, lemon juice can be added to kratom teas to facilitate the extraction of the plant alkaloids, and salt is added to prevent constipation (Hassan et al. 2013; Singh et al. 2017). The dried leaves are also commonly powdered and boiled to create a syrup that is then mixed with palas palm *(Lincuala paludosa)* and made into pills (Hassan et al. 2013). These pills are commonly smoked in Malaysia using a bamboo pipe (Hassan et al. 2013).

Products Sold in the United States

64. Kratom products have been sold in the U.S. for approximately 20 years and continue to increase in popularity. Kratom is often purchased as capsules, powders, or liquid extracts, along with the aforementioned traditional teas and juices (Prozialeck et al. 2012; Singh et al. 2016). Though less popular, kratom-containing vapes (similar to e-cigarettes) have gained attention as of early 2024, as have resins and gummies. Additionally, kratom has been marketed as an herbal dietary supplement (Singh et al. 2016). Kratom can be bought online and in "head" shops or gas stations across the country (Singh et al. 2016). Kratom is widely promoted as a legal psychoactive product on numerous websites in the U.S. (U.S. Drug Enforcement Administration (DEA) 2024). These websites post vendors listings, tea preparation instruction, recommended doses, alleged medicinal uses, and user reports of herbal-associated experiences (U.S. Drug Enforcement Administration (DEA) 2024).

Kratom Alkaloids

65. *M. speciosa* contains more than 40 different alkaloids, including mitragynine and 7-hydroxymitragynine (7-HMG), which have agonist opioid-like effects on opioid receptors (Firmansyah et al. 2021; Ahmad et al. 2022). Firmansyah et al. (2021) reported that six of the more than 40 alkaloids within kratom have psychoactive properties, such as mitragynine, 7-HMG, mitraphylline, speciociliatine, speciogynine, and paynantheine. Similar to alkaloids derived from the *Coffea* species and other members of the Rubiaceae, kratom-derived alkaloids have dose-dependent stimulant properties (Ahmad et al. 2022).

66. Brown et al. (2017) conducted a systematic review of literature regarding the chemical contents of kratom and reported as many as 57 secondary metabolite compounds within the species, including 37 that were found only in *M. Speciosa*. It was reported that indole and oxindole alkaloids were the chemical constituents present in kratom that were the most likely cause of the plant's pharmacological effects. The very low concentrations of most of these alkaloids in kratom leaves and derivative products make them of little toxicological concern. Moreover, apart from mitragynine and 7-hydroxymitragynine, little toxicological data (*in vivo, in vitro,* or otherwise) exists on these alkaloids.

67. The noted variance in alkaloid content based on kratom leaf maturity, growing condition, time of harvest, plant strain, and location can significantly influence the pharmacological effects seen among users (Shellard et al. 1978; Boffa et al. 2018; Zhang et al. 2020; Leksungnoen et al. 2022; Sengnon et al. 2023). That is, some products, including raw leaves, may contain mitragynine, 7-hydroxymitragynine, speciociliatine, and speciofoline concentrations more than several-fold higher than similar products sourced from other areas or supply chains, or even from the same product from a single kratom distributor or processor. Some companies have implemented quality controls and testing procedures for heavy metal contamination and alkaloid content (Bigtrea 2023; Hill et al. 2023; 1836 Kratom 2024). Standardization of protocols promoting consistent concentrations in end products could greatly improve industry norms.

Background on Kratom Toxicology and Pharmacology

Beneficial Effects

68. Kratom is commonly used as a stimulant, prompting increased stamina and pain alleviation, and for management of symptoms of opioid withdrawal (Prozialeck et al. 2012; Hassan et al. 2013; Warner et al. 2016). Additionally, it is used by those with hypertension and for diarrhea, cough, and fever (Singh et al. 2016; Swogger et al. 2022). Some of these effects are anecdotal, and kratom's pharmacological utility has not been studied in well controlled clinical trials as of early 2024. However, existing studies have demonstrated the effectiveness of kratom beyond placebic outcomes, with studies indicating that the hallmark effects of kratom, such as analgesia and pain and anxiety relief, are likely genuine.

69. Both studies and anecdotal evidence support the potential for kratom and its active alkaloids to act as reduced-harm products for individuals previously addicted to opioids and other substances, along with mitigating HIV risk behaviors (Prozialeck et al. 2019; Grundmann et al. 2021; Prozialeck et al. 2021; Sharma and McCurdy 2021; Wilson et al. 2021; McCurdy et al. 2024). Kratom may act as a substance replacement, comparable to how e-cigarettes are viewed as a reduced harm alternative to traditional cigarettes within the U.S. (Balfour et al. 2021; Friedman and Warner 2022; Warner et al. 2023).

70. A study reported that the death rate attributable to past-year kratom use was 0.0369-0.0544 per 100,000 users, utilizing American Kratom Association (AKA) import statistics and assuming 4-6 g/day consumed. When compared to the estimated death rate from past-year use of any opioid (417 per 100,000) and heroin (4,778 per 100,000), it was concluded by the authors that risks associated with kratom use were thousands-fold lower than those of opioid and heroin use (Henningfield et al. 2019). In spite of this report, most experts have not found it possible to attribute a death due only to kratom use. In virtually every case, other drugs were found that may have interacted with kratom or, alone, could be responsible for the deaths. Often, deaths were due to the person passing out or having a seizure and then died due to injuries to the head. These deaths are often attributed, erroneously, to the kratom.

Potential Adverse Effects

71. When data on the toxicity of kratom and its alkaloids is viewed relative to historic toxicological classifications, it can be classified as moderately toxic (Hodge and Sterner 1949). However, when animal data are considered, traditional kratom preparations (e.g., teas, leaves) represent a low acute or repeated dose toxicity profile. Animal studies have consistently determined that there are little or no chronic adverse effects expected from kratom use at typical doses, and animals exposed to high levels of kratom recover quickly after exposure ceased. It should be noted that kratom and mitragynine have been cited as factors in several studies of overdose deaths across the U.S. (Olsen et al. 2019). However, these results are confounded in that nearly all users had taken other substances as well.

72. Both the National Institute of Drug Abuse (NIDA) and the United Nations Office on Drugs and Crime (UNODC) have noted the addictive potential and prevalence of withdrawal symptoms following kratom use but recognized the reduced addictive potential and mild withdrawal symptoms relative to opioids (Commission on Narcotic Drugs 2021; National Institute on Drug Abuse 2023). However, the UNODC reported that abuse liability was not consistent throughout the available animal studies (Commission on Narcotic Drugs 2021). Reported potential withdrawal physical symptoms in humans are excessive tearing, aching of muscles and bones,

jerky limb movements, frequent urination, constipation, nausea, sweating, chills, and cravings, along with psychological symptoms, including hostility, aggression, inability to work, insomnia, anxiety, fatigue, irritability, mood disturbance, and hallucinations (Hassan et al. 2013; Stanciu et al. 2019). Withdrawal symptom outcomes are reportedly dependent on duration and frequency of kratom use (Stanciu et al. 2019).

Impacts of Polysubstance Use

73. Post et al. (2019) reviewed kratom exposure reported to the United States National Poison Data System from 2011 to 2017. It was reported that 1,807 reports were made to U.S. Poison Control Centers (PCCs) during the examined period, with 65% of these cases representing single-substance use and the remaining 35% representing multi-substance or poly drug use.

74. As reported by Prevete et al. (2023)

> "While in Asia kratom has been used with a limited number of associated health hazards, most of the safety issues have been reported in the West, mainly among users ingesting kratom on a daily basis in combination with other drugs, such as prescription medicines (e.g., antipsychotic, antidepressant, and hypnotic drugs, among others) and psychoactive substances (e.g., amphetamine, methamphetamine, cyclopropyl fentanyl, opioids, ethanol)" (Prevete et al. 2023)

75. Put simply, the few adverse health effects reported among kratom users in Western countries are generally associated with simultaneous use of other, more hazardous substances, prescription or otherwise. These adverse effects are rare to nonexistent when kratom is used in a traditional capacity. Additionally, toxicity associated with kratom use has been attributed to contaminants including unscheduled substances, microbes, and toxic metals (Prevete et al. 2023). For instance, Smith et al. (2022) reported mental and psychological distress among kratom users, specifically those who combined kratom with nonmedical opioids and methamphetamine. The evidence suggests that kratom and its alkaloids and metabolites have the potential to alter the dose at which other consumed substances have life-threatening effects. It is difficult to attribute any adverse health effects to kratom use alone in many studies, due to the likelihood of users

simultaneously using other substances, such as illicit drugs or alcohol, or even contaminants within certain kratom products.

Basis of Opinion 2

Introduction to the Risk Assessment Process

76. The risk assessment process is a methodology that is widely accepted and used by many scientific bodies and governmental agencies around the world. It is an appropriate method to use for characterizing the health risks potentially associated with use of kratom products (especially when comparing potential health risks associated with kratom use to those associated with use of opioids to yield similar effects).

77. Over the past 50 years, risk assessments have received widespread attention and acceptance within the scientific and regulatory communities (Starr 1969; National Research Council (NRC) 1983; Commission on Risk Assessment 1997). In its 1983 report, the National Academy of Sciences defined "risk assessment" as:

> "[C]haracterization of the potential adverse health effects of human exposures to environmental hazards. Risk assessments include several elements: description of the potential adverse health effects based on an evaluation of results of epidemiologic, clinical, toxicologic, and environmental research; extrapolation from those results to predict the type and estimate the extent of health effects in humans under given conditions of exposure; judgements as to the number and characteristics of persons exposed at various intensities and durations; and summary judgements of the existence and overall magnitude of the public-health problem. Risk assessment also includes characterization of the uncertainties inherent in the process of inferring risk" (National Research Council (NRC) 1983).

78. The endorsement of the risk assessment methodology is discussed in the three documents issued by the National Academies of Sciences over the past 40 years (National Research Council (NRC) 1983, 1994, 2009).

79. Risk assessment comprises four major steps: hazard identification, dose-response assessment, exposure assessment, and risk characterization (National Research Council (NRC) 1983).

80. **Hazard identification** is the most recognized of the actions of regulatory agencies. It is defined here as the process of determining if human exposure to an agent could cause an increase in the incidence of a health condition (e.g., cancer, birth defect), or if a nonhuman receptor (e.g., fish, birds, other wildlife) might be adversely affected following exposure. It involves characterizing the nature and strength of the evidence of causation. Although the question of whether a substance causes cancer or other adverse health effects in humans is theoretically a yes-no question, there are few chemicals or physical agents with definitive human data (National Research Council (NRC) 1983). Therefore, the question is often restated in terms of effects in laboratory animals or other test systems: Does the agent induce cancer in test animals? Positive answers to such questions are typically taken as evidence that an agent may pose a cancer risk for an exposed human. Information from short-term in vitro tests and structural similarity to known chemical hazards may, in certain circumstances, be considered adequate information for hazard identification.

81. **Dose-response assessment** is the process of characterizing the relationship between the dose of an agent administered or received and the incidence of an adverse health effect as a function of exposure to the agent. This process considers factors such as intensity of exposure and age pattern of exposure, along with variables that might affect response such as sex, lifestyle, and other modifying factors. A dose-response assessment usually requires extrapolation from high to low doses, from animals to humans, and from laboratory animal species to wildlife species. A dose-response assessment should describe and justify the methods of extrapolation used to predict incidence, and it should characterize the statistical and biologic uncertainties in these methods.

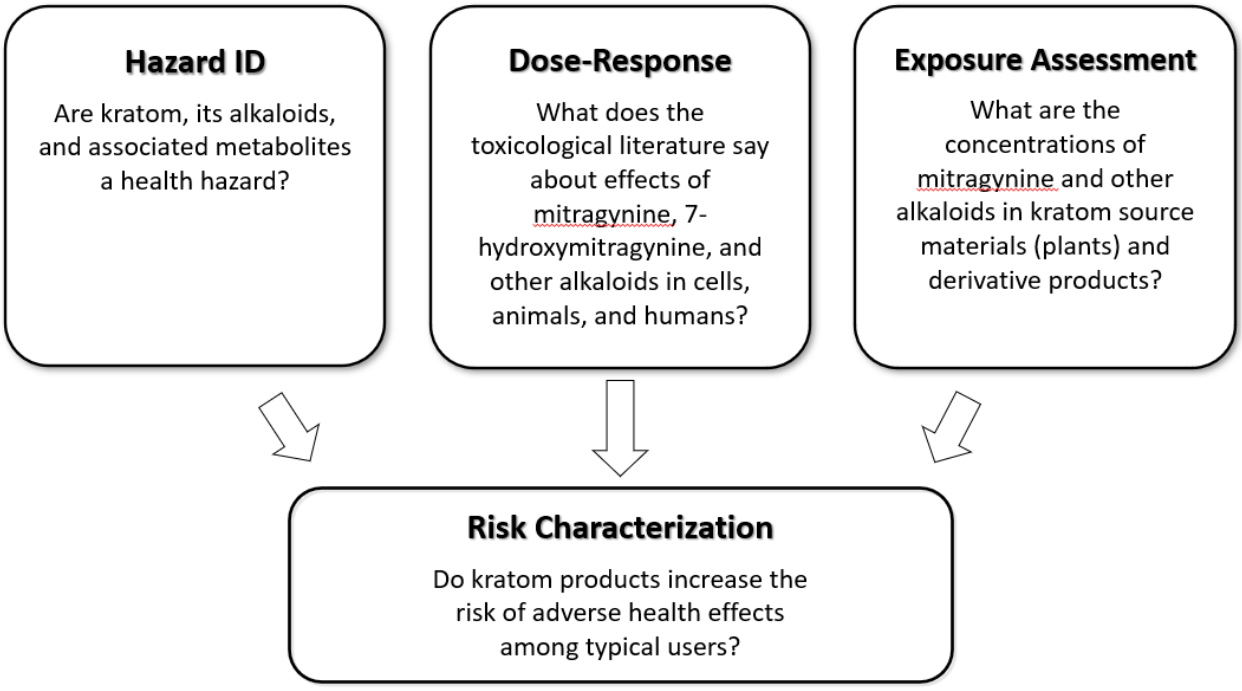
82. **Exposure assessment** is the process of measuring or estimating the intensity, frequency, and duration of human or animal exposure to an agent currently present in the environment, or of estimating hypothetical exposures that might arise from the release of new

chemicals into the environment. In its most complete form, an exposure assessment should describe the magnitude, duration, schedule, and route of exposure; the size, nature, and classes of the human, animal, aquatic, or wildlife populations exposed; and the uncertainties in all estimates. An exposure assessment can often be used to identify prospective control options and to predict the effects of available control technologies on exposure control.

83. **Risk characterization** is the process of estimating the incidence of a health effect under various conditions of human or animal exposure described in the exposure assessment. It is performed by combining the exposure and dose-response assessments. The summary effects of the uncertainties in the preceding steps should be described in this step (National Research Council (NRC) 1983).

84. A graphical representation of how this approach applies to kratom can be seen in Figure 1:

Figure 1. Risk assessment approach specific to kratom products.



Use of Risk Assessments

85. The Federal government has used the risk assessment process since as early as the 1950s. The FDA commissioner at the time, Arnold Lehman, wrote the first guidelines on how to make decisions when evaluating the safety of drugs, which later became known as the risk assessment process (Paustenbach et al. 1990).

86. Between the 1950s and 1970s, risk assessments were conducted by various government agencies but were often not identified as "risk assessments." The process was codified in 1983 by the National Academy of Sciences in what has been called "the Red book" (e.g., Risk Assessment in the Federal Government). Since then, risk assessment has been used by virtually every government agency in the United States and by various entities abroad. The general guidance has been routinely updated.

87. Agencies including OSHA, FDA, and CPSC use the risk assessment process any time there is concern about a chemical or radionuclide in any media including food, air, water, sediments, pharmaceutical agents, agricultural agents, medical implants, cosmetics, and consumer products, among other applications. It is essentially the foundation for regulating or characterizing the risk posed by virtually any product made from chemicals.

Experience Designing Risk Assessments

88. Over the past 40 years, since the beginning of the era of formal health risk assessment in 1983, I have conducted more than 1,000 health risk assessments and have published more than 100 papers and more than 50 book chapters related to the field of health risk assessment. I was a founding member of the Society for Risk Analysis (the official society for this profession) and, later, was an officer. I wrote the first textbook on health risk assessment, which was published in 1988, with a sequel released in 2002. It has been the best-selling text on this topic. A new edition became available in 2024 (Paustenbach 2024).

89. Over the years, I have conducted hundreds of risk assessments related to contaminated sediments, contaminated foods, consumer products, the workplace, ambient air, drinking water, soils, house dust, and other media. Beginning with my role as a college instructor

in 1978, and throughout my work over the past 43 years, I may have introduced the field of risk assessment to more full-time practitioners than anyone in this field.

90. Beginning in 1984, I was manager of one of the first health risk assessment groups in corporate America when I worked for Stauffer Chemical in Connecticut. I frequently collaborated with Dow Chemical, who pioneered this field within the private sector. In that role, our team assessed the risks of various pesticides, consumer products, commercial products, and air emissions. I also assessed a number of hazardous waste sites during that time.

91. Next, I was recruited by Syntex Pharmaceuticals to lead their newly formed health risk assessment group. In that role, I had the privilege of leading efforts to figure out how to manage the Times Beach site in Missouri, as well as 37 other dioxins-related sites. I was also responsible for reducing occupational exposure to several pharmaceuticals during their manufacture following observation of various occupational diseases in the cohorts.

92. After those challenges were addressed, I formed ChemRisk, one of the first consulting firms, if not the first, dedicated solely to applying risk assessment methodologies to tackle occupational and environmental health issues. ChemRisk eventually became the largest risk assessment group in the world within the private sector. Over the 30+ years I led the firm (or the group at Exponent, a scientific consulting firm), more than 600 professionals were taught the profession within that firm. Many of them have since become leaders in industrial hygiene, exposure science, epidemiology, toxicology, and risk assessment.

93. In April 2019, I left ChemRisk, following an earlier acquisition by Cardno, and founded Paustenbach and Associates, which was devoted to applying the fields of toxicology, occupational hygiene, engineering, and risk assessment to the characterization of chemical or radiological hazards. In 2024, Paustenbach and Associates was acquired by TRC, where I continue to perform the same type of work and manage our group focused on occupational and environmental hazards.

94. Over the years, I have been an adjunct professor in at least five different universities teaching risk assessment, toxicology, industrial hygiene, environmental law, and exposure science.

In my opinion, it is safe to say that I have been a team member in conducting assessments on at least half of the most challenging sites, chemicals, or consumer products that have been conducted over the past 40 years in the United States.

Basis of Opinion 3

The Health Effects of Kratom Can Be Attributed to More Than 50 Indole Alkaloids

95. The health effects of kratom can be attributed to more than 50 indole alkaloids present in the plant. Of these, mitragynine and 7-hydroxymitragynine are the most biologically significant. It is my opinion that alkaloid concentrations vary across kratom product types, but data from the peer-reviewed literature indicates that these typical concentrations contained in teas or capsules, taken in a reasonable manner, would not increase the risk of acute or chronic adverse health effects among typical users.

96. The health effects of kratom are primarily attributed to its diverse array of alkaloids, with mitragynine and 7-hydroxymitragynine being the most prominent (Grundmann 2017; Singh et al. 2017; Eastlack et al. 2020). These alkaloids interact with opioid receptors in the brain, which can produce pain-relieving, euphoric, and sedative effects.

97. A study conducted by Garcia-Romeu et al. (2020) describes kratom use patterns among regular users in the United States, for both medicinal and recreational consumption. The majority of respondents reported moderate dosing, with 49.0% consuming 1–3 grams per dose and 33.4% consuming 4–6 grams across product types, which the study design and convenience sample did not distinguish. Smaller groups reported using less than 1 gram (8.6%) and more than 7 grams (8.9%) per dose. The study also revealed high frequency of use, with 59.1% of participants using kratom daily, 18.2% of participants using kratom 4-6 times per week, and 13.4% of participants using kratom 2-3 times per week. The preferred methods of consumption were as a prepared beverage (37.0%), ingested as raw powder (43.6%), and in capsule form (18.9%). The authors reported that this varied consumption underscores the integration of kratom into daily routines to potentially manage pain or as an alternative to opioids. The modes of consumption influence the pharmacokinetics of kratom's alkaloids, affecting both therapeutic outcomes and the potential for

adverse effects, highlighting the need for careful consideration of kratom's use, regulatory oversight, and further research to understand its full implications and mitigate associated risks (Garcia-Romeu et al. 2020).

Kratom Dosing

98. The dosing of kratom for humans varies significantly based on its intended effects, which range from stimulation to sedation. Low doses of approximately 1-5 grams of kratom leaves are considered stimulating, similar to the effect of drinking coffee, providing increased alertness and physical energy (Warner et al. 2016). Medium doses ranging from 5 to 15 grams produce more opioid-like effects, such as pain relief and euphoria, but can also cause side effects such as dizziness and nausea. High doses exceeding 15 grams tend to produce sedation and can cause severe side effects, including stupor and potentially toxic effects (Warner et al. 2016).

99. Online websites usually advertise kratom as a legal substitute for opioids and other substances, claiming various therapeutic benefits. According to an online post, "Kratom dosage recommendations typically range from 2 to 12 grams, with doses under 2 grams considered microdoses and doses over 6 grams considered high doses" (Moore 2024b). The website reported that the self-treatment of depression symptoms could be treated with kratom doses between 2 and 6 grams while doses for mild to moderate chronic pain were between 4 and 8 grams. The recommended dosage for opioid withdrawal was between 2 and 12 grams, for anxiety was up to 6 grams, and for energy and focus was between 2 and 4 grams (Moore 2024b)

Kratom Alkaloids

Mitragynine

100. Mitragynine is the most abundant alkaloid in kratom, accounting for approximately 60-70% of the alkaloid content of most such plants. It acts on the mu-opioid receptors (μORs) as a partial agonist (Prozialeck et al. 2012). This interaction is responsible for the analgesic properties of kratom but with a reportedly lower risk of respiratory depression compared to opioids (Prozialeck et al. 2012). The mechanism underlying this is discussed later in my report. However, mitragynine also interacts with other receptor systems, including adrenergic, serotonergic, and

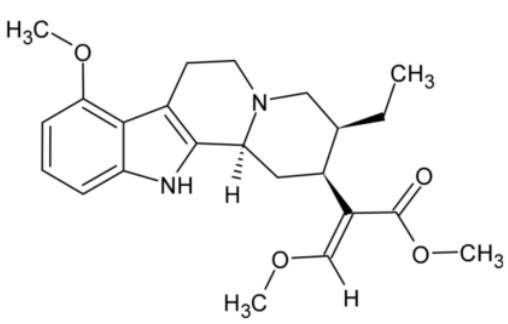
dopaminergic pathways, which can contribute to both its therapeutic effects and potential adverse side effects (Prozialeck et al. 2012). Mitragynine is the most significant kratom alkaloid with respect to pharmacological and toxicological evaluations, largely due to its abundance in the plants.

7-Hydroxymitragynine (7-HMG)

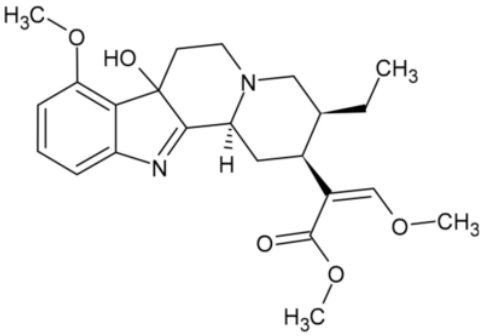
101. Though present in much smaller amounts (up to 2% of total plant alkaloid content), 7-hydroxymitragynine (7-HMG) is significantly more potent than mitragynine with respect to pharmacological and toxicological potential (Sharma and McCurdy 2021). Specifically, 7-HMG is about 13 to 46 times more potent than mitragynine, primarily because of the addition of a hydroxyl group at the C7 position on the indole ring (Prozialeck et al. 2012). This modification increases its binding affinity and efficacy at μORs, which are primarily responsible for kratom's analgesic (pain-relieving, or anti-nociceptive) effects (Obeng et al. 2020). The presence of this hydroxyl group facilitates better interactions with the receptor, enhancing kratom's opioid receptor agonist properties (Obeng et al. 2020; Obeng et al. 2021).

102. Biologically, 7-HMG induces pain relief responses in a dose-dependent manner, acting predominantly as a full agonist at μORs, which differentiates its effects from those of mitragynine, which acts as only a partial agonist (Obeng et al. 2021). Despite its relatively low concentrations in kratom products, 7-HMG's pharmacological profile suggests significant implications for pain management, while also raising concerns regarding its abuse potential due to the potent and efficacious opioid receptor activation (Kruegel et al. 2019). Further, as a metabolite of mitragynine, the effects of 7-HMG are modulated not only by direct intake from kratom products (which is quite low), but also formation from mitragynine within the body (Kruegel et al. 2019). The structures of mitragynine and 7-HMG are shown in Figure 2:

Figure 2. Chemical structure of mitragynine and 7-hydroxymitragynine (7-HMG).



Mitragynine

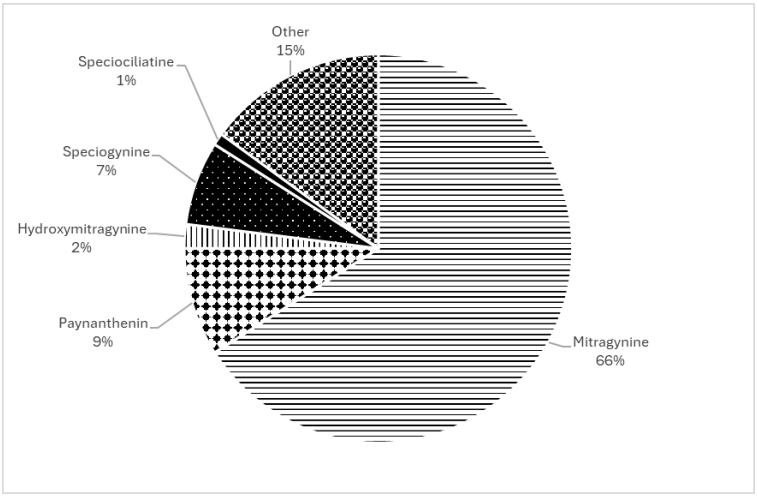


7-Hydroxymitragynine

Other Stereoisomers and Alkaloids

103. It was previously believed that kratom leaves contained over 25 different alkaloids, which contribute to their complex pharmacological profile (Tanguay and International Drug Policy Consortium 2011; Hassan et al. 2013). However, recent publications have identified more than 50 alkaloids, and the concentrations of each alkaloid vary depending on numerous factors including the region in which plants were grown, timing of harvest, and plant age (Leon et al. 2009; Adkins et al. 2011; Cinosi et al. 2015). The total alkaloid content in kratom leaves generally ranges from 0.5% to 1.5%, indicating a highly concentrated presence of these active compounds even in small amounts of leaf material (Hassan et al. 2013).

Figure 3. Predominant indole alkaloids present in kratom leaves. Redrawn from Cinosi et al. (2015).



104. As seen above in Figure 3, the primary indole alkaloids present in the young leaves of M. speciosa are mitragynine and its stereoisomer analogues, speciogynine, paynantheine and speciociliatine (Hassan et al. 2013). Each of these stereoisomers has the same empirical chemical formula as mitragynine ($C_{23}H_{30}N_2O_4$), with the difference between them being structural orientation.

105. As described above, the most significant alkaloids in terms of biological activity are mitragynine and 7-hydroxymitragynine (7-HMG), which make up approximately 60% and up to 2% of the total alkaloid content of kratom, respectively (Philipp et al. 2010; Kapp et al. 2011; Prozialeck et al. 2012). Paynantheine accounts for about 9% of the total alkaloids, playing a significant role in the muscle relaxant properties of the plant (Hassan et al. 2013). Speciociliatine and speciogynine comprise approximately 9% and 7% of the total, respectively, adding to the pharmacological diversity and effects of kratom (Hassan et al. 2013; Warner et al. 2016).

106. Other alkaloids like mitraphylline, rhynchophylline, mitralactonal, raubasine, and mitragynaline each make up less than 1% of the total alkaloid content, suggesting specialized or lesser-understood roles in kratom's overall physiological effect profile (Warner et al. 2016). Pharmacological and toxicologic data for many of the other remaining alkaloids are limited (Ellis et al. 2020). Table 1 presents the concentrations of select *M. speciosa* alkaloids in kratom leaves. Although mitragynine and 7-hydroxymitragynine are the most studied alkaloids of kratom, there are several other alkaloids that contribute significantly to its effects and potential therapeutic applications.

Table 1. Concentrations of select alkaloids in the leaves of M. speciosa.

Alkaloid Kratom (Prozialeck et al. 2012)	**Description and Effects**	**Percentage in young leaves (Takayama 2004)**	**Kratom Leaves Extract (Warner et al. 2016)**	**Alkaloid profile of Mitragyna speciosa Korth (Hassan et al. 2013)**	**Reference**

Mitragynine	Primary active alkaloid, known for effects including pain relief and mood enhancement. Acts as a partial agonist at the mu-opioid receptors.	66.2%	60% (Philipp et al. 2010; Kapp et al. 2011; Prozialeck et al. 2012)	66%	Takayama et al. (2002); Prozialeck et al. (2012); Kruegel et al. (2016); Warner et al. (2016); Sharma et al. (2019); Chear et al. (2021); Hanapi et al. (2021)
7-Hydroxy-mitragynine	Significantly potent, acts as a full agonist at mu-opioid receptors, primarily responsible for analgesic properties. Much more potent than mitragynine.	2.0%	2% (Prozialeck 2012; Philipp 2010; Kapp 2011)	2%	Prozialeck et al. (2012); Warner et al. (2016); Hanapi et al. (2021)
Speciociliatine	A weak opioid antagonist, structurally similar to mitragynine, which may help mitigate some of the addictive potential associated with stronger opioid agonists in kratom.	0.8%	9%	1%	Prozialeck et al. (2012); Hassan et al. (2013); Hanapi et al. (2021)
Speciogynine	Acts as a smooth muscle relaxer, contributing to the plant's use in alleviating cramps and gastrointestinal discomfort.	6.6%	7%	7%	Prozialeck et al. (2012); Hassan et al. (2013); Hanapi et al. (2021)

Paynantheine	The second most abundant alkaloid in kratom, known for its muscle relaxant properties.	8.6%	10%	9%	Prozialeck et al. (2012); Hassan et al. (2013); Hanapi et al. (2021)
Mitraphylline	Vasodilator, antihypertensive, muscle relaxer, diuretic, antiamnesic, immunostimulant, anti-leukemic	-	-	<1%	Hassan et al. (2013)
Ajmalicine (Raubasine)	Acts as a cerebrocirculant and antiaggregant, enhancing cerebral blood flow and preventing blood clot formation. Also an α-1 adrenergic receptor antagonist.	-	<1%	<1%	Prozialeck et al. (2012); Hassan et al. (2013); Hanapi et al. (2021)
Corynan-theidine	An opioid antagonist similar to yohimbine, which may reduce the psychoactive effects of stronger agonists in kratom and reduce dependency risks.	-	-	<1%	Takayama (2004); Hassan et al. (2013); Hanapi et al. (2021)
Corynoxine A and B	Dopamine mediators that potentially aid in managing dopamine-related disorders such as Parkinson's	-	-	<1%	Hassan et al. (2013); Hanapi et al. (2021)

	disease and mood disorders.				
Rhynchophylline	A vasodilator that also acts as an anti-inflammatory and antihypertensive, and inhibits platelet aggregation.	-	<1%	<1%	Hassan et al. (2013); Hanapi et al. (2021)

Speciociliatine

107. Speciociliatine is a weak opioid receptor antagonist, bearing a structural similarity to mitragynine (Prozialeck et al. 2012). As a weak antagonist, it potentially mitigates some of the effects of mitragynine and 7-hydroxymitragynine, thereby possibly reducing the risk of dependence and abuse associated with kratom (Prozialeck et al. 2012). According to Kamble et al. (2021), speciociliatine more potently stimulates μOR-mediated G protein-coupled signaling *in vitro* than mitragynine.

108. Some *in vivo* studies have demonstrated that speciociliatine can act as an opioid agonist. For example, it has been shown to produce antinociceptive effects in animal models, which are indicative of opioid-like activity (Hanapi et al. 2021). In contrast, certain *in vitro* studies reported that speciociliatine exhibited weak antagonistic activity at opioid receptors; some cellular models determined that speciociliatine could block or dampen the receptor activity typically stimulated by other potent agonists like mitragynine and 7-hydroxymitragynine (Takayama et al. 2002; Kruegel et al. 2016). This suggests that the stereochemistry at the C-3 position of the speciociliatine molecule could be crucial in enhancing its binding affinity and subsequent pharmacological response (Karunakaran et al. 2022).

Speciogynine

109. Known for its role as a smooth muscle relaxer, speciogynine is an important alkaloid in terms of the therapeutic applications of kratom (Prozialeck et al. 2012). By contributing

muscle relaxant properties, it can enhance the therapeutic potential of kratom for users seeking relief from physical discomfort without psychoactive effects (Hanapi et al. 2021).

Paynantheine

110. Paynantheine is considered the second most abundant alkaloid in some kratom extracts, accounting for about 10% of the total alkaloid content (World Health Organization (WHO) 2021). While paynantheine is known for its muscle relaxant properties, it does not primarily act through opioid receptor pathways like some of the other kratom alkaloids. Instead, its mode of action is more associated with mechanisms similar to those of other muscle relaxants, which may involve modulation of calcium influx or influence on other non-opioid receptor pathways (Takayama 2004).

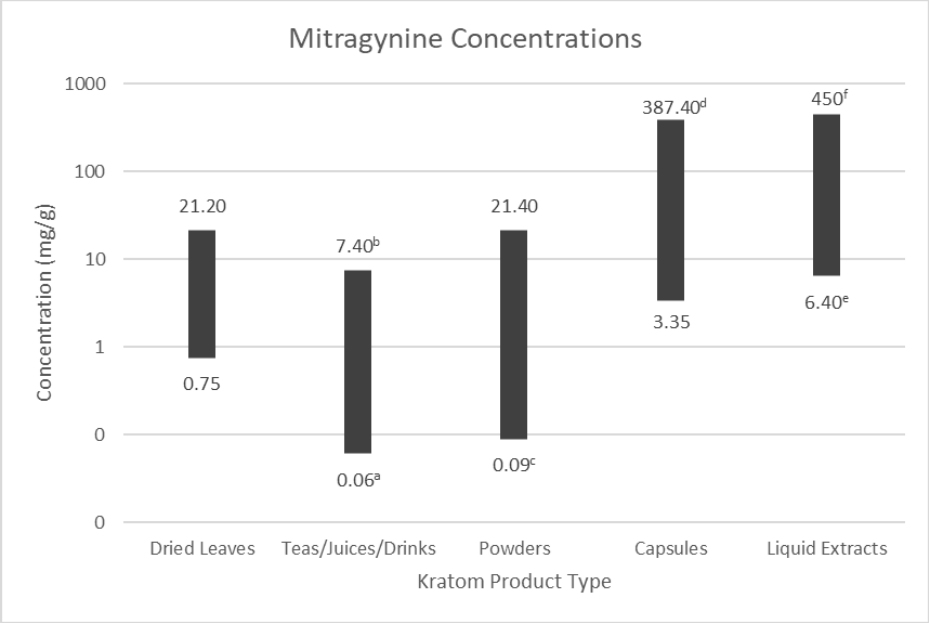
Corynantheidine

111. Corynantheidine is an opioid antagonist at μORs, essentially blocking the receptor and preventing the typical opioid effects such as pain relief, which would otherwise be mediated by agonists like 7-hydroxymitragynine or morphine. In kratom, corynantheidine's opioid antagonistic properties may serve to balance the effects of opioid agonists, potentially reducing the addictive qualities and other negative side effects associated with opioid-like compounds (Hanapi et al. 2021).

Alkaloid Content of Kratom Products

112. Kratom is supplied in various forms, including as leaves, powders, capsules, extracts, teas, gummies, and resins. Vape shops and online vendors often provide a range of kratom products, catering to both novice and experienced kratom users. The availability of such a wide array of kratom products online and in vape shops highlights the substance's growing popularity as both a recreational substance and a self-medication method (Prozialeck et al. 2012; Henningfield et al. 2018).

Figure 4. Range of mitragynine concentrations, standardized to mg/g, across kratom product types based on data from several authors (Kikura-Hanajiri et al. 2009; Singh et al. 2018c; Fowble and Musah 2019; Sharma et al. 2019; Prozialeck et al. 2020; Todd et al. 2020; Fleming et al. 2023). Redrawn from Heywood et al. (2024)



It was assumed that the density of the liquids were approximately the same as water, or 1 g = 1 ml.

[a]21.8 mg / 30 ml = 0.06 mg/g.

[b]0.74% / 100 = 0.0074; 0.0074 x 1,000 mg/g = 7.40 mg/g.

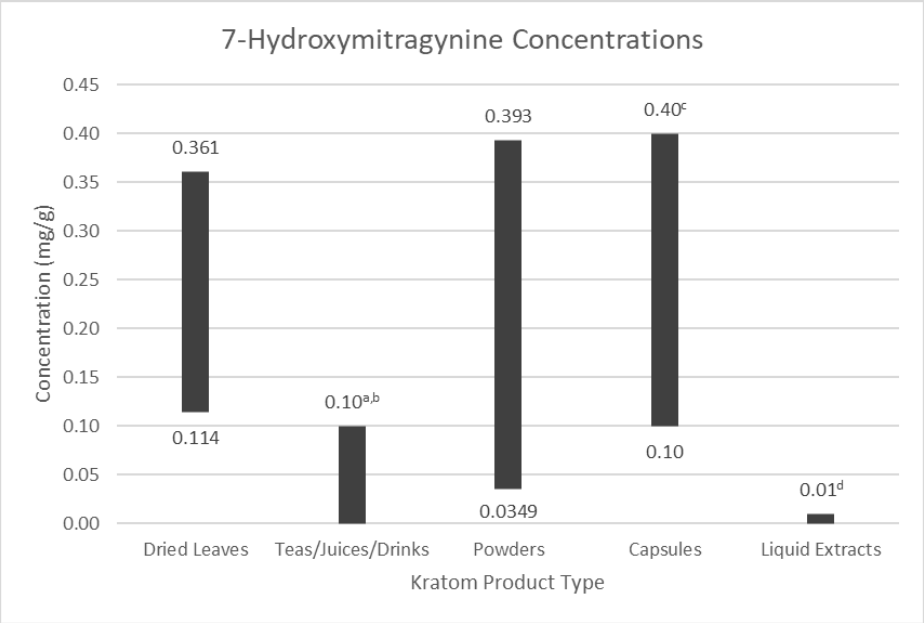
[c]1.6% / 100 = 0.016; 0.016 x 1.1 g = 0.0176 g; 0.0176 g x 1,000 mg/g = 17.6mg; 17.6mg / 200 g = 0.09 mg/g.

[d]38.74% / 100 = 0.3874; 0.3874 x 1,000 mg/g = 387.40 mg/g.

[e]32 mg / 5 ml = 6.40 mg/g.

[f]45% / 100 = 0.45; 0.45 x 15 ml = 6.75 ml; 6.75 mL x 1,000 mg/g = 6,750 mg; 6,750 mg / 15 mL = 450 mg/g.

Figure 5. Range of 7-hydroxymitragynine concentrations, standardized to mg/g, across kratom product types based on date from several authors (Kikura-Hanajiri et al. 2009; Sharma et al. 2019; Todd et al. 2020; Fleming et al. 2023). Redrawn from Heywood et al. (2024).



It was assumed that the density of the liquids were approximately the same as water, or 1 g = 1 ml.

[a]Minimum was not reported and thus set to 0 mg/g.

[b]0.01% / 100 = 0.0001; 0.0001 x 1,000 mg/g = 0.10 mg/g.

[c]0.04% / 100 = 0.0004; 0.0004 x 1,000 mg/g = 0.40 mg/g.

[d]Liquid extracts – sample value was reported as <0.01 mg/g.

Leaves

113. Traditionally, fresh or dried kratom leaves from the *M. speciosa* are chewed or made into tea; they are seldom smoked (European Monitoring Centre for Drugs and Drug Addiction 2015). Kratom leaves are distinguished by the color of the veins running through them, which can be white, green, or red. This color variation is commonly used in the marketing and classification of kratom in Western countries, where the leaves are primarily sold as powdered extracts.

114. Each vein color is often associated with different effects, which are attributed to variations in the chemical composition and concentration of alkaloids within the leaves (Veltri and Grundmann 2019). Chewing the fresh leaves of kratom releases alkaloids such as mitragynine into the bloodstream more directly. This practice is common among laborers in countries like Thailand and Malaysia, where kratom is used to reduce muscle pain and enhance productivity during long hours of physical labor (Veltri and Grundmann 2019).

115. The alkaloid content in kratom leaves, particularly mitragynine and 7-hydroxymitragynine, varies significantly depending on factors like the plant's geographic origin and the methods used in its processing. Dry kratom leaves typically contain about 0.5% to 1.5% alkaloids by weight (Hassan et al. 2013). Mitragynine is the predominant alkaloid, comprising over 60% of the total alkaloid content, while 7-hydroxymitragynine accounts for about 2% (Lydecker et al. 2016; Kruegel and Grundmann 2018). Other minor alkaloids such as speciociliatine, speciogynine, and paynantheine contribute 9%, 7%, and 10%, respectively, to the total alkaloid profile (Takayama 2004; Kruegel and Grundmann 2018). The concentration of these alkaloids can vary widely in commercially available kratom products in the U.S., ranging from 0.7% to 38.74% for mitragynine and 0.01% to 0.75% for 7-hydroxymitragynine in products like capsules, dried leaves, powders, resins, and concentrated extracts (Kikura-Hanajiri et al. 2009; Sharma et al. 2019).

116. Kratom grown in the United States has been identified as having a distinct chemotype, richer in oxindole alkaloids compared to its counterparts in Asia and Africa (Raffa 2014). In young Thai kratom plants, speciogynine is the dominant alkaloid found in the leaves, whereas in the fruits of Malaysian kratom plants, speciociliatine is more prevalent (Kitajima et al. 2005; Raffa 2014).

Wet Leaves

117. Fresh, wet kratom leaves contain all the plant's natural alkaloids in their original concentrations. When kratom is freshly harvested, the alkaloids are present in their most natural state (Zhang et al. 2020). However, the total alkaloid content in fresh leaves is generally lower by

weight compared to dry leaves, primarily because fresh leaves contain a significant amount of water. When kratom leaves are fresh, the alkaloids are naturally protected within the plant's cell structure. Chewing fresh leaves can release these alkaloids effectively, though the bioavailability might be different compared to dried and powdered forms where cell walls have been broken down, potentially making alkaloids more readily available upon ingestion (Hassan et al. 2013; Zhang et al. 2022).

Dry Leaves

118. Drying kratom leaves is a common method for preserving them and concentrating their alkaloids. As the water content in the leaves evaporates, the alkaloids become more concentrated by weight. Therefore, dry leaves typically have a higher alkaloid content than fresh leaves (Zhang et al. 2022). Drying methods can affect the alkaloid profile; for example, drying leaves in the sun can degrade certain alkaloids, while shade drying might preserve them better (Zhang et al. 2022). A study by Zhang et al. (2022) quantified nine different kratom alkaloids and found that mitragynine, paynantheine, and corynoxine concentrations were significantly higher in leaves from shaded environments. This indicated that lower light intensities may enhance the synthesis of certain key alkaloids, which are important for kratom's psychoactive and medicinal properties (Zhang et al. 2022).

119. Other studies have shown that alkaloids such as paynantheine, speciogynine, mitraphylline, speciociliatine, and corynantheidine had concentrations ranging from 0.1% to 12.8% by weight in dry leaf powder or extracts (Hassan et al. 2013; Cinosi et al. 2015; Montserrat-de la Paz et al. 2015; Obeng et al. 2020). The stability of certain alkaloids during drying can vary, with some being more susceptible to degradation due to environmental factors such as light, temperature, and humidity. The dried leaves can then be used in various forms, such as powder, which is commonly used for making tea or capsules.

Seasonal and Geographic Differences in Alkaloid Concentrations

120. *M. speciosa* from Thailand has higher mitragynine content than *M. speciosa* from Malaysia (Takayama et al. 1998). Specifically, looking at Thailand, a study by Leksungnoen et al.

(2022) investigated variations in mitragynine content in kratom across different geographic locations, examining how environmental factors impacted alkaloid levels. Factors such as light intensity, relative humidity, soil moisture content, soil pH, and calcium levels in the soil impacted the variations in mitragynine (Leksungnoen et al. 2022). Four regions in Thailand, north, northeast, central, and south, were also investigated and found to influence the metabolic profile of the plant; mitragynine content ranged from 7.5 to 26.6 mg per gram of dry leaf weight across all regions, with an average of 16.0 ± 5.7 mg per gram (Leksungnoen et al. 2022).

121. The highest mitragynine concentrations of 18.5 to 21.8 mg per gram of dry leaf weight were generally found in samples from the central and southern regions of Thailand (Leksungnoen et al. 2022). It was reported that these areas experienced higher light intensity and relative humidity, including acidic soils with higher calcium content found to be conducive for higher mitragynine content. This was further qualified when the lowest concentrations (approximately 11.4 mg per gram of dry leaf weight) were found in the northern and northeastern regions of Thailand, which generally have lower light intensity and different soil characteristics (Leksungnoen et al. 2022).

122. Application of fertilizer significantly increased plant growth metrics such as height, width, trunk size, leaf number and area, and total dry mass (Zhang et al. 2020, 2022). This suggested that kratom responded well to nutrient supplementation, enhancing overall biomass without signs of nitrogen toxicity, even at high application rates. While fertilizer increased the overall biomass, it did not increase the concentration of mitragynine per unit of leaf dry mass (Zhang et al. 2022). This indicated that while the plants grow larger and produce more leaves, the amount of mitragynine per leaf remained constant. Zhang et al. (2020) also noted a substantial increase in the harvestable mass of other alkaloids such as speciogynine, paynantheine, speciociliatine, mitraphylline, corynantheidine, isocorynantheidine, and corynoxine, with the highest fertilizer rates resulting in a 9- to 12-fold increase (Zhang et al. 2020). This suggested enhanced secondary metabolism under higher nutrient availability.

Teas

123. Brewed tea is a traditional and still commonly used method of kratom consumption in the United States and elsewhere. Kratom powder, kratom extract, or the dried leaves (loose or in a tea bag) are steeped in hot water, which extracts the alkaloids into the liquid. Per anecdotal instructions, lemon or other citrus may be added to help extract the alkaloids more efficiently and improve the taste (Kats Botanicals 2024a). The potency of teas, which depends on the efficacy of alkaloid extraction, is variable based on the water temperature, the amount of kratom used, and the steeping time. Additionally, the concentration of mitragynine in kratom teas can vary based on the preparation method, the form of kratom used (such as raw leaf or extract), and the specific batch. One study reported that oral administration of lyophilized kratom tea resulted in 1.5 to 1.8 times higher bioavailability of mitragynine compared to mitragynine administered alone, suggesting that traditional kratom tea preparations may enhance the systemic exposure of mitragynine (Avery et al. 2019).

124. Another study developed a method to simultaneously quantify several kratom alkaloids, including mitragynine, in various forms of kratom products, including teas, noting that the concentration range could be quite broad across different commercial products (Sharma et al. 2019). The results of the analysis indicated that the lyophilized tea contained 0.7% mitragynine, 0.35% speciociliatine, and 0.25%, paynantheine (Sharma et al. 2019)

Juices

125. Another way to consume kratom as a liquid is via mixture with fruit juices. This method is anecdotally reported to decrease the bitterness of kratom but to also potentially increase its efficacy due to the antioxidants present in grapefruit and fruit juices (Kats Botanicals 2024c). The user can choose to mix kratom with orange, mango, pineapple, lemonade, hot chocolate, and coffee. Leong Bin Abdullah and Singh (2021b) conducted a study in Malaysia where mean serum mitragynine levels of 10.3 mg/l (range from 2.5 mg/l to 22.4 mg/l) were reported among regular users of brewed kratom juice. The authors concluded that, "[r]egular kratom users who consumed an average daily quantity of four or more glasses of kratom juice (freshly brewed kratom juice)

registered a significantly higher serum mitragynine level compared with those who consumed less than four glasses of kratom juice" (Leong Bin Abdullah and Singh 2021b).

Powders

126. Kratom powder is made by drying kratom leaves and grinding them into a fine powder; it can be used in teas, mixed with beverages, or encapsulated. According to a study that described the prevalence and variety of kratom products available in the online marketplace, kratom powder is the most common form of kratom sold online, with 92% of vendors offering it (Williams and Nikitin 2020).

127. A study examining the alkaloid content of six commercially available dried and powdered, non-concentrated kratom products found mitragynine concentrations ranged from 4.71% to 8.72% and 7-hydroxymitragynine concentrations ranged from 0.01 to 0.04% (Sharma et al. 2019).

Capsules

128. Kratom capsules consist of powdered leaves enclosed in a gelatin or vegetable-based capsule. Capsules typically come in several doses or sized, ranging from 300 mg to 1 g of total weight (not alkaloid content) per capsule (Kats Botanicals 2024b). The subsequent dose depends on various factors including the user's tolerance, the desired effects, and the specific strain of kratom used. As reported b Veltri and Grundmann (2019), it is essential for users to start with a lower capsule doses to gauge tolerance before gradually increasing to avoid potential side effects. The alkaloid content in the capsules would directly reflect that of the powder used, often about 0.5% to 1.5% mitragynine by dry weight of the leaf material (Hassan et al. 2013).

129. Prozialeck et al. (2020) evaluated the mitragynine content in processed kratom leaf material and concentrated kratom extracts purchased from various shops in the western suburbs of Chicago. The mitragynine levels ranged from approximately 3.35 mg/g to 11.33 mg/g of kratom product in finely ground leaf material. However, the capsules showed a much higher mitragynine content due to being derived from extracts (Prozialeck et al. 2020).

Liquid Extracts

130. Liquid extracts are potent forms of kratom made by extracting the alkaloids from the leaves using a solvent like ethanol or water to pull out the alkaloids from plant material. The mixture is then reduced down to a potent liquid that contains a higher concentration of these active compounds. Due to their potency, liquid extracts are generally used in smaller doses than other forms of kratom. Users often add them to beverages or take them directly using a dropper. The effects are typically faster and more intense due to the higher concentration of alkaloids (Kruegel and Grundmann 2018).

Manufacturer Data

131. According to one manufacturer's website, kratom extracts contain 200 mg mitragynine in each 15 ml bottle and each bottle contains six servings, which is about 33 mg of mitragynine per serving (CBD Kratom Cannabis Dispensary 2024). Other websites advertise liquid extracts that contain 60 mg of mitragynine per serving in products known as "Kratom Shots" (Super Speciosa Raw Leaf 2024b). Another manufacturer website detailed that their "MIT45 GO," which was a gel pouch, contained 150 mg of extract with a 65% mitragynine concentration, which they claimed was 97.5 mg of pure mitragynine. Another product they sell known as "MIT45 shot," which was a liquid, contained 250 mg of extract with a 45% mitragynine concentration, which they claimed was 112.5 mg of mitragynine (Croxford 2024). Both of the above listed products were reported to have three servings in each shot product.

132. Some liquid extracts come in mixtures with kava that contain 35 mg of kratom extract (not specific to alkaloid content) and 25 mg of kava extract per serving (Super Speciosa Raw Leaf 2024c).

Gummies

133. Kratom gummies are made by incorporating kratom extract into a gelatinous base along with flavorings and sweeteners. They provide a fixed dose per gummy, which can range from 2.33 mg to 50 mg of mitragynine (Moore 2024a). The most common alkaloids in kratom, mitragynine and 7-hydroxymitragynine, are present in these gummies, however, the process used to create the kratom extract infused in gummies may alter the alkaloid profile, potentially

enhancing or reducing the efficacy of minor alkaloids (Prozialeck et al. 2020). The bitter taste of kratom is a common complaint among users, and gummies are reported to offer a flavorful alternative that makes ingestion more pleasant (Veltri and Grundmann 2019)

Manufacturer Data

134. According to manufacturer websites, Kratom gummies contain approximately 30 mg of mitragynine per serving (Kraken Kratom 2024). As an example, for the Blueberry Extra Strength Kratom Gummies from Super Speciosa, "[e]ach gummy packs a powerful punch, with approximately 35 mg of mitragynine—equivalent to 2.33 grams of kratom powder at a 1.5% mitragynine level" (Super Speciosa Raw Leaf 2024a).

Tinctures

135. Similar to liquid extracts, tinctures are alcoholic or glycerin-based solutions that have extracted the alkaloids over weeks, but tinctures are said to be more potent (Korthals' Collection 2024). There are two types of kratom tinctures: full spectrum kratom tinctures and isolated kratom tinctures (Korthals' Collection 2024). Full spectrum kratom tinctures contain all the alkaloids present in the original plants and isolated kratom tinctures contain specific alkaloids (Korthals' Collection 2024). They are used sublingually (under the tongue) for quick absorption.

Resins

136. Kratom resin is a concentrated form of the plant created by boiling kratom leaves or powder to extract the alkaloids and then solidifying the extract into a dense, sticky substance (Kraken Kratom 2021). Resins are much more potent than regular dried leaves or powder. The concentration of alkaloids in kratom resin can be several times higher than in dried leaf equivalents, depending on the concentration process.

Background on Pharmacokinetics and Pharmacodynamics

137. Pharmacokinetic studies describe the way mammals process, that is, absorb, metabolize, distribute, and eliminate compounds to which they are exposed. Pharmacodynamic and toxicodynamic studies refer to the study of the biochemical and physiological effects of drugs on the body, as well as the mechanisms by which these effects are produced. Such studies indicate

that kratom's physiological effects occur within minutes of product use and generally peak within one to four hours of use, depending on dose. It is my opinion that based on the elimination half-life data for mitragynine and 7-hydroxymitragynine in humans, kratom associated alkaloids are generally eliminated (or greater than 95% eliminated) from the human body within two to 24 hours of product use, depending on dose and use frequency.

138. Pharmacokinetic studies describe the ways that organisms biologically process compounds to which they are exposed intentionally, such as via drug administration, or unintentionally, such as via secondhand or environmental exposures. These are often termed studies of the time course of absorption, distribution, metabolism, and elimination (ADME). In contrast, pharmacodynamic studies describe the physiological effects of a compound on the body.

139. As noted previously, kratom is consumed in various forms such as chewing leaves, drinking teas and juices, via capsules, powdered in iced cocktails, or via smoking, though the latter is uncommon due to the high volume of leaves needed and their high cellulose content, which makes smoking less effective (Tanguay and International Drug Policy Consortium 2011; Hassan et al. 2013; Brown et al. 2017; Henningfield et al. 2018; Veltri and Grundmann 2019; Braley and Hondrogiannis 2020; Eastlack et al. 2020). The potency of kratom varies with the strain and age of the plant; older, green-veined leaves are typically more potent than younger, red-veined ones, and users should consider these differences when selecting products (Brown et al. 2017; Veltri and Grundmann 2019; Braley and Hondrogiannis 2020; Swogger et al. 2022).

Pharmacokinetics

140. Following consumption, mitragynine is absorbed rapidly due to its high lipid solubility and ability to cross the blood-brain barrier. The elimination of mitragynine from the body follows a biphasic pattern, initially distributing quickly then diminishing more slowly indicates that while the plant's physiological effects may start quickly, they could also last several hours, depending on the dose. The primary metabolic processing occurs in the liver via cytochrome P450 (CYP) enzymes, producing various metabolites that may influence both its therapeutic

effects and potential adverse effects. In contrast, 7-hydroxymitragynine, though less abundant in kratom, is more potent and metabolized similarly to mitragynine.

Pharmacodynamics

141. Mitragynine's interaction with opioid receptors, particularly μORs and δORs where it acts as a partial agonist, underlies the analgesic and mildly euphoric effects observed at lower doses. Though found in smaller amounts, 7-hydroxymitragynine is a stronger μOR agonist, enhancing its pain-relieving efficacy. Beyond ORs, these alkaloids also modulate other neurotransmitter systems such as the adrenergic, serotonergic, and dopaminergic pathways, affecting mood and pain perception. Their ability to engage these receptors without the full receptor activation seen with traditional opioids suggests they might offer pain relief with potentially lower risks of dependency and fewer side effects (such as a reduced risk of respiratory depression).

142. The differing effects and potencies across kratom alkaloids render such products multifaceted pharmacological substances with stimulant or sedative outcomes based on dose and the proportion of mitragynine to 7-hydroxymitragynine. The pharmacokinetic and pharmacodynamic properties provide a foundation for understanding how mitragynine and 7-hydroxymitragynine might be used therapeutically, the risks associated with their use, and the strategies for mitigating these risks. This information is essential for healthcare providers, researchers, and policymakers in determining the safe and effective use of kratom and its constituents in clinical settings or regulating their use in consumer products.

Pharmacokinetics of Mitragynine and 7-Hydroxymitragynine

In Vitro Pharmacokinetics

Mitragynine

143. Ya et al. (2019) conducted a systematic review of studies related to the pharmacokinetics of mitragynine. The in vitro studies included investigated mitragynine solubility, permeability, plasma protein binding, and metabolism (Hanapi et al. 2013; Lim et al. 2013; Manda et al. 2014; Ramanathan et al. 2015; Kong et al. 2017a; Manda et al. 2017). Ya et al. (2019)

reviewed studies that evaluated mitragynine absorption in vitro and across the gut epithelium, both of which suggested mitragynine absorption was passive, rapid, and unaffected by inhibitors of P-glycoprotein. Additionally, a bioavailability of 21% was estimated, and it was reported that mitragynine was able to penetrate and redistribute in the brain (Ya et al. 2019).

Solubility

144. In simulated intestinal fluid (180 min, pH = 6.8), mitragynine had a solubility ranging from 10.79 ± 0.25 to 17.57 ± 2.01 µM (Manda et al. 2014; Ramanathan et al. 2015; Ya et al. 2019). In simulated gastric fluid (pH = 1.2), it was reported that mitragynine has a solubility of 8.03 ± 0.5 µM during 120 min of incubation and of 35.13 ± 0.5 µM during 60 min of incubation. Mitragynine solubility in water at pHs of 4 and 7 was determined to be 130.0 ± 0.1 and 82.7 ± 1.9 µM, respectively (Ya et al. 2019).

Permeability

145. It was reported that mitragynine absorptive flux through a parallel artificial membrane permeability assay was 0.23 ± 0.0005 (10-6 cm/sec) and the secretory flux was 11.14 ± 0.06 (10-6 cm/sec) (Kong et al. 2017a; Ya et al. 2019).

146. Through a brain endothelioma MDR-MDCK (multidrug resistance protein in Madin-Darby Canine Kidney) cells monolayer, the absorptive flux was 15.3 ± 1.7 (10^{-6} cm/sec) at 5 µM and 16.2 ± 1.8 (10^{-6} cm/sec) at 10µM (Manda et al. 2014). The secretory flux in MDR-MDCK monolayers was 17.2 ± 1.6 (10-6 cm/sec) for 5 µM mitragynine and 18.1 ± 1.7 (10^{-6} cm/sec) for 10 µM (Manda et al. 2014). Through gut wall Caco-2 cell (human colonic adenocarcinoma cells) monolayers, the absorptive flux was 24.2 ± 2.6 (10^{-6} cm/sec) at 5 µM and 25.3 ± 2.2 (10^{-6} cm/sec) at 10 µM (Manda et al. 2014). Jagabalan et al. (2019) measured mitragynine permeability to the intact intestinal wall. Mitragynine absorptive permeability through intestine in situ was 1.11×10^{-4} cm/s.

Protein Binding and Metabolism

147. It was reported that 85–95% of mitragynine is bound to plasma protein and that most of its metabolism, which involves phase I and phase II, occurs in the liver (Manda et al. 2014;

Kong et al. 2017a; Ya et al. 2019). Several studies have determined that mitragynine enhanced the expression of metabolic enzymes including CYP1A2, CYP3A4, CYP2D6, and CYP2C9 (Hanapi et al. 2013; Lim et al. 2013; Manda et al. 2017).

148. Further, in a study by Kamble et al. (2019) the metabolism of mitragynine was predominantly carried out via CYP3A4 with minor contributions by CYP2D6 and CYP2C9. The formation of five oxidative metabolites (Met2, Met4, Met6, Met8, and Met11) was catalyzed by the CYP3A4. The authors found a total of thirteen metabolites were identified, with four oxidative metabolites and a metabolite formed by demethylation at the 9-methoxy group being the major metabolites of mitragynine. Mitragynine was found to be extensively metabolized in human liver microsomes primarily to O-demethylated and mono-oxidative metabolites. The CYP3A4 enzyme plays a predominant role in the metabolic clearance of mitragynine and also in the formation of 7-hydroxymitragynine (7-HMG), a known active minor alkaloid identified in the leaf material (Kamble et al. 2019).

7-Hydroxymitragynine (7-HMG)

149. The *in vitro* pharmacokinetics of 7-hydroxymitragynine were well documented in the study by Manda et al. (2014). The authors evaluated the *in vitro* absorption, distribution, metabolism, and excretion (ADME) properties of three kratom alkaloids: mitragynine, 7-hydroxymitragynine, and mitraphylline. The study examined the stability and absorption of 7-hydroxymitragynine, which underwent a degradation of about 27% in simulated gastric fluid, suggesting a partial conversion to mitragynine (a 23% conversion was reported). However, it was more stable in simulated intestinal fluid, with only 6% degradation reported. Additionally, it was reported that 7-hydroxymitragynine exhibited moderate permeability across Caco-2 and MDR-MDCK cell line monolayers, with no significant efflux. This is discussed in more detail later in my report and indicated effective intestinal absorption.

150. Regarding distribution, 7-hydroxymitragynine exhibited high plasma protein binding (>90%), which indicated a higher likelihood of remaining in the bloodstream and potentially prolonged systemic circulation. It was reported that 7-hydroxymitragynine was

metabolized by human liver microsomes, and had a relatively rapid metabolism with a biological half-life of approximately 24 minutes. The authors did not specify the excretion patterns of 7-hydroxymitragynine. However, given its high plasma protein binding and metabolism by liver microsomes it is likely excreted primarily in the form of metabolites. Further, the authors indicated that 7-hydroxymitragynine was shown to inhibit P-glycoprotein with an EC_{50} of 32.4 ± 1.9 µM, which indicated potential interactions with other compounds that are substrates of P-glycoprotein, potentially affecting the absorption and elimination of these other compounds.

In Vivo Pharmacokinetics

Mitragynine

151. Kong et al. (2017b) and Avery et al. (2019) both investigated the pharmacokinetics of mitragynine in rats. Both studies administered a single intravenous dose of mitragynine (Kong: (10mg/kg) in 10% Tween 20 aqueous solute; Avery: (5mg/kg) and utilized ultra-fast liquid chromatography–mass spectrometry (UFLC-MS) method to quantify the mitragynine in the collected plasma). Kong et al. (2017b) reported that their analysis revealed mitragynine showed a two-compartmental drug elimination pattern and a half-life of approximately 13 hours. Avery et al. (2019) reported that mitragynine demonstrated a biexponential decrease in the concentration-time profile, suggesting fast distribution. Kong et al. (2017b) also assessed brain concentrations of mitragynine and reported the percent AUCbrain/AUCplasma of mitragynine was calculated as 65.8 ± 4.5%, demonstrating that the alkaloid permeates the blood-brain barrier (BBB). Additionally, Avery et al. (2019) compared the intravenous dose (5mg/kg) to an oral dose (20/mg/kg) of lyophilized kratom tea and organic fraction of lyophilized kratom tea at the same mitragynine dose. They reported that, compared to mitragynine dosed alone, the absolute oral bioavailability of mitragynine was 1.5 to 1.8-fold higher. It was reported that the clearance of mitragynine was slow, or approximately 2.26 ± 0.21 L/h/kg (plasma) or 3.78 ± 0.18 L/h/kg (brain) (Kong et al. 2017b).

152. Ramachandram et al. (2019) conducted a pharmacokinetic review of mitragynine. Of the three rat studies reviewed, it was reported that following oral administration (20-50 mg/kg

mitragynine), maximum plasma concentration (C_{max}) ranged from 0.42 to 0.70 µg/mL, time to reach C_{max} (T_{max}) ranged from 1.26 to 4.50 h, and elimination half-life ranged from 3.85 to 9.43 h−1. Though inconsistent, apparent total clearance ranged from 1.6 to 7.0 L/h/kg, and apparent volume distribution ranged from 37.90 to 89.5 L/kg. The authors noted that different routes of administration and extraction methods contributed to the variability in pharmacokinetic data.

153. Berthold et al. (2022) investigated the tissue distribution of mitragynine following oral administration (165 mg/kg) in mice. Noncompartmental analysis for brain and plasma mitragynine revealed the following distribution of mitragynine: liver, kidney, lung, spleen, and brain.

7-Hydroxymitragynine (7-HMG)

154. Maxwell et al. (2021) investigated 7-HMG pharmacokinetics in female beagle dogs. One mg/kg of 7-HMG was orally administered to three animals and plasma samples were obtained. The pharmacokinetic parameters were derived from non-compartmental analysis. The authors reported that 7-HMG absorption occurred "rapidly." The peak plasma concentration was 56.4 ± 1.6 ng/ml and occurred 15 minutes following oral dosing. The area under the plasma concentration-time curve (AUC) of 7-HMG was reported as 178.6 ± 25.9 (h•ng/ml), clearance (Cl/F) was 4.4 ± 0.6 (L/h/kg), and the volume of distribution (Vd/F) was 23.8 ± 6.7 L/kg. The mean elimination half-life for 7-HMG was 3.6 ± 0.5 h and the authors reported that elimination was slow relative to absorption.

155. Berthold et al. (2022) conducted a pharmacokinetic and pharmacodynamic study of mitragynine and 7-HMG at high oral doses in C57BL/6 mice to understand the extent to which 7-HMG metabolized from mitragynine accounts for the antinociceptive, or pain-relieving, effects of mitragynine. Male and female mice were given a single oral administration of 165 mg/kg mitragynine. They determined that, compared to mitragynine dosed alone, 7-HMG was 2.8-fold more potent to produce antinociception. It was reported that the distribution of 7-HMG following administration was similar to that of mitragynine (liver, kidney, spleen, lung, brain). They noted

that the organ-to-plasma exposure ratio in the lung was much greater for mitragynine (15.9) than for 7-HMG (1.5).

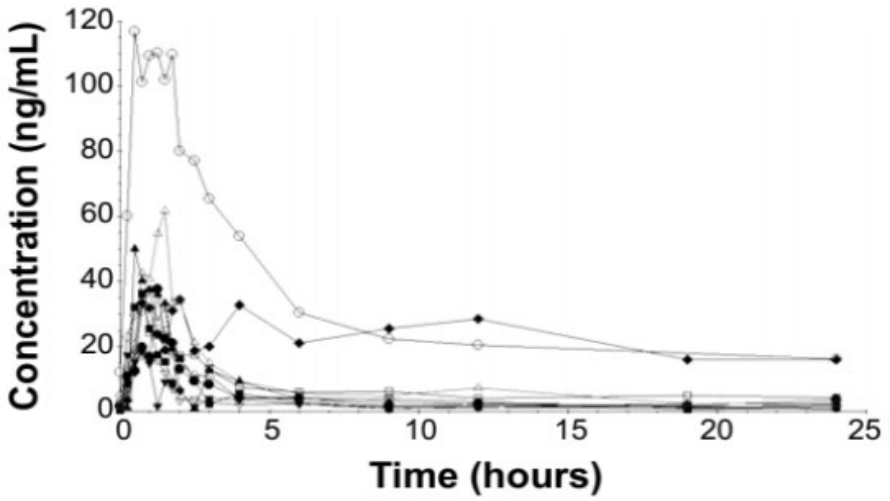
Human Pharmacokinetics

Trakulsrichai et al. (2015)

156. Trakulsrichai et al. (2015) conducted a prospective study of kratom users to evaluate the pharmacokinetics of mitragynine in humans and assess the linearity in pharmacokinetics in chronic users. Ten male chronic, regular, and healthy users were enrolled. Subjects ingested 60 mL of kratom tea every day for seven days prior to the start of the experiment. The tea was prepared with fresh Kratom leaves and with three concentrations of mitragynine: 0.1042, 0.166, and 0.1917 mg/mL. Following the seven-day pre-exposure period, each participant was randomly assigned to take 60 mL or 120 mL of tea. Blood was drawn from participants 17 times over the course of 24 hours, including at time zero (pre-exposure) and at 15, 30, and 45 minutes and 1, 1.25, 1.5, 1.75, 2, 2.5, 3, 4, 6, 9, 12, 19, and 24 hours. Urine samples were also collected.

157. The time to reach the maximum plasma concentration was 0.83 ± 0.35 h, the terminal half-life was 23.24 ± 16.07 h, the apparent volume of distribution was 38.04 ± 24.32 L/kg, and the urine excretion of unchanged form was 0.14%. The authors concluded that their results demonstrated linearity and were consistent with the oral two-compartment model with a terminal half-life of approximately one day. The plasma mitragynine concentration-time curve reported by Trakulsrichai et al. (2015) included below as Figure 6.

Figure 6. Plasma mitragynine concentration-time curve in human subjects following oral intake of 60 mL of kratom tea with varying (0.1042, 0.166, or 0.1917 mg/mL) of mitragynine. Redrawn from Trakulsrichai et al. (2015).



Tanna-et-al. (2022)

158. To assess pharmacokinetics in humans, Tanna et al. (2022) administered 2 g of a kratom product (as a tea) to three male and three female, healthy participants. The participants' blood was drawn prior to tea administration; at 0.25, 0.5, 0.75, 1.25, 1.75, 2.25, 3.25, 4.25, 6.25, 8.25, and 12.25 h post tea administration (collected via venous catheter); and at 24, 48, 72, 96, and 120 h post tea administration (via blood draw). Urine samples were collected twice in 12h intervals and then in 24h intervals for four days. The concentration-time profile for mitragynine was evaluated with a compartmental model and pharmacokinetics were assessed using noncompartmental analysis.

159. The authors reported that the two-compartment model best described the data. The compartmental model-derived pharmacokinetic parameters of mitragynine are as follows: maximum plasma concentration (C_{max}) = 65.6 nM, time to reach C_{max} (t_{max}) = 1.13 h, area under the plasma concentration-time profile (AUC) = 431 nM x h, alpha half-life ($t_{1/2}$) = 1.76 h, terminal (beta) half-life ($t_{1/2}$) = 37.3 h, apparent central volume of distribution = 1,170 L, and apparent peripheral volume of distribution = 5,620 L. Additionally, the authors discussed significant pharmacokinetic differences between kratom alkaloids with a 3S stereochemistry configuration (mitragynine, speciogynine, paynantheine) and alkaloids with the 3R stereochemistry

configuration (mitraciliatine, speciociliatine, isopaynantheine), which they stated were due to differences in apparent intercompartmental distribution clearance, volumes distribution, and clearance.

160. Tanna et al. (2022) reported that 7-hydroxymitragynine, specifically, was quantifiable in plasma for up to 24 hours in participants following dosing, which indicated a relatively short detection window compared to other alkaloids. They noted that the compound displayed biphasic disposition profiles; the concentration in the blood showed two distinct phases of decline. The terminal phase of 7-hydroxymitragynine was parallel with mitragynine in participants, suggesting that its elimination may be formation rate-limited, meaning its disappearance from the plasma was controlled by the rate at which it was formed from mitragynine. The authors also noted that the maximum concentration (C_{max}) and the area under the plasma concentration-time curve (AUC_{inf}) of 7-hydroxymitragynine were approximately 24-27% of the corresponding values for mitragynine, respectively. This suggested that 7-hydroxymitragynine was present at lower concentrations and had a shorter duration of detectability in plasma compared to mitragynine.

161. The human pharmacokinetics of mitragynine as reported by Tanna et al. (2022) and Huestis et al. (2024) detailed later, is summarized below in Table 2.

Table 2. Pharmacokinetic parameters for mitragynine as reported by Tanna et al. (2022)and Huestis et al. (2024).

Huestis et al. (2024)

Participant #	**Formulation**	**Dose (mg)**	**Schedule**	**C_{max} (ng/ml)**	**Median T_{max} (hours)**	**$T_{1/2}$ (hours)**	**Median T_{last} (hours)**
12	1 capsule	6.65	SD	17.1 (15.2)	1	8.5 (11.8)	12
12	2 capsules	13.3	SD	32.1 (12.2)	1.3	15.8 (16.3)	23.4
12	4 capsules	26.6	SD	57.0 (25.1)	1.3	29.3 (17.5)	71.6
13	8 capsules	53.2	SD	125 (51.8)	1.3	43.4 (23.3)	120
		Dose (mg)	**Schedule**	**C_{max}**	**Median $T_{max,ss}$[a] (hours)**	**$T_{1/2,ss}$[a] (hours)**	**Median $T_{last,ss}$[a] (hours)**

11	1 capsule	6.65	MD, day 15	21.4 (18.9)	1	25.7 (24.0)	23.9
8	2 capsules	13.3	MD, day 15	33.5 (7.43)	1.7	44.1 (24.2)	95.4
9	4 capsules	26.6	MD, day 15	74.6 (22.8)	1.7	67.9 (20.6)	168
9	8 capsules	53.2	MD, day 15	143 (50.4)	1.7	51.1 (15.9)	169

Tanna et al. (2022)

Participant #	**Formulation**	**Dose (mg)**	**C_{max} (ng/ml)**	**T_{max} (hours)**	**$AUC_{0\text{-}inf}$ (nM*h)**	**$T_{1/2}$ (hours)**	**CL/F (l/h)**	**Vz/F (l)**
5	2 g leaf powder in tea	38.96	32.6 (20.0-70.1)	1 (0.75-1.5)	167 (129-542)	45.3 (31.9-50.2)	233 (71.7-302)	12,700 (5,190-19,700)

162. C_{max}: maximum plasma concentration; T_{max}: time to C_{max}; $AUC_{0\text{-}inf}$: area under the plasma-concentration time curve from time zero to infinity; $T_{1/2}$: elimination half-life; Cl/Fand CL/F: apparent clearance; Vz/F: apparent volume of distribution; $AUC_{o\text{-}Tlast}$: area under the plasma-concentration time curve from dosing through last detection; T_{last}: time to last detection; C_{trough}: lowest plasma concentration between doses.

163. [a]Steady-state (SS) values in multi-dosing study.

Huestis et al. (2024)

164. Huestis et al. (2024) performed a pharmacokinetic study on mitragynine and 7-hydroxymitragynine in healthy adult participants. The study was a randomized, double-blind, placebo-controlled, dose-escalation trial that evaluated the pharmacokinetics of single and multiple oral doses of encapsulated dried kratom leaf powder.

165. The authors reported that mitragynine was absorbed after oral administration of encapsulated dried kratom leaf powder. The median time to reach maximum plasma concentration (T_{max}) after single doses (SD) ranged from 1.0 to 1.3 hours, indicating relatively rapid absorption. The T_{max} was slightly longer for multiple doses (MD), ranging from 1.0 to 1.7 hours. The authors noted that the formulation of the kratom product (encapsulated powder versus tea) may have affected the absorption rate, with encapsulated forms possibly leading to a longer T_{max}. The study did not report details regarding the distribution of mitragynine within the body.

166. Regarding metabolism, the authors noted that mitragynine was metabolized primarily in the liver, with the CYP3A4 enzyme playing a major role in its metabolism to 7-hydroxymitragynine. The terminal half-life ($T_{1/2\beta}$) of mitragynine increased with the dose, which indicated that higher doses resulted in longer detection times in plasma. Following single dosing, mitragynine concentrations were measurable in some subjects up to 10 days post-dosing, suggesting a prolonged excretion process. The study indicated that a washout period of at least 14 days may be necessary after multiple dosing regimens to ensure clearance of mitragynine from the body. In conclusion, mitragynine was rapidly absorbed after oral administration, metabolized by liver enzymes (particularly CYP3A4), and excreted over an extended period, with detectable plasma concentrations persisting for several days after dosing.

167. The authors reported that median times to reach peak 7-hydroxymitragynine concentrations following oral ingestion (T_{max}) ranged from 1.2 to 1.8 hours after a single dose (SD) and 1.3 to 2.0 hours after multiple doses (MD) of kratom ranging from 6.65 to 53.2 mg mitragynine per dose. The authors did not describe the distribution phase for 7-hydroxymitragynine. However, the pharmacokinetic parameters, such as the apparent volume of distribution (Vz/F), are relevant to understanding its distribution, which can be inferred from the reported data.

168. As noted elsewhere, 7-hydroxymitragynine is primarily formed through the metabolism of mitragynine by the CYP3A4 enzyme, as indicated by *in vitro* studies using human liver microsomes. Huestis et al. (2024) reported that the 7-hydroxymitragynine to mitragynine concentration ratios were higher following single dosing relative to multiple dosing regimens, and higher ratios were observed at lower doses. This suggested dose-dependent metabolism. In addition, the terminal half-life ($T_{1/2\beta}$) of 7-hydroxymitragynine increased with the dose, with a median $T_{1/2\beta}$ of 4.0 hours after the highest single dose and 9.1 hours after the highest multiple dose, which indicated a prolonged excretion process. The authors reported that the accumulation of 7-hydroxymitragynine was low to moderate, with no or low accumulation after multiple dosing, which is relevant for understanding its clearance from the body.

169. In conclusion, Huestis et al. (2024) provided a comprehensive overview of the pharmacokinetic behavior of 7-hydroxymitragynine, showing that it is absorbed, metabolized from mitragynine, and excreted over time, with a longer half-life observed at higher doses.

Pharmacokinetics When Co-Administered with Other Compounds

170. The pharmacokinetics of individual alkaloids cannot always predict the pharmacology of the overall mixture of alkaloids in kratom products due to the presence of multiple compounds at different concentrations, which could influence each compounds pharmacokinetics and pharmacodynamics.

171. Kamble et al. (2021) investigated the pharmacokinetics of 11 alkaloids following oral administration of kratom in rats, including Corynantheidine (COR) and its receptor affinity. The study aimed to evaluate the systemic exposure of individual kratom alkaloids following the administration of different kratom preparations, using a bioanalytical method developed and validated in accordance with FDA guidelines. The method employed ultraperformance liquid chromatography-tandem mass spectrometry (UPLC-MS/MS) for the simultaneous quantification of the 11 kratom alkaloids in rat plasma. The method was optimized to reduce analysis time to 11 minutes and improve detection, with a linear range of 1-200 ng/ml for each alkaloid. Pharmacokinetic studies were conducted on male Sprague-Dawley rats, with systemic exposure assessed by measuring the area under the curve ($AUC_{0\text{-}24h}$) using the linear-up log-down trapezoidal method. The study also reported recovery and matrix effects, ensuring minimal ion suppression and high recovery rates for the alkaloids.

172. The authors reported that, of the 11 kratom alkaloids, only mitragynine, 7-hydroxymitragynine, speciociliatine, and corynantheidine showed systematic exposure in rats at eight hours post-dose. A commercial OPMS liquid kratom product resulted in a 1.6 to 2.4-fold higher dose-normalized systemic exposure of these alkaloids compared to traditional lyophilized kratom tea. The study also reported that the commercial product led to delayed T_{max} and increased $AUC_{0\text{-}24h}$ for mitragynine, speciociliatine, and corynantheidine, suggesting slower absorption and greater systemic exposure. These results highlighted the differences in pharmacokinetics between

traditional and commercial kratom preparations, which could have implications for the effects and safety profiles across different product types. However, detailed pharmacokinetic data such as absorption, distribution, metabolism, and excretion (ADME) were not explicitly detailed in this study.

173. Mongar et al. (2024) conducted a pharmacokinetic study investigating the effects of itraconazole on the metabolism of mitragynine and 7-hydroxymitragynine in human volunteers. The authors developed and validated a liquid chromatography-mass spectrometry (LC/MS) method for analyzing mitragynine and 7-hydroxymitragynine in human plasma. Their analysis demonstrated good linearity, precision, and accuracy, meeting FDA guidelines for bioanalytical methods. Sixteen participants were recruited, and various pharmacokinetic parameters, such as maximum plasma concentration (C_{max}), time to reach C_{max} (T_{max}), half-life ($T_{1/2}$), and area under the curve (AUC), were measured.

174. In the study, the pharmacokinetics of itraconazole indicated that it acted as a strong CYP3A4 inhibitor. This is relevant because mitragynine is metabolized into the more potent compound 7-hydroxymitragynine primarily through the CYP3A4 pathway. When itraconazole was co-administered, it inhibited this metabolic pathway, which led to a significant reduction in the formation of 7-hydroxymitragynine from mitragynine. The authors specified that itraconazole had an absolute bioavailability of about 55%, and its greatest absorption occurred when taken after a full meal. Itraconazole and its metabolite, hydroxy-itraconazole, were both substrates and inhibitors of CYP3A4, which affected their own metabolic clearance as well as that of other drugs, such as mitragynine. The half-lives of itraconazole and hydroxy-itraconazole were reported to be 20-24 hours and 12 hours, respectively.

175. During the study by Mongar et al. (2024), the pharmacokinetic parameters of mitragynine and 7-hydroxymitragynine were assessed in two periods: without itraconazole (Period 1) and with itraconazole pretreatment (Period 2). In Period 2, the peak plasma concentration (C_{max}) and the area under the curve (AUC) of 7-hydroxymitragynine decreased significantly, indicating that itraconazole effectively inhibited the metabolism of mitragynine to 7-hydroxymitragynine.

The time to reach maximum concentration (T_{max}) of 7-hydroxymitragynine was also delayed in Period 2, further supporting the inhibitory effect of itraconazole on the conversion of mitragynine to 7-hydroxymitragynine.

176. The results of the study indicated that when itraconazole was co-administered with mitragynine it led to a decrease in the formation of 7-hydroxymitragynine due to the inhibition of the CYP3A4 enzyme by itraconazole, which is reflected in the pharmacokinetic parameters of 7-hydroxymitragynine. This interaction is crucial for understanding the potential effects on the analgesic properties of mitragynine and the safety profile when combined with itraconazole.

177. Both the (Kamble et al. 2021) and (Mongar et al. 2024) studies collectively emphasized the complexity of kratom's pharmacokinetics and the significant impact of interactive effects on the metabolism of its active compounds.

Pharmacokinetics of Other Kratom Alkaloids

178. Kratom contains more than 40 different alkaloids, including mitragynine and 7-hydroxymitragynine (7-HMG), which have agonist morphine-like effects on opioid receptors (Firmansyah et al. 2021; Ahmad et al. 2022). Firmansyah et al. (2021) reported that six of the more than 40 alkaloids within kratom have psychoactive properties, such as ragynine, 7-HMG, mitraphylline, speciociliatine, speciogynine, and paynantheine. Kratom is part of the Rubiaceae family, which is native to Africa and Asia and also includes the shrubs responsible for coffee production (*Coffea arabica* and *C. canephora*). Similar to alkaloids derived from the *Coffea* species and other members of the Rubiaceae family, kratom-derived alkaloids have dose-dependent stimulant properties (Ahmad et al. 2022). The *Mitragyna* genus consists of 10 species, including four species found in Africa and six species found in Asia (Brown et al. 2017; Ahmad et al. 2022).

179. Brown et al. (2017) conducted a systematic review of literature regarding the chemical contents of kratom and reported as many as 57 secondary metabolite compounds within the species, including 37 that were found only in *M. Speciosa*. It was reported that indole and oxindole alkaloids were the chemical constituents present in kratom that were the most likely cause

of the plant's pharmacological effects. The reported indole and oxindole alkaloids in kratom include mitragynine, 7-hydroxymitragynine, paynantheine, mitralactonal, mitragynaline, speciociliatine, speciogynine, mitrasulgynine, 3,4,5,6-tetradehydromitragynine, mitragynaline, mitragynalinic acid, corynantheidinaline, corynantheidinalinc acid, 3 dehydromitragynine, 9-methoxymitralactonine, 3-isopaynantheine, ajmalicine, isocorynantheidine, mitragynine pseudoindoxyl, mitraphylline, mitragynine oxindole A, mitragynine oxindole B, corynoxine, corynoxine B, mitraciliatine, 7_-hydroxy-7H-mitraciliatine, isomitraphylline, rhynchophylline, rhyncocilline, cilaphylline, isospeciofoleine, isospeciofoline, and isorotundifoline (Brown et al. 2017; Firmansyah et al. 2021; Ahmad et al. 2022).

180. There is limited to no toxicological or pharmacological data for most kratom alkaloids. Pharmacokinetic and pharmacodynamic data, in particular, is quite sparse. From the literature, outside of information on mitragynine and 7-hydroxymitragynine, data were identified for speciogynine, speciociliatine, paynantheine, and corynantheidine. This is discussed briefly below.

Speciogynine Pharmacokinetics

181. The pharmacokinetics of speciogynine (SPG) following oral administration in rats are discussed briefly in Kamble et al. (2021). It was reported that SPG and paynantheine (PAY) were quantifiable in the systemic circulation up to one hour post-dose. The maximum plasma concentrations (C_{max}) for SPG were found to be 2.9 ± 0.8 ng/ml at a T_{max} (time to reach maximum plasma concentration) of 10.0 ± 0.0 minutes following the administration of lyophilized kratom tea (LKT). For a commercial OPMS liquid kratom product, the C_{max} was also 2.9 ng/ml, but the quantifiable levels were observed in only one rat at a T_{max} of 5.0 minutes. Beyond these time points, the concentrations of SPG dropped below the limit of quantitation, which was 1 ng/ml. This indicated that SPG was rapidly absorbed and cleared from the plasma within the first hour following oral administration in rats.

Speciociliatine Pharmacokinetics

182. Kamble et al. (2021) reported the key pharmacokinetic parameters for speciociliatine (SPC) following oral administration of LKT and OPMS liquid. The authors reported that the maximum plasma concentration (C_{max}) for SPC was 21.1 ± 3.3 ng/ml following LKT administration and 23.8 ± 1.4 ng/ml following OPMS liquid administration. They noted that the time to reach maximum plasma concentration (T_{max}) for SPC was 1.8 ± 0.7 hours after LKT administration and 3.2 ± 1.6 hours after OPMS liquid administration. The area under the plasma concentration-time curve from 0 to 24 hours ($AUC_{0\text{-}24h}$) for SPC was 107.4 ± 25.4 h·ng/mL following LKT administration and 222.7 ± 22.2 h·ng/mL following OPMS liquid administration. The dose-normalized AUC0-24h ($AUC_{0\text{-}24h/dose}$) for SPC was 52.9 ± 12.5 h·kg·ng/ml/mg following LKT administration and 101.2 ± 10.1 h·kg·ng/ml/mg following OPMS liquid administration, indicating a 1.9-fold increase in systemic exposure to SPC with the OPMS liquid compared to LKT. These results suggested that SPC had a slower rate of absorption and increased systemic exposure when administered as part of the OPMS liquid kratom product compared to the traditional LKT.

Paynantheine Pharmacokinetics

183. The pharmacokinetics of paynantheine (PAY) following oral administration in rats were described in Kamble et al. (2021). Paynantheine, along with speciogynine (SPG), was quantifiable in the systemic circulation up to one hour post-dose. The maximum plasma concentration (C_{max}) values for PAY were observed at 5.3 ± 1.5 ng/ml at a T_{max} (time to reach maximum plasma concentration) of 10.0 ± 0.0 minutes following the administration of lyophilized kratom tea (LKT). For a commercial OPMS liquid kratom product, the C_{max} was 2.0 ± 1.7 ng/ml at a T_{max} of 5.0 ± 0.0 minutes. After these time points, the concentrations of PAY dropped below the limit of quantitation, which is less than 1 ng/ml. This indicated that PAY was rapidly absorbed and cleared from the plasma within the first hour after oral administration in rats.

Corynantheidine Pharmacokinetics

184. The pharmacokinetics of corynantheidine (COR) are discussed in the context of its evaluation following oral administration in rats. In the study by Kamble et al. (2021) it was

indicated that COR was one of the kratom alkaloids that showed measurable systemic exposure eight hours post-dose when administered either as traditional lyophilized kratom tea (LKT) or as a commercial product (OPMS liquid kratom).

185. It was also mentioned that COR had an antagonistic effect on μORs, with a K_i (inhibition constant) of 118 nM, and a higher affinity for α1-adrenergic receptors with a K_i of 41.7 nM. This suggested that COR contributed to the overall pharmacological effects of kratom by modulating opioid and adrenergic receptor activity. Additionally, COR, along with mitragynine, has been shown to inhibit CYP2D6 enzymes, which could affect the metabolism of kratom alkaloids and potentially other substances metabolized by this enzyme.

Comparison to Pharmacokinetics of other Botanical and Medicinal Products

186. Theobromine, caffeine, yohimbine, and kratom (mitragynine) are naturally derived alkaloids with psychoactive properties that affect the central nervous system. Caffeine is found in coffee beans, tea leaves, and cocoa beans; theobromine is primarily found in cocoa beans and chocolate; yohimbine is extracted from the bark of the Yohimbe tree; kratom is derived from the leaves of the *M. speciosa* tree. They interact with specific receptors or enzymes to influence physiological and psychological states, varying from stimulation to sedation. Commonly used both medicinally and recreationally, these compounds enhance alertness, mood, and physical performance, although they differ in their primary effects and mechanisms of action. Each demonstrates a capability to be utilized in managing fatigue and altering mood, making them significant in both therapeutic settings and everyday use. Their natural alkaloid classification highlights a shared chemical characteristic that contributes to their bioactive profiles.

187. Pharmacokinetic properties associated with 7-hydroxymitragynine are generally considered to be similar with mitragynine. The compounds display some similarity and differences in their pharmacokinetic properties.

Absorption

188. The pharmacokinetic absorption profiles of theobromine, caffeine, yohimbine, and kratom (mitragynine) have similar rates of uptake from the gastrointestinal tract. Theobromine is

absorbed more slowly, reaching peak plasma levels around 2-3 hours after ingestion, whereas caffeine is absorbed most rapidly, achieving peak concentrations within 15-45 minutes. Yohimbine and kratom are absorbed relatively quickly as well, with peak plasma levels typically reached within about one hour for yohimbine and approximately 1.5 hours for kratom (Mumford et al. 1996).

Distribution

189. These compounds are distributed throughout the body, though the extent to which they cross the blood-brain barrier, their affinity for specific tissues, and their volume of distribution vary. Theobromine is widely distributed through all body fluids with a larger volume of distribution than caffeine, indicating extensive tissue reach. Caffeine, known for its ability to cross the blood-brain barrier easily, is well-distributed throughout the body, including the brain, enhancing its stimulatory effects on the central nervous system. Yohimbine, like caffeine, crosses the blood-brain barrier and shows a high affinity for both fat tissues and the central nervous system, suggesting a targeted therapeutic action on these areas. Mitragynine is characterized by its high plasma protein binding and extensive distribution, which may influence its duration of action and efficacy in pain relief (Arnaud 1988, 2011).

Metabolism

190. The metabolism of theobromine, caffeine, yohimbine, and kratom's primary active component mitragynine occurs primarily in the liver, involving the cytochrome P450 enzyme system, albeit involving different specific enzymes (e.g., CYP1A2 for caffeine, CYP3A4 and CYP2D6 for mitragynine). This shows that the compounds share a common metabolic pathway but varied specifics in metabolic processing (Owen et al. 1987; Philipp et al. 2011a; Philipp et al. 2011b). The engagement of the cytochrome P450 system in their metabolism suggests potential interactions with other compounds metabolized by this system, affecting efficacy and safety profiles.

Excretion

191. All four substances are predominantly excreted through the urine, underscoring the kidneys' role in clearing these compounds from the body. Each compound is excreted not only as metabolites but also in its unchanged form to varying extents, indicating that direct renal excretion occurs alongside metabolite elimination. Kratom (mitragynine) is mostly excreted in the urine in the form of metabolites, with a significant portion also excreted unchanged, similar to the excretion patterns observed with caffeine and yohimbine.

Conclusions on Kratom Pharmacokinetics

192. The pharmacokinetics of kratom, particularly its primary alkaloids mitragynine and 7-hydroxymitragynine, reveal a complex interaction within the human body that is influenced by various factors including dose, frequency of use, and the form of kratom preparation. Studies have shown that these alkaloids are rapidly absorbed into the bloodstream, with peak concentrations typically reached within hours of ingestion. Mitragynine exhibits a biphasic elimination pattern with a terminal half-life suggesting rapid metabolism and minimal potential for accumulation in tissues with typical use patterns. The pharmacokinetics of kratom are further complicated by the presence of multiple compounds in the plant, which can affect each compound’s absorption, distribution, metabolism, and excretion. This complexity is underscored by findings that commercial kratom products can lead to higher systemic exposure and delayed absorption compared to traditional preparations.

193. The pharmacodynamics of mitragynine and 7-hydroxymitragynine, as partial agonists of the human μ-opioid receptor (μOR), suggest potential therapeutic applications, particularly in pain management, while also highlighting the risks of adverse interactions with drugs metabolized by cytochrome P450 (CYP) enzymes. The high plasma protein binding of 7-hydroxymitragynine indicates a prolonged presence in the bloodstream, which, coupled with its rapid metabolism, suggested a complex excretion pattern likely involving metabolites. However, these alkaloids are partial agonists at the human μOR, showing functional selectivity for G-protein signaling without significantly recruiting β-arrestin, which is associated with the adverse effects of traditional opioids. This selective binding potentially contributes to fewer side effects, making

kratom a candidate for therapeutic use with a possibly lower risk profile compared to traditional opioids.

Basis of Opinion 4

The Importance of In Vivo Studies of Kratom

194. Human data is the gold standard when evaluating the toxicological and pharmacological effects of a compound. Specifically, controlled clinical studies that include an array of doses can help provide information about a compound's beneficial and adverse effects. For kratom, several previously detailed pharmacokinetic studies clarified how mitragynine and 7-hydroxymitragynine are absorbed, distributed, metabolized, and excreted from the human body (Trakulsrichai et al. 2015; Tanna et al. 2022; Huestis et al. 2024). However, these studies, while informative, provided minimal insight into the beneficial or adverse effects of kratom alkaloids at specific doses. That is, it can be concluded from the pharmacokinetic analyses that tested doses (which at times exceeded 50 mg/kg-bw mitragynine) did not yield overt signs of human toxicity, but they by design did not establish doses at which specific health effects occurred. Similarly, the epidemiological and case report/series data for kratom, which I discuss later in my report, cannot be used to determine dose-specific alkaloid effects.

195. The above limitations of human pharmacokinetic, epidemiological, and case report/series inquiries highlight the importance of *in vivo* data. No animal studies present a perfect model of potential human outcomes. Yet *in vivo* methods can help inform toxicological conclusions and risk assessments in the absence of robust human data on adverse effect levels, which is the case for kratom. Moreover, such studies can be more easily extrapolated to human effects than *in vitro* tests even though the latter methods have provided valuable insight into the mechanisms driving kratom's physiological activity.

196. The *in vivo* data on kratom and its alkaloids has yet to fully resolve whole body or organ/tissue-specific dose effects across a broad suite of routes and frequency of administration (i.e., no-observed-adverse-effect-levels (NOAELs) and lowest-observed-adverse-effect-levels (LOAELs) need to be better established). Nonetheless, the information generally indicates that

alkaloid doses associated with typical kratom use may provide some beneficial effects, and few to no adverse effects, in most persons.

How My Literature Searches Were Performed (Methods)

197. To further explore the potential health effects of kratom, a detailed review of the existing literature was performed. Specifically, the literature was reviewed for references on direct kratom administration via leaves, teas, powders, capsules, and other derivations; mitragynine and 7-hydroxymitragynine; and other indole alkaloids. Search terms including "kratom," "mitragynine," "mitragynine metabolites," "7-hydroxymitragynine," and "alkaloid" were used to query kratom-related results, alongside terms including "in vivo" and "toxic*," which were used to identify published papers relevant to the health endpoints and processes of interest.

198. Peer consultation was received for studies with ambiguous inclusion/exclusion interpretations, with two or more reviewers jointly determining the relevance of such papers to this review. Additional studies were identified from review of references cited in the publications pulled in the initial search. The studies identified through this search are discussed in detail below, along with the implications of kratom products, mitragynine and its metabolites, and other alkaloids on specific health outcomes.

Kratom *In Vivo* Toxicology and Pharmacology

199. As described previously, kratom's pharmacological and toxicological properties are attributable to the numerous indole alkaloids present in the leaves of *M. speciosa* and subsequent metabolites of these alkaloids. Some literature describes the *in vivo* toxicology of direct kratom administration, such as via decoctions or various aqueous, ethanolic, or methanolic extracts (Hassan et al. 2013; Hiranita et al. 2022; Hughes et al. 2022). However, the majority of pertinent *in vivo* literature is focused on the toxicology of independently administered mitragynine, mitragynine salts, or 7-hydroxymitragynine. The effects of mitragynine were first studied by Field (1921) and Grewal (1932b). Additional research has examined the toxicology of mitragynine metabolites and mitragynine pseudoindoxyl, along with the effects of various associated alkaloids, most notably speciociliatine and corynantheidine (Hiranita et al. 2022). A summary of key *in vivo*

toxicological values for kratom, its alkaloids, and its metabolites is presented below in Tables 3 and 4.

Table 3. Acute toxicity of mitragynine, 7-hydroxymitragynine, and kratom alkaloid extract in vivo. Redrawn from (Heywood et al. 2024)

	Animal	**Sex[a]**	**Route of Administration**	**Value (mg/kg-bw)**	**Study**
Mitragynine	Frogs		Intravenous	LD_{Lo} = 130	(Grewal 1932b)
	Rabbits		Intravenous	LD_{Lo} = 130	
	Mice		Subcutaneous	LD_{Lo} = 370	
	Mice		Intraperitoneal	LD_{Lo} = 200	
	Rats		Intravenous	LD_{50} = 27.8	(Macko et al. 1972)
	Swiss Albino Mice	Male	Oral	LD_{50} = 477.1	(Sabetghadam et al. 2013a)
	Swiss Albino Mice	Male	Intraperitoneal	LD_{50} = 1,098	
	Swiss Albino Mice	Male	Intravenous	LD_{50} = 27.8	(Smith et al. 2019)
	Swiss Albino Mice	Male	Oral	LD_{50} = 547.7	
	129Sv6 Mice		Oral	LD_{50} = 750	(Chakraborty et al. 2021)
7-Hydroxy-mitragynine	Swiss Albino Mice	Male	Intravenous	LD_{50} = 24.7	(Smith et al. 2019)
Kratom Alkaloid Extract	Swiss Albino Mice		Oral	LD_{50} = 173.2	(Reanmongkol et al. 2007)
	Swiss Albino Mice	Male	Oral	LD_{50} = 591.6	(Sabetghadam et al. 2013a)

LD_{Lo}, lowest observed lethal dose; LD_{50}, median lethal dose.

[a]Not reported if blank.

Table 4. Summary of studies reporting median effective dose (ED) values for mitragynine, 7-hydroxymitragynine, or kratom alkaloid extracts in vivo. Redrawn from Heywood et al. (2024)

	Animal	**Sex[a]**	**Route of Administration**	**Effect**	**Value (mg/kg-bw)[b]**	**Study**
Mitragynine	Sprague-Dawley Rats	Male	Oral	Analgesia	ED_{50} = 16.8-20.2[c]	Macko et al. (1972)
	Sprague-Dawley Rats		Intraperitoneal	Analgesia	ED_{50} = 14.4	
	CF1 Mice		Oral	Analgesia	ED_{100} = 92	
	CF1 Mice		Subcutaneous	Analgesia	ED_{0} = 92	
	Beagle Dogs		Oral	Antitussive	ED_{50} = 1.8-2.3[d]	
	Swiss Albino Mice		Oral	Analgesia	ED_{50} = 21.96	Sabetghadam et al. (2013a)
	129S1 Mice		Oral	Analgesia	ED_{50} = 2.05	Kruegel et al. (2019)
	129S1 Mice		Subcutaneous	Analgesia	ED_{50} = 106	
	129Sv6 Mice		Intraperitoneal	Antinociception	ED_{50} = 4	Chakraborty et al. (2021)
	129Sv6 Mice		Subcutaneous	Antinociception	ED_{50} = 57.3	
	Sprague-Dawley Rats	Males and Females	Intraperitoneal	Lower lip retraction	ED_{50} = 26.8	Leon et al. (2021)
7-Hydroxy-mitragynine	129S1 Mice	Not Specified	Subcutaneous	Antinociception	ED_{50} = 0.6	Kruegel et al. (2019)
	C57BL/6J Mice	Not Specified	Subcutaneous	Antinociception	ED_{50} = 0.3	Chakraborty et al. (2021)

	129Sv6 Mice	Not Specified	Subcutaneous	Antinociception	ED_{50} = 0.2	
Kratom Alkaloid Extract	Swiss Albino Mice	Male	Oral	Antinociception	ED_{50} = 194.4	Sabetghadam et al. (2013a)
	C57BL/6J Mice		Oral	Antinociception	ED_{50} = 17.1	Chakraborty et al. (2021)
	129Sv6 Mice		Oral	Antinociception	ED_{50} = 35	

[a]Not reported if blank.

[b]Reported as the mean unless otherwise specified.

[c]Low estimate based on Randall and Selitto methods, high estimate based on D-Amour and Smith methods.

[d]Mean of three animals per dose for low estimate (range = 1.0-2.6 mg/kg-bw); mean of five animals per dose for high estimate (range = 1.3-3.3 mg/kg-bw).

Mitragynine

200. In a study robust for its time, Grewal (1932b) performed *in vivo* and *ex vivo* analyses of mitragynine in frogs, mice, rabbits, and cats, along with presenting some information on human-specific effects. The author evaluated the effects of mitragynine fumarate and mitragynine acetate salts. In an analysis of repeated-dose toxicity, Grewal (1932b) administered 15 mg/kg-bw mitragynine to rabbits for "several" weeks. This was reported to yield "… a progressive loss of weight …" and coat deterioration and balding. The author reported that lowest lethal dose (LD_{Lo}) of mitragynine administered intravenously in frogs and rabbits was 0.13 g/kg for both species. In mice, the LD_{Lo} was reported to be 0.37 g/kg via subcutaneous injection and 0.20 g/kg via intraperitoneal injection. In frogs, the author stated that acute effects included paralysis and respiratory cessation, with death observed after eight hours. In mice and rabbits, symptoms of acute mitragynine toxicity included "… exaggerated reflexes, tremors, muscular incoordination … convulsions, and … respiratory failure." Grewal (1932b) added that humans dosed orally with 50 mg mitragynine showed "… motor excitement, followed by giddiness, Rombergism, and tremors

of the extremities, face, and tongue." In short, mitragynine demonstrated *in vivo* acute toxicity that could be described as moderate (Hodge and Sterner 1949).

201. Decebrate (immobilized) cats dosed intravenously with 15 to 20 mg mitragynine were examined for intestinal movement to determine the effects of mitragynine on smooth muscle fibers. It was reported that the tested mitragynine doses reduced the intestinal threshold for stimulation associated with the vagus nerve. Administration of atropine was reported to eliminate the effects of mitragynine, prompting the author to conclude that mitragynine was "… not likely to act directly on muscle fibers" (Grewal 1932b).

202. In examining the effects of mitragynine on striped muscle fibers, Grewal (1932b) added that unspecified doses of mitragynine elicited "… a progressive fall in the power of muscle contraction" in unspecified frog species. The excitability was reported to decrease continuously over time, with frog muscle fatigue increasing at an expedited rate in the presence of mitragynine relative to its absence. It was reported that mitragynine depressed cardiac muscle contraction in the hearts of frogs and rabbits while simultaneously increasing coronary vessel outflow. Similarly, intravenous administration of 2.5 mg mitragynine in anesthetized frogs, rabbits, and cats demonstrated that mitragynine "… had the general effect of dilating the vessels … caus[ing] a marked fall in blood pressure, which recover[ed] quickly after a single dose." Repeated administration of mitragynine elicited a continued depression of blood pressure and increased cardiac output associated with the aforementioned vasodilation. However, the author concluded that these effects were "… not due to any direct action of [mitragynine] on the heart …" but due to "… dilating blood vessels" (Grewal 1932b).

203. Collectively, the results reported by Grewal (1932b) are a helpful introduction to the *in vivo* effects of mitragynine and kratom. Despite this, the dated approach of the study and poorly specified methods make generalization to current products and exposure scenarios difficult. Moreover, though Grewal (1932b) failed to specify doses for many examined endpoints, those doses specified are, in light of modern toxicological and pharmacological knowledge, relatively high and unrepresentative of typical use scenarios for kratom products. Further, the results reported

specific to muscle activity in cats, rabbits, and frogs, while informative, do not provide detailed mechanistic insight about mitragynine's activity, relative to more recent literature. They indicate that mitragynine has few direct cardiotoxic effects, but that it does act as a vasodilator.

204. In another early study, Macko et al. (1972) evaluated the antinociceptive, antitussive, nalorphine antagonism, cardiotoxicity, and acute and subacute toxicity of mitragynine in Sprague-Dawley rats, cats, and dogs. The reported antinociceptive oral ED_{50} for mitragynine salts in rats ranged from 16.8 to 20.2 mg/kg-bw based on the methods of D'Amour and Smith (1941) and Randall and Selitto (1957). Analgesic effects were also reported in dogs at 2.0, 4.0, 8.0, 12.0, 16.0, and 24.0 mg/kg-bw mitragynine via oral administration, though dose-dependence was unclear or absent and adverse side effects including shivering and decreased spontaneous motor activity were also observed across doses. In the hot plate test in mice (54.5 ºC), 92 mg/kg-bw mitragynine yielded 100% analgesia via oral administration, but no analgesia was reported for a similar dose via subcutaneous administration. In dogs, the antitussive oral ED_{50} for mitragynine HCl and mitragynine ethane disulfonate salts were reported to be 2.3 and 1.8 mg/kg-bw, respectively. The authors stated that intraperitoneal (36.8 mg/kg-bw) and oral (55.2 mg/kg-bw) mitragynine salts failed to inhibit gastrointestinal activity in dogs based on the charcoal meal test.

205. Cardiovascular specific results reported by Macko et al. (1972) were mixed. In anesthetized dogs, intravenous mitragynine salt doses of 0.092 to 9.2 mg/kg-bw were reported to cause no adverse effects on blood pressure. In anesthetized cats administered intravenous mitragynine salts, weak hypotension was reported at 0.46 to 0.92 mg/kg-bw in some animals and only at 4.6 mg/kg-bw in other animals. Intravenous mitragynine salts at 0.46 to 2.3 mg/kg-bw yielded "… a transient lowering of mean arterial blood pressure" in one cat, which later died due to respiratory failure at 4.6 mg/kg-bw.

206. In analyses of acute and subacute effects, Macko et al. (1972) reported comparatively equivocal toxicity for mitragynine. Oral doses of up to 806 mg/kg-bw failed to produce overt signs of toxicity in rats. Similarly, oral administration of mitragynine salts at 8 mg/kg-bw/day for five days yielded only diarrhea in 25% of test rats with no other adverse effects,

and dogs dosed orally with 16 mg/kg-bw/day for five days followed by 32 mg/kg-bw/day for two days exhibited no adverse effects. Oral administration of mitragynine salts at 5 or 50 mg/kg-bw/day, five days per week for six weeks yielded no significant adverse effects in rats. In dogs, oral administration of 20 mg/kg-bw/day mitragynine salts six days per week for three weeks yielded no adverse effects. However, subsequent administration of 40 mg/kg-bw/day in these dogs from days 22 to 50 resulted in "… leukopenia, granulocytopenia, lymphocytosis, monocytosis, and atypical and immature lymphocytes." Such effects diminished after dosing was ceased. Given a lack of significant adverse effects, the authors did not report any LD_{50} values.

207. Collectively, the results reported by Macko et al. (1972) indicate several things. First, and similar to Grewal (1932b), the acute toxicity of mitragynine was moderate to low (Hodge and Sterner 1949). Second, virtually no adverse effects were reported for moderate duration (i.e., <6 week) repeated mitragynine dosing, apart from diarrhea. The mitragynine doses in many marketed kratom products, when adjusted for the weight of typical human users, are generally below those to which Macko et al. (1972) repeatedly exposed test animals, with few to no adverse effects. Third, the reported analgesic effects of mitragynine align with expectations, including the lack of analgesia noted for subcutaneous administration, since this route precludes rapid metabolism of mitragynine to 7-hydroxymitragynine in the liver (with the latter compound being the more active analgesic agent).

208. More recently, Moklas et al. (2008) evaluated the effects of mitragynine on locomotor activity similar to the muscle-focused analyses of Grewal (1932b). Male Sprague-Dawley rats (n = 40) were administered 1.0, 5.0, 10.0, and 30.0 mg/kg-bw mitragynine via intraperitoneal injection 30 to 40 minutes prior to behavioral analysis in a 75 x 75 x 42 cm locomotor box. The activity of the animals was evaluated based on frequency of travel between 15 x 15 cm sub squares within the box (measured as total transits between grid squares over 60 minutes). (Moklas et al. 2008) reported that locomotor activity was not significantly altered in animals dosed with mitragynine relative to vehicle and saline controls. This is not surprising given the animal doses and peritoneal route of administration, which, similar to above, limits rapid

metabolism of mitragynine to other more active alkaloids in the liver, relative to oral or intravenous routes.

209. In an analysis conducted congruent to the OECD Test Guidelines on Acute Oral Toxicity, Sabetghadam et al. (2013a) derived oral doses expected to cause death in 50% of test animals (the LD_{50}) via the EPA's AOT425statPgm software package. Male Swiss albino mice were exposed to sequential oral doses of mitragynine from 175 to 1,300 mg/kg-bw. It was reported that the oral LD_{50} value for mitragynine was 477.1 mg/kg-bw. The authors also calculated the effective dose (ED) for mitragynine via a hot plate test with Swiss albino mice. In this analysis, varying doses of mitragynine were administered to animals, which were placed on a hot plate at 52 ºC 15 minutes later and observed for 105 minutes in total. The authors defined the nociceptive reaction based on hind paw licking and body shaking. The maximum possible nociceptive effect (MPE) was derived from tested reaction latency time and later used to derive an effective dose among 50% of test animals (ED_{50}). The oral ED_{50} value for mitragynine was reported to be 21.96 mg/kg-bw. Subsequent derivation of the therapeutic index (TI), where $TI = LD_{50}/ED_{50}$, resulted in a value of 21:1 for mitragynine. The authors contextualized this TI value relative to those for remifentanil, morphine, cocaine, and ethyl alcohol, which are 33,000:1, 70:1, 15:1, and 10:1, respectively (Sabetghadam et al. 2013a). These results confirm that mitragynine's acute toxicity is moderate to low, and, based on its nociceptive action, the therapeutic index indicates the compound (and kratom) has a safer margin of use than alcoholic beverages.

210. Sabetghadam et al. (2013b) treated male and female Sprague-Dawley rats orally with 1, 10, and 100 mg/kg-day mitragynine dissolved in Tween-20 solution (total volume of 5 ml/kg) for 28 days. The authors reported that "[t]he lowest dose corresponded to the average daily intake of ketum users in Malaysia" and the highest dose was derived as one tenth of the LD_{50} from Sabetghadam et al. (2013b), which was 1,098 mg/kg-bw via intraperitoneal administration. Protocols were conducted in accordance with OECD guideline No. 407. The authors stated that no overt signs of toxicity were observed among animals at any dose, though piloerection was reported in male and female rats in the 10 mg/kg-bw and 100 mg/kg-bw/day groups "… during the last

week of treatment." They added that mild ptosis was observed in both sexes at 100 mg/kg-bw/day. With respect to hematology, red blood cells and white blood cells were decreased in animals dosed with 100 mg/kg-bw/day, neutrophils were increased in those dosed with 10 mg/kg-bw/day, and platelet count was significantly decreased in animals at all doses relative to controls. A significant increase in liver weight was reported in the 100 mg/kg-bw/day animals relative to controls, though no changes were reported for the kidneys, brain, lungs, spleen, and heart. Following histopathological analysis, adverse effects were noted in the liver and brain at the highest dose (Sabetghadam et al. 2013b).

211. Given the lack of overt signs of toxicity, the study by Sabetghadam et al. (2013b) implied that few adverse effects are expected with repeated doses of mitragynine that exceeded those of human kratom users by up to 10-fold. While some hematologic and hepatic effects were noted at 10 and 100 mg/kg-bw/day, the clinical significance of these changes (especially for blood parameters) is minimal to absent. Further, such changes only occurred at doses several-fold above those expected in human kratom users.

212. In a study otherwise focused on pharmacokinetic parameters, Kong et al. (2017b) administered a single intravenous 10 mg/kg-bw mitragynine dose (with an injection volume of 1 ml/kg) to adult female Sprague-Dawley rats. The kinetic results of this study are reviewed elsewhere in my report. The authors reported minimal data specific to toxic effects, but did report that intravenous injection of mitragynine resulted in "… good permeability across the blood brain barrier (BBB) ..." and that the percent distribution in brain tissues relative to that in plasma (AUC_{brain}/AUC_{plasma}) was 65.83 ± 4.54% (Kong et al. 2017b). Such penetration of the BBB has both pharmacological and toxicological significance and can help explain the adverse and abuse-liability related effects of mitragynine and kratom use. However, since human users typically ingest kratom, the blood concentrations of mitragynine in this study are likely over 10-fold higher than concentrations experienced by typical human users due to both the dose used and intravenous route of administration.

213. Smith et al. (2019) examined the effects of both mitragynine and 7-hydroxymitragynine on male Swiss Webster mice while developing a lateral flow assay for potential use by health care and law enforcement professionals. Specific to mitragynine, animals were dosed intravenously with 62.5, 250, or 500 mg/kg-bw during determination of an LD_{50}, and with 8, 15, 26, 31, or 62 mg/kg-bw mitragynine via intravenous administration during comparison to heroin potency. The reported intravenous LD_{50} for mitragynine was 27.8 mg/kg-bw, while the reported intravenous LD_{50} for heroin in this study was 23.7 mg/kg-bw. The authors specifically stated that "[m]ice appeared to expire from respiratory depression after receiving a bolus iv injection [of mitragynine] … with most overdose events occurring within the first 10 min of drug exposures." It was reported that seizure events were observed among surviving animals within 20 minutes of mitragynine administration (Smith et al. 2019). The authors hypothesized that mitragynine agonism at the mu opioid receptor (μ-OR, or MOR) contributed to its lethal effects in mice. As noted above, humans typically consume kratom orally, making the intravenous route of administration in this study, and the blood mitragynine concentrations obtained thereby, unrepresentative of real-world use scenarios. Nonetheless, the noted association between mitragynine's opioid receptor activity and lethal respiratory depression is informative with respect to adverse effects at high doses.

214. Kruegel et al. (2019) performed two *in vivo* analyses with mitragynine dosing. In one analysis, 129S1 mice were treated with 10 mg/kg-bw subcutaneous mitragynine, after which plasma and brain samples were collected at 15- and 60-minutes post-dosing and evaluated with LC-MS/MS. It was reported that mitragynine and 7-hydroxymitragynine (7-HMG) were detected at both time points in the brain, with higher brain penetration (approximately 1:1 brain/plasma) for 7-HMG than for mitragynine (approximately 1:5 brain/plasma). In another analysis, doses previously found to yield equivalent antinociceptive effects were administered subcutaneously to 129S1 mice; such doses were 140 mg/kg-bw for mitragynine and 0.7 mg/kg-bw for 7-HMG. Later tail flick analysis at 15 minutes post-exposure showed no significant differences between animals dosed with mitragynine and 7-HMG, along with "… no significant difference in the mean brain

concentration of [7-HMG] found in the mitragynine group (formed as a metabolite) compared to that found in the [7-HMG] group (from direct administration)." They reported that brain concentrations of mitragynine were substantially higher in the mitragynine dosed group than in animals dosed with 7-HMG, despite minimal to no difference in observed antinociceptive effects. Collectively, this prompted Kruegel et al. (2019) to conclude that 7-HMG "… formed as a metabolite is sufficient to explain the opioid-mediated analgesic activity of mitragynine and that the parent compound does not make a significant contribution to its own analgesic activity in mice." The specific effects reported by these authors for 7-HMG are discussed later in my report.

215. The effects noted by Kruegel et al. (2019) for mitragynine's antinociceptive action, relative to that of 7-HMG, coincide with evidence discussed elsewhere in my report. That is, while mitragynine does have direct antinociceptive activity, it's metabolism into 7-HMG following kratom intake is a more important predictor of health effects reported among human users.

216. Hassan et al. (2019) examined the effects of mitragynine on functional memory in male Sprague-Dawley rats. Immediately following daily Morris water maze training sessions, animals were treated intraperitoneally with 1, 5, or 10 mg/kg-bw/day mitragynine for 12 days. While no impairment was reported in animals treated with 1 mg/kg-bw/day, rats treated with 5 to 10 mg/kg-bw/day showed significantly prolonged escape latencies beginning on training days four to eight, relative to vehicle controls. The authors also examined electrophysiological effects in animals treated intravenously with a single dose of 5 or 10 mg/kg-bw mitragynine. It was reported that a single 10 mg/kg-bw dose reduced synaptic potentials, prompting the authors to conclude that mitragynine's effects on memory were induced by inhibition of Ca^{2+} flux (Hassan et al. 2019). In a similar study, Aznul et al. (2022) reported impaired escape latency in adolescent Sprague-Dawley rats orally administered 30 mg/kg-bw/day mitragynine for 15 days, but not in animals dosed with 3 or 10 mg/kg-bw/day mitragynine. All of the mitragynine doses (3, 10, 30 mg/kg-bw/day) were reported to result in reduced object recognition and social interaction time (Aznul et al. 2022).

217. Also in Sprague-Dawley rats, Foss et al. (2020) examined α-adrenoreceptor antagonism, locomotor activity, and allodynia (i.e., a traditionally nonpainful stimulus that unexpectedly yields pain) following mitragynine dosing. Animals were dosed intraperitoneally for all tests. In animals dosed with 0, 1, 5, or 10 mg/kg-bw/day mitragynine for five days after previously being injected with 6 mg/kg-bw oxaliplatin, the authors reported that the 5 and 10 mg/kg-bw/day mitragynine doses reduced oxaliplatin-induced allodynia. Single doses of 0, 1, 5, and 10 mg/kg-bw mitragynine were not reported to affect locomotor activity, though 30 mg/kg-bw significantly increased such activity. Further, mitragynine amelioration of oxaliplatin-induced allodynia was reduced in animals co-treated with yohimbine, prazosin, or naltrexone, indicating that kratom's effects are based on adrenergic activity, not just activity on opioid receptors.

218. In a study of multiple kratom-related compounds, Wilson et al. (2021) dosed male C57BL/6J mice twice daily for four days with an escalating dose of 10 to 75 mg/kg-bw oral mitragynine and evaluated antinociceptive effects with a warm-water tail-withdraw assay at 48 ºC. It was reported that response on day one was equivalent across mitragynine and control animals. However, reduced latencies representative of induced hyperalgesia were reported on day five. Additionally, the authors reported that the multi-day treatment with mitragynine resulted in "… minor withdrawal effects …" relative to controls, measured as decreased forepaw licking, mouse straightening, and rearing. Animals administered oral mitragynine also showed significantly increased diarrhea occurrence and forepaw tremor. In a related study, Hassan et al. (2019) reported that 5 mg/kg-bw or higher intravenous mitragynine administered to male Sprague-Dawley rats following escalating treatment with 10 to 50 mg/kg-bw morphine twice daily for six days significantly attenuated opioid withdrawal symptoms. Analyses reported by Wilson et al. (2021) for exposure to kratom alkaloid extract and mitragynine pseudoindoxyl are reported below. Collectively, these studies indicate that mitragynine yielded analgesic activity in test animals and that the doses given for multiple days yielded withdrawal effects once administration ceased. Such withdrawal effects are due to the compound's activity at opioid receptors, which is discussed more thoroughly elsewhere in this report.

219. Hill et al. (2022) explored the respiratory inhibition effects of mitragynine and 7-hydroxymitragynine, relative to morphine and the CYP3A enzyme inhibitor ketoconazole, in male CD-1 mice. Specific to mitragynine, animals were administered 3 to 90 mg/kg-bw mitragynine by oral gavage in 0.1 ml volume (dissolved in three molar equivalents of acetic acid and diluted in deionized water). Respiratory effects were evaluated with whole body plethysmography and antinociceptive effects were evaluated via hot plate test (52.5 ºC). The authors reported that 3 mg/kg-bw mitragynine yielded no effects on respiration, while 10 mg/kg-bw induced significant and prolonged respiratory depression. The respiratory effects of the 10 mg/kg-bw dose were not increased at 30 or 90 mg/kg-bw mitragynine, but pretreatment with ketoconazole significantly reduced respiratory depression at as low as 5.5 mg/kg-bw mitragynine. For antinociceptive effects, mitragynine administration induced significantly increased hot plate latency, though this effect was not observed in ketoconazole pretreated mice. The authors concluded that the modulation of ketoconazole on mitragynine's effects supported "… the hypothesis that the effects of mitragynine are likely to be mediated by its active metabolite [7-hydroxymitragynine]." This study indicated that higher doses of mitragynine (specifically doses above those experienced by typical kratom users) could inhibit respiration. Further, the fact that ketoconazole diminished these effects of mitragynine demonstrates that the CYP3A enzyme is at least in part responsible for mitragynine's metabolism into other compounds.

7-Hydroxymitragynine

220. In a follow-up to Matsumoto et al. (1996a), Matsumoto et al. (1996b) administered male ddY mice 2.5 to 10 mg/kg-bw 7-HMG orally or subcutaneously and subsequently examined outcomes via the hot plate (55 ºC) and tail flick tests. It was reported that dose-dependent antinociceptive effects of 7-HMG were observed in both tests, with MPE values peaking at 15 to 30 minutes post dosing for both routes of administration.

221. Similar to their abovementioned analysis of mitragynine, Kruegel et al. (2019) evaluated the antinociceptive effects of 7-HMG in 129S1 mice with hot plate and tail flick tests. An ED_{50} of 0.6 mg/kg-bw was reported in these mice following subcutaneous administration,

indicating higher direct therapeutic effect than subcutaneous mitragynine. The authors reported that 7-HMG's antinociceptive activity was significantly reduced when administered to μ-OR knockout mice and with naloxone pretreatment, though such effects were retained in κ-OR knockout mice and δ-OR knockout mice. They stated that findings were "… consistent with agonism of [μ-OR] as the primary mechanism of analgesia …" for both mitragynine and 7-HMG. At a basic level, these results highlight that 7-HMG contributes to the hallmark physiological effects of kratom (e.g., pain relief, easing of opioid withdrawal symptoms) more readily than does mitragynine. This is due to action at the above noted opioid receptors, since Kruegel et al. (2019) demonstrated that test animals lacking such receptors experienced few effects following 7-HMG administration.

222. As detailed above, Smith et al. (2019) evaluated the acute toxicity of 7-HMG in male Swiss Webster mice. Animals were intravenously administered 6.25, 12.5, 25, or 50 mg/kg-bw 7-HMG. The authors reported that additional animals were orally administered single doses of 6.25, 12.5, 25, or 50 mg/kg-bw 7-HMG in a study aimed at determining an oral LD_{50}, but that "… none of the mice expired at any of the doses." It was added that severe respiratory depression and seizures were observed among orally dosed animals, though dose-specific effects were not specified. For intravenous administration, the authors reported a 7-HMG LD_{50} of 24.7 mg/kg-bw. This LD_{50} indicates that 7-HMG is moderately toxic. However, the intravenous route of administration used by Smith et al. (2019) differs from the oral intake seen among typical kratom users. Moreover, since raw kratom contains <0.1% 7-HMG (by weight) and processed consumer derivatives rarely contain more than 1 mg 7-HMG, if not a fraction of an mg, the acute toxic effects noted in this study occur at doses hundreds-fold above what even high-end kratom users would experience assuming typical human weight.

223. In addition to their analysis of mitragynine, examined the independent effects of 7-HMG in male CD-1 mice. Animals were administered 3 to 30 mg/kg-bw 7-HMG via oral gavage, after which antinociceptive effects were evaluated with a hot plate test (52.5 ^{o}C) and respiratory depressant effects via whole body plethysmography. It was reported that 7-HMG dose-

dependently depressed animal respiration from lowest to highest dose. The authors further added that "[o]ral administration of calculated equi-effective doses for mitragynine (5.5 mg kg^{-1}), 7-OH mitragynine (1.9 mg kg^{-1}), and morphine (3.8 mg kg^{-1}) induced significant respiratory depression … maintained for the entire period of observation … AUC analysis of equi-effective doses did not highlight any significant difference in the overall degree of respiratory depression induced over the entire observation period." Significant increases in mouse hot plate latency were also reported as a sign of 7-HMG's antinociceptive effects. In contrast to mice treated with mitragynine, animals dosed with 7-HMG after pretreatment with ketoconazole still showed significant respiratory depression and antinociceptive action. The authors concluded that for "… mitragynine, the results presented … suggest that induction of the CYP3A metabolic pathway may increase the chance of toxicity being achieved with kratom, due to enhanced formation …" of 7-HMG. More simply, this study confirmed alongside many others that 7-HMG is a strong contributor to kratom's reported effects, and certainly a stronger contributor than mitragynine. Further, as noted by Hill et al. (2022) above, more rapid metabolism of mitragynine to 7-HMG (that is, either via induction of the CYP3A pathway or in persons with stronger expression of these enzymes) could predispose individual persons or animals to greater sensitivity to kratom.

Kratom Alkaloid Extracts

224. Reanmongkol et al. (2007) evaluated the effects of methanol and alkaloid extracts of *M. speciosa* leaves (expressed as mg/kg-bw for the total extract) on male Swiss mice and Wistar rats. The authors evaluated the LD_{50} via the up-down method from Bruce (1985) and additionally performed hot plate tests (at 55 °C with 15 minutes of post-exposure latency), tail flick tests, and locomotor activity tests at oral doses of 50, 100, 200 mg/kg-bw methanol extract or 5, 10, or 20 mg/kg-bw alkaloid extract. It was reported that the oral LD_{50} in mice for methanol extract was 4,900 mg/kg-bw and that for alkaloid extract was 173.20 mg/kg-bw. Additionally, the authors reported that all doses of methanol extract and the 20 mg/kg-bw dose of alkaloid extract increased pain response latency in the hot plate test in mice (i.e., had analgesic effects) and that no dose of methanol or alkaloid extract influenced tail flick activity (i.e., no demonstrated nociceptive effects)

or locomotor activity. These results indicate that kratom extracts, which are similar to traditional methods of preparation as teas, have low acute oral toxicity while alkaloid extracts have moderate acute oral toxicity under tested scenarios.

225. Dried leaves of *M. speciosa* collected in Malaysia were used to derive methanolic (6.0-8.0% mitragynine), aqueous (0.1-0.5% mitragynine), and total alkaloid (22.0-24.0% mitragynine) extracts by Azizi et al. (2010). The authors later dosed male Sprague-Dawley rats with 50, 100, and 200 mg/kg-bw/day for methanolic and aqueous extracts and 5, 10, and 20 mg/kg-bw/day total alkaloid extracts via oral gavage. Extracts were dissolved in cosolvent solution containing a 4:1:4 ratio of propylene glycol, Tween 50, and water, respectively, at a volume of 5 ml/kg-bw. The authors reported that, during a scoping study, treatment with 200 mg/kg-bw total alkaloid extract resulted in rat death. The authors reported that no test animals for the methanolic, aqueous, and alkaloid extract groups showed significant differences in liver weight relative to body weight at any dose. Similar to above, this indicates that alkaloid extracts have moderate acute oral toxicity under certain scenarios.

226. Harizal et al. (2010) prepared a kratom alkaloid methanol extract (1.6% mitragynine) from the leaves of dried *M. speciosa* collected in Malaysia. This was used to dose adult male Sprague-Dawley rats with a single dose of 100, 500, or 1,000 mg/kg-bw via oral gavage in a method adapted from OECD guidelines No. 407 and 420. Toxic concentrations of morphine were used as positive controls. It was reported that oral administration of 100 and 500 mg/kg-bw doses of alkaloid extract produced no significant adverse effects in the acute phase following administration, while animals dosed with 1,000 mg/kg-bw "… showed very low level of activity (slow movement) and rapid breathing which lasted 30 min after drug administration. No mortality was observed in … extract treated groups." Similarly, the authors reported no significant changes in body weight, food and water consumption, absolute and relative organ weight, or full blood count (FBC) relative to vehicle controls at 14 days post-exposure. Significant increases in blood pressure were noted for all dose groups relative to vehicle controls at 14 days, while animals in the

1,000 mg/kg-bw group demonstrated liver damage, including centrilobular necrosis, over the same period (Harizal et al. 2010).

227. As detailed above, Sabetghadam et al. (2013a) derived oral LD_{50}, ED_{50}, and TI values for mitragynine. The authors also derived such values for kratom alkaloid extract in male Swiss albino mice, with the LD_{50} for alkaloid extract derived via sequential doses from 175 to 2,000 mg/kg-bw. It was reported that the oral LD_{50} value for the alkaloid extract was 591.6 mg/kg-bw. This value places the acute toxicity in the range of "moderately toxic" according to the Sterner categorization, which was first published in 1949 (Hodge and Sterner 1949). The ED_{50} calculated based on the MPE from animal response on a 52 ºC hot plate at 15 minutes post-exposure was reported to be 194.4 mg/kg-bw. From this, the TI was reported to be 3:1 for kratom alkaloid extract, which indicated a higher safety risk with use of alkaloid extract than with mitragynine alone (TI = 21:1) (Sabetghadam et al. 2013a). The purpose of this study was to identify a dose that dulled the senses and tactile response of the animals. In other words, the hot plate study is intended to determine when an animal has ingested enough of the agent to be unable to readily recognize an "insult" to the body. This is similar to the lessened response of an inebriated person to being punched sharply in the arm due to the effects of alcohol. That person, for example, may wake up in the morning with a large bruise and inflammation yet not remember that it happened.

228. Kamble et al. (2021) administered lypholized kratom tea (LKT) or OPMS brand liquid kratom products to male Sprague-Dawley rats (n = 4 per product group) via oral gavage and examined subsequent toxic and pharmacokinetic parameters for alkaloids including mitragynine, 7-hydroxymitragynine, corynantheidine, speciogynine, speciociliatine, paynantheine, corynoxine, corynokine B, mitraphylline, ajmalicine, and isospeciofoline via UPLC-MS/MS of animal plasma. Doses of LKT and OPMS were normalized for mitragynine based on a human equivalent dose calculated based on body-weight methods published by the FDA. Specifically, animals were orally administered a weight appropriate dose of LKT or OPMS via a 4 ml/kg volume standardized solution of the kratom product and water. In total, animals were administered 366 mg/kg-bw of LKT and 0.8 ml/kg OPMS liquid, equating to human dose equivalents of 5.7 mg/kg-bw

mitragynine and 9.6 mg/kg-bw mitragynine, respectively. The authors reported that mitragynine absorption was reduced and systemic exposure increased with use of OPMS liquid relative to LKT preparation and that only mitragynine, 7-hydroxymitragynine, corynantheidine, and speciociliatine demonstrated measurable systemic exposures. No toxic effects were reported among test animals (Kamble et al. 2021). These results indicate that no adverse health effects were reported in test animals dosed with commercially prominent kratom products. Similar outcomes (that is, no toxic effects) are expected in typical human users of such products.

229. As discussed above, Wilson et al. (2021) administered kratom alkaloid extract to male C57BL/6J mice twice daily for four days with an escalating oral dose of 30 to 125 mg/kg-bw/day to evaluate antinociceptive effects with a warm-water tail-withdraw assay (48 ºC). No significant effects were reported for kratom alkaloid extract on day one, though significantly reduced tail-withdraw latency indicative of hyperalgesis was reported on day five. The kratom alkaloid extract was reported to produce "… minor withdrawal effects …" including decreased forepaw licking, mouse straightening, and rearing, though no other significant signs of opioid withdrawal were reported (Wilson et al. 2021).

230. Hughes et al. (2022) examined the effects of kratom alkaloid in extracts *Caenorhabditis elegans* nematodes. It is difficult to extrapolate effects in nematodes to humans, and these results are less informative than those reported in other mammals. Nonetheless, the authors reported that kratom extracts, along with individual alkaloids, dose-dependently decreased the brood size and parent worm health in analyzed nematodes, including at doses analogous to human tea consumption. However, the authors concluded that "[o]nly at very high doses did kratom cause developmental and genotoxic effects in nematodes, indicating its relative safety" (Hughes et al. 2022).

231. Hassan et al. (2023) administered male and female Sprague-Dawley rats (n = 5 per dose group) kratom decoction (cut fresh leaves boiled in water for four hours until the volume was reduced by two thirds) at approximately 10, 50, and 150 mg/kg-bw/day mitragynine (based on human dose equivalents extrapolated form body surface area and OECD 407 guidelines). Animals

were orally administered the decoction once daily for 28 days. No significant changes in body weight were reported at any dose. Relative to controls, females in the 10 mg/kg-bw/day group had significantly reduced blood platelet counts, with nonsignificant changes in platelet count among the 50 mg/kg-bw/day male group. Other hematological variables showed significant changes, but no consistent dose-response effects. Further, authors reported no significant difference in organ weight among any treatment groups apart from significant increases in liver weight in the male 150 mg/kg-bw/day group, female 50 and 150 mg/kg-bw/day groups, and satellite control animals. Based on their results, the authors concluded that "… kratom in the form of a brewed solution appears to be safe."

Mixed and Other Alkaloids

232. In a comparative study of nociceptive effects across kratom-associated alkaloids, Obeng et al. (2020) performed a hot plate test at 52 ºC based on rat (unspecified strain) latency to jump or lick/shake hind paws. Alkaloids were administered intravenously at doses of 0.32, 1.0, 3.2, or 10 mg/kg-bw. It was reported that 7-hydroxymitragynine and speciociliatine produced 100% of the maximum potential nociceptive effect (MPE), "… with 7-hydroxymitragynine being more potent than speciociliatine and morphine but less potent than fentanyl." Mitragynine was stated to have the least nociceptive efficacy among the examined alkaloids, which also included 7-hydroxymitragynine, speciociliatine, corynantheidine, 9-hydroxycorynantheidine. The authors stated that speciociliatine demonstrated antinociceptive effects at 10 mg/kg-bw, but that this dose resulted in fatality in approximately 25% of tested animals. When one compares the typical dose which humans administer to themselves during tea drinking, it would suggest that if humans were as susceptible to the acute toxicity of kratom-associated alkaloids, then drinking six cups of potent tea, or if there were a significant inhaled dose, this might pose an acute risk to the user.

233. The authors did not calculate a TI for the alkaloid speciociliatine due to the small differential between its ED_{50} and LD_{50}. Based on their results, which also included *in vitro* radioligand binding and functional assays, plasma and microsomal binding assays, and metabolic stability assays, Obeng et al. (2020) concluded that 7-hydroxymitragynine was a more potent

agonist at the μ-OR and κ-OR (kappa opioid receptor, or KOR) than mitragynine, while speciociliatine also acted as a mild agonist at the μ-OR. This is an interesting study in that the authors have identified that the ED_{50} and LD_{50} are not much different; thus, an indication of a steep dose-response curve. Such curves give toxicologists pause because small differences in dose can mean the difference between an analgesic effect and serious frank toxicity.

234. In addition to the above analyses for mitraygnine and kratom alkaloid extract, Wilson et al. (2021) administered mitragynine pseudoindoxyl to male C57BL/6J mice twice daily for four days with an escalating subcutaneous dose of 1 to 35 mg/kg-bw/day to evaluate antinociceptive effects with a warm-water tail-withdraw assay (48 ºC). No significant differences were reported for animals dosed with mitragynine pseudoindoxyl relative to controls, indicating a lack of induced hyperalgesia. In contrast, significant signs of opioid physical dependence were reported after repeated administration. These signs included forepaw licking and wet dog shakes. Diarrhea, jumping, and rearing frequency were also increased in animals administered escalating doses of mitragynine pseudoindoxyl, suggesting potential development of opioid physical dependence, though these signs were of smaller magnitude than those noted for escalating doses of morphine. The significance of this study is that this variation of the molecule, mitragynine pseudoindoxyl, if present in the compound which is ingested or inhaled, can have a large impact on the human response. This is important because these agents related to kratom apparently contain differing ingredients and concentrations depending on the source and processing method.

235. No significant effects were reported for kratom alkaloid extract on day one, though significantly reduced tail-withdraw latency indicative of hyperalgesia was reported on day five. The kratom alkaloid extract was reported to produce "… minor withdrawal effects …" including decreased forepaw licking, mouse straightening, and rearing, though no other significant signs of opioid withdrawal were reported (Wilson et al. 2021). As discussed previously, this study indicated that derivatives of mitragynine could contribute to withdrawal effects, primarily based on activity at opioid receptors, but that these effects were less severe than traditional opiates such as morphine.

Effects When Co-Administered with Other Compounds

236. Berthold et al. (2024) studied the effects of cannabidiol on subsequent mitragynine dosing in male Sprague-Dawley rats. In one scenario, animals were dosed orally with 0.8 ml/kg-bw OPMS Gold brand kratom 30 minutes after previous administration of 50 mg/kg-bw cannabidiol via oral gavage. In another scenario, animals were dosed with 0.4 ml/kg-bw of the OPMS gold solution 30 minutes after previous oral administration of 25 mg/kg-bw cannabidiol, with dosing repeated every 12 hours for four days. Health and pharmacokinetic parameters were measured via blood samples collected at different intervals across scenarios.

237. The authors reported that the OPMS Gold test product contained 11.8 mg/ml mitragynine (mean total dose = 9.4 mg/kg-bw), 2.8 mg/ml speciociliatine, 2.2 mg/ml paynantheine, and 1.5 mg/ml speciogynine. For these alkaloids and 7-hydroxymitragynine, pharmacokinetic parameters including C_{max}, T_{max}, and $AUC_{0\text{-}24h}$ were all significantly increased by pre-administration of cannabidiol. Specifically, C_{max} was increased by 2.3-fold, T_{max} was increased by 5.4-fold, and $AUC_{0\text{-}24h}$ was increased by 2.8-fold for mitragynine. Blood mitragynine:7-hydroxymitragynine ratios were not significantly altered in animals previously administered cannabidiol, despite an expectation that such pretreatment would impair metabolism of mitragynine due to competitive inhibition of CYP3A4 (Berthold et al. 2024). These results indicate the importance of co-administration of other compounds on kratom's toxicological effects.

Conclusions Based on the In Vivo Data

238. As discussed above, kratom and its alkaloids show beneficial effects, foremost pain relief (analgesia) and stimulant outcomes, at doses experienced by typical kratom users. These effects were associated most strongly with mitragynine and 7-hydroxymitragynine, including metabolism of the former compound into the latter compound following kratom intake. Generally, few adverse effects were observed across *in vivo* studies, though this is in part due to the majority of such literature focusing on potential beneficial effects and an associated lack of testing for adverse outcomes. Where studied, the adverse effects in animals (such as minor changes in organ-specific histopathology) may not be mirrored in humans, especially since the doses at which such effects occurred were generally higher than those experienced by typical kratom users.

239. Specific to acute outcomes, animal data showed that oral LD_{50} values for mitragynine and kratom alkaloid extracts were approximately 500-750 and 170-600 mg/kg-bw, respectively. Based on the methods of Hodge and Sterner (1949), this indicates that kratom is generally "slightly" to "moderately" acutely toxic, with the greatest concern associated with 7-hydroxymitragynine (which represents a very small component of the alkaloid content of most products). As noted elsewhere in my report, from a risk assessment perspective, the alkaloid concentrations in kratom products are sufficient to yield some beneficial effects but far too low to contribute to an increased risk of adverse health effects in typical users. This is confirmed by the above *in vivo* data.

In Vitro Studies Providing Information on Kratom Metabolism

240. Studies of isolated cells, termed in vitro analyses, help provide information on potential organ-specific effects and the toxicological mechanisms driving health outcomes following exposure to a given compound. In vitro data indicate that kratom alkaloids are primarily metabolized by Cytochrome P450 (CYP) isoforms CYP3A4 and CYP2D6 and that physiological effects are driven by alkaloid activity at opioid, adrenergic, serotonergic, and dopaminergic receptors. Kratom's pain relief (analgesic) and euphoric effects can be explained by kratom alkaloid binding at opioid receptors (ORs). However, it is my opinion that the activity of mitragynine, 7-hydroxymitragynine, and other kratom alkaloids at ORs is weak relative to that of numerous drugs (e.g., morphine, fentanyl) and endogenous endorphins (e.g., β-endorphin, enkephalins, dynorphins).

Basis of Opinion 5

241. *In vitro* studies of kratom and its alkaloids and alkaloid metabolites report that cytochrome P450 (CYP) 3A4 and other CYP isoforms (e.g., CYP2D6) are primarily responsible for the metabolism of this herbal product. The major alkaloids of kratom, mitragynine and paynantheine, are further metabolized by these CYP enzymes and other key drug-metabolizing enzymes that are discussed below.

Mitragynine

242. (Hanapi et al. 2013) reported on the metabolism of mitragynine by various CYP isoforms including CYP2D6, CYP2C9, and CYP3A4, which are all significant in metabolism of not only kratom alkaloids, but also other exogenous compounds. A high-throughput luminescence assay with the P450-Glo Screening System was implemented containing recombinant human enzymes CYP2D6, CYP2C9, and CYP3A4 at exposures of 0.02-200 μM for 30-45 minutes. The authors reported that mitragynine inhibitory concentration (IC_{50}) values of 0.45 ± 0.33 μM, 9.70 ± 4.80 μM, and 41.32 ± 6.74 μM for CYP2D6, CYP2C9, and CYP3A4, respectively. Substantial inhibition of CYP2D6 and moderate inhibition of CYP2C9 and CYP3A4 was observed (Hanapi et al. 2013).

243. Basiliere and Kerrigan (2020a) used recombinant CYP enzymes (rCYPs) to investigate isoforms involved in mitragynine metabolism. The authors compared phase I metabolites in 16 human urine samples collected postmortem for which there was known mitragynine consumption before fatality to the results from the rCYP *in vitro* studies. Each rCYP was incubated separately in the presence of 50 um of mitragynine for 0, 60, 120, 180 and 240 minutes. They confirmed that four rCYP enzymes (2C18, 2C19, 2D6, and 3A4) contributed to metabolism of mitragynine by liquid chromatography-quadrupole/time of flight-mass spectrometry and observed 7-hydroxymitragynine (7-HMG) to be produced exclusively by CYP3A4. Other papers have confirmed the inhibition activity of kratom alkaloids on CYP isoforms, with CYP3A4 demonstrated to have activity in subsequent metabolism of mitragynine to 7-hydroxymitragynine (Lim et al. 2013; Kruegel et al. 2019; Todd et al. 2020; Chakraborty et al. 2021; Tanna et al. 2023).

244. Kamble et al. (2020) exposed human liver microsomes to mitragynine, 7-hydroxymitragynine, speciogynine, speciociliatine, corynantheidine, or paynantheine and evaluated outcomes in CYP1A2, CYP2C8, CYP2C9, CYP2C19, CYP2D6, and CYP3A4/5. The most significant inhibition was reported for CYP2D6 with mitragynine (IC_{50} = 2.2 μM), corynantheidine (IC_{50} = 4.2 μM), and paynantheine (IC_{50} = 6.0 μM), with more significant effects reported for CYP3A4/5 using the midazolam 1'-hyroxylase reaction (7.3 to >45 μM) rather than

the testosterone 6β-hydroxylase reaction (>45 μM). For mitragynine, specific IC_{50} values were >45 μM (CYP1A2), 33.5 μM (CYP2C8), >45 μM (CYP2C9), 10.5 μM (CYP2C19), 2.2 μM (CYP2D6), 11.4 μM (CYP3A45 with midazolam 1'-hydroxylase reaction) and >45 μM (CYP3A4/5 with testosterone 6β-hydroxylase reaction) (Kamble et al. 2020).

Associated Kratom Alkaloids (Metabolism)

245. The metabolism of other kratom alkaloids (corynantheidine, speciogynine, speciocilialitine, mitraciliatine, and isopaynantheine) by CYP enzymes has been studied *in vitro* in hopes of understanding the varying effects of kratom consumption based on alkaloid content. Philipp et al. (2011b) determined the phase I and II metabolites of speciogynine, a diastereomer of mitragynine, in both rat and human urine samples after the administration of 40 mg/kg-bw doses of pure speciogynine in aqueous suspension by gastric intubation. Human urine samples of different patients were submitted to the authors' laboratory. The authors identified nine phase I and eight phase II metabolites in rat urine, as well as three phase I and five phase II metabolites in human urine. It was suggested that the varying results were due to the more uncontrolled setting in which the human samples were collected. Additional research on these metabolites may uncover different metabolite profiles in rats and humans. Further Philipp et al. (2011b) characterized metabolites mitraciliatine and isopaynantheine using liquid chromatography-linear ion trap-mass spectrometry to confirm that humans form isomeric compounds from the two diastereomers of mitragynine and paynantheine (Philipp et al. 2011b).

246. As discussed above specific to mitragynine, Kamble et al. (2020) exposed human liver microsomes to mitragynine, 7-hydroxymitragynine, speciogynine, speciociliatine, corynantheidine, or paynantheine and evaluated outcomes in CYP1A2, CYP2C8, CYP2C9, CYP2C19, CYP2D6, and CYP3A4/5. Across the examined kratom alkaloids, the authors reported that IC_{50} values were >45 μM for CYP1A2, 33.5 to >45 μM for CYP2C8, 15.7 to >45 μM for CYP2C9, 8 to >45 μM for CYP2C19, 2.2 to >45 μM for CYP2D6, and 7.3 to >45 μM for CYP3A4/5. The alkaloid-specific results of this study are summarized below in Table 5.

Table 5. Reported IC50 values for human CYP isoforms in human liver microsomes for mitragynine (MTG), speciogynine (SPG), speciociliatine (SPC), corynantheidine (COR), 7-hydroxymitragynine (7HMG), paynantheine (PAY). Redrawn from Kamble et al. (2019).

CYP450s	Reaction	IC_{50} (μM)					
		MTG	SPG	SPC	COR	7HMG	PAY
CYP1A2	Phenacetin *O*-deethylase	> 45	> 45	> 45	> 45	> 45	> 45
CYP2C8	Amodiaquine *N*-deethylase	33.5	> 45	36.6	> 45	> 45	> 45
CYP2C9	Diclofenac 4′-hydroxylase	> 45	> 45	> 45	27.7	> 45	15.7
CYP2C19	S-Mephenytoin 4′-hydroxylase	10.5	19.5	8	> 45	27.7	38.4
CYP2D6	Dextromethorphan *O*-demethylase	2.2	12.9	> 45	4.2	> 45	6.0
CYP3A4/5	Midazolam 1′-hydroxylase	11.4	13.5	26.4	7.3	> 45	7.9
CYP3A4/5	Testosterone 6β-hydroxylase	> 45	> 45	> 45	> 45	> 45	> 45

247. Kamble et al. (2022) examined the metabolism of speciociliatine and its pharmacological effects in human and preclinical species (mouse, rat, dog, and cynomolgus monkey) based on outcomes in liver microsomes and hepatocytes. The authors determined that speciociliatine was metabolized primarily through monooxidation and *O*-demethylation metabolic pathways in liver microsomes and hepatocytes across species (Kamble et al. 2022). The breakdown of speciociliatine was reported to be primarily regulated by CYP3A4 with minor contributions by CYP2D6. The authors also reported that speciociliatine had high permeability coefficients (P_{app}) in Caco-2 cells, with $P_{app\ (A->B)}$ and $P_{app\ (B->A)}$ values of 14.18 ± 0.98 and 22.90 ± 1.87 cm/sec with minimal to no efflux through the intestine based on the efflux ratio of 1.61. Put simply, this means that speciociliatine has good intestinal absorption in the tested cells but may be limited by active efflux mechanisms.

248. The authors concluded that speciociliatine exhibited appreciable metabolic stability in hepatocytes across species and the metabolite profiles of cynomolgus monkeys and/or dogs could be used as non-rodent species in toxicological evaluations of speciociliatine. It should be noted that as this is the first major study to examine speciociliatine as a metabolite of kratom and further studies into its pharmacology are warranted (Kamble et al. 2022).

Other Enzymes Important in Metabolism

249. Purified kratom alkaloids and methanolic extracts of kratom are reported to alter the activity of key drug metabolizing enzymes, such as P450s and UDP-glucuronosyltransferases (UGTs), that are responsible for xenobiotic elimination (Tanna et al. 2023). Mitragynine, specifically, has been found to inhibit several of the P450 enzymes in human liver microsomes, including CYP2C8, CYP2C9, CYP2C19, CYP2D6, and CYP3A (Tanna et al. 2023).

Other Enzymes Important in Metabolism

250. Haron and Ismail (2015) determined the effects of mitragynine, 7-hydroxymitragynine, ketamine, and buprenorphine concentrations on 4-methylumbelliferone glucuronidation, a major phase II drug metabolism reaction that is catalyzed by uridine 5'-diphospho-gluconosyltransferase, in rat and human liver microsomes and recombinant human UGT isoforms (UGT1A1 and UGT2B7). Their findings suggested a possibility of drug-drug interactions during co-administration of 7-hydroxymitragynine, ketamine, and buprenorphine with other drugs that are UGT2B7 substrates (Haron and Ismail 2015). This is due to the observed inhibition of UGT2B7 activity by 7-HMG, ketamine, and buprenorphine. The authors reported that 7-HMG may also pose a drug-drug interaction risk if taken with other drugs that are highly metabolized by UGT1A1 as it strongly inhibited 4-MU glucuronidation with an IC_{50} value of 7.13 $\pm$ 1.16 µM (Haron and Ismail 2015).

Other Enzymes Important in Metabolism

251. More recently, Melchert et al. (2023) examined the effects of mitragynine and other kratom alkaloids *in vitro* on the hepatic hydrolase carboxylesterase 1 (CES1). The authors reported that mitragynine, speciocialitine, paynantheine, and corynantheidine displayed K_i values for CES1 of 20.6, 8.6, 26.1, and 12.5 µM, respectively. The authors concluded that a drug-drug interaction mediated via CES1 inhibition appeared unlikely at doses ranging from 2 to 20 grams of various kratom alkaloids per dose, based on the pharmacokinetic data, determined Ki values, and a physiologically based inhibition screen mimicking alkaloid exposures in humans (Melchert et al. 2023).

Opioid and Non-Opioid Receptor Activity of Kratom In Vitro

252. Published *in vitro* studies of kratom, kratom alkaloids and their metabolites largely focused on receptor binding, primarily at opioid receptors, as well as a selection of non-opioid receptors. Prior authors have reviewed such *in vitro* receptor activity (Anand and Hosanagar 2022; Hiranita et al. 2022; Tanna et al. 2023). Other *in vitro* studies examined cellular outcomes including cytotoxicity, mutagenicity, cardiotoxicity, and neurotoxicity (Saidin et al. 2015; Kong et al. 2017a; Tay et al. 2019; Leong Bin Abdullah and Singh 2021a). Generalizing effects seen in *in vitro* analyses to effects or harms in mammalian and human systems is difficult to impossible. However, *in vitro* analyses can inform subsequent *in vivo*, human, or other study of the therapeutic, toxic, and other effects of kratom and its alkaloids.

253. Specific to the study of receptor binding affinities, available *in vitro* data reveal that future study of the potential interaction of kratom, its alkaloids, and its alkaloid metabolites with pharmaceuticals and other xenobiotics (e.g., alcohol, illicit drugs) among users (i.e., among polysubstance users) is needed.

Opioid Receptor Activity

254. Todd et al. (2020) examined the chemical composition and biological effects of speciofoline, mitragynine, and 7-hydroxymitragynine by conducting assays using Chinese hamster ovary cells (CHO-K1), human embryonic kidney cells (HEK293), and human liver microsomes. The authors reported that speciofoline did not exhibit measurable binding affinity for the μ-, δ-, or ƙ- opioid receptors. All three examined kratom alkaloids inhibited select cytochrome P450 enzymes and the authors suggested "… a potential risk for adverse interactions when kratom is co-consumed with drugs metabolized by these enzymes" (Todd et al. 2020). Similar to findings by Todd and colleagues (2020), Vandeputte et al. (2020) reported on the biological activity at the μOR in HEK293 cells and noted that the EC_{50} for mitragynine's activity on β-arrestin 2 and G-protein signaling were 773 (513-1,148) nM and 1,089 (280-4,442) nM, respectively. Havemann-Reinecke (2020) reported that kratom powder had a 350-fold lower affinity to the μOR than morphine in a radioligand binding assay with HEK293 cells.

255. Obeng et al. (2020) evaluated the opioid binding affinities (K_i) of kratom alkaloids and reported values for mitragynine (μOR = 161 ± 10 nM, ƙOR = 198 ± 30 nM), 7-hydroxymitragynine (μOR = 7.16 ± 0.94 nM, ƙOR = 74.1 ± 7.8 nM, δOR = 236 ± 6 nM), speciociliatine (μOR = 54.5 ± 4.4 nM, ƙOR = 116 ± 36 nM), corynantheidine (μOR = 118 ± 12 nM, ƙOR = 1,910 ± 50 nM), and 9-hydroxycorynantheidine (μOR = 105 ± 1 nM). This indicates that mitragynine has a lower K_i than tramadol (μOR = 12,486 nM) and codeine (μOR = 734.2 nM) for the μOR, but higher K_i than drugs such as oxycodone (μOR = 25.87 nM), fentanyl (μOR = 1.346 nM), morphine (μOR = 1.168 nM), and buprenorphine (μOR = 0.2157 nM) (Volpe et al. 2011). Binding activity for kratom alkaloids was also recently reported by Ellis et al. (2020) *in silico* and with subsequent *in vitro* validation. While mitragynine's activity at ORs prospectively drives most of its effects on mood, this adrenergic, dopaminergic, and serotonergic binding also contributes to such mood modulation.

256. In a follow up, Obeng et al. (2021) concluded that the μOR binding affinity of mitragynine was lower than that of 7-hydroxymitragynine. Similar results were reported by Chakraborty et al. (2021) and Chear et al. (2021) with additional K_i values reported in the latter study for corynoxine (μOR = 16.4 ± 1.4 nM), corynoxine B (μOR = 109.8 ± 8.1 nM), mitragynine oxindole B (μOR = 1,715.9 ± 12.0 nM), and speciogynine (μOR = 728 ± 61.0 nM). There is *in vitro* evidence that mitragynine concentrations decrease congruent to increases in 7-hydroxymitragynine concentrations, potentially making the OR effects of the latter compound more biologically relevant over time (i.e., time since dosing) than those of the former Kruegel et al. (2019). In other words, 7-hydroxymitragynine is binding more tenaciously to the opioid receptors and thus may have potential to help persons attempting to cease use of traditional opioids.

Non-Opioid Receptor Activity

257. Anand and Hosanagar (2022) examined non-opioid receptor activation by mitragynine and reported that such receptors included alpha-2 adrenergic, D2 dopamine, and serotonin 5-HT2C and 5-HT7. Other authors reported that mitragynine exhibited partial agonism at $\alpha_{1A,D}$ receptors, and competitive antagonism at $\alpha_{1A,B,D,2C}$ receptors (Obeng et al. 2020). (Leon et

al. 2021) reported speciogynine and paynantheine demonstrated high binding affinity to 5-HT1A and 5-HT2B receptors relative to mitragynine. Collectively, the serotonergic and dopaminergic receptor activity reported for kratom alkaloids contribute to the modulatory effects on mood noted by kratom users and thereby may drive popular attraction to the product.

258. Obeng et al. (2020) reported binding affinities (K_i) for mitragynine at receptors including α-$_{1A}$ (1,340 ± 100 nM), α-$_{1B}$ (4,770 ± 120 nM), α-$_{1D}$ (5,480 ± 540 nM), α-$_{2A}$ (4,720 ± 120 nM), α-$_{2B}$ (9,290 ± 30 nM), and α-$_{2C}$ (2,320 ± 140 nM). Analysis of non-opioid receptor activation by mitragynine and 7-HMG include alpha-2 adrenergic, D2 dopamine, and serotonine 5-HT_{2C} and 5-HT_7 (Boyer et al. 2008). Leon et al. (2021) reported that mitragynine exhibited low binding affinity to serotonergic receptors, while speciogynine and paynantheine demonstrated high binding affinity to serotonergic 5-HT_{1A} and 5-HT_{2B} receptors. Other authors have evaluated the receptor binding of kratom alkaloids in more detail (Ellis et al. 2020; Mukhopadhyay et al. 2023). The receptor and CYP activity of mitragynine and 7-hydroxymitragynine are compared to such activity for morphine, fentanyl, and tianeptine (an antidepressant) are presented below in Table 6

Table 6. Receptor and CYP activity for mitragynine, 7-hydroxymitragynine, morphine, fentanyl, and tianeptine.

		Mitragynine	**7-Hydroxy mitragynine**	**Morphine**	**Fentanyl**	**Tianeptine**	**Studies**
Opioid Receptor (OR)	μOR	K_i = 161-238 nM	K_i = 7.16-16 nM	K_i = 0.58-1.50 nM	K_i = 0.35-1.346 nM	K_i = 383 nM	Maguire et al. (1992); Volpe et al. (2011); Obeng et al. (2020); Todd et al. (2020); Torralva et al. (2020); Charoenratana et al. (2021); Chear et al. (2021)
		Partial Agonist	Partial Agonist	Agonist	Agonist	Agonist	
	κOR	K_i = 198-482 nM[a]	K_i = 74.1-113 nM	K_i = 27-48.9 nM	K_i = 125-290 nM		Maguire et al. (1992); Gharagozlou et al. (2006); Obeng et al. (2020); Todd et al. (2020); Torralva et al. (2020)
	δOR	K_i = >10,000 nM[a]	K_i = 236-137 nM	K_i = 178->10,000 nM	K_i = 180-244 nM	K_i = 10 μM	Maguire et al. (1992); Obeng et al. (2020); Todd et al. (2020); Torralva et al. (2020)
Adrenergic	α-1A	K_i = 1,340 ± 100 nM		K_i = >100,000 nM	K_i = 1,407 ± 81 nM		Obeng et al. (2020); Torralva et al. (2020)
	α-1B	K_i = 4,770 ± 120 nM		K_i = >100,000 nM	K_i = 1,100 ± 250 nM		
	α-1D	K_i = 5,480 ± 540 nM		K_i = >100,000 nM	K_i = 4,110 ± 450 nM		

Serotonergic	5-HT_{1A}	K_i = 5,880 ± 828		K_i = >100,000 nM	K_i = 2,240 ± 240 nM		Torralva et al. (2020); Leon et al. (2021)
	5-HT_{2A}	K_i = 5,010 ± 1,150 nM		K_i = >100,000 nM	K_i = 31,400 ± 3,000 nM		
	5-HT_{2B}	K_i = 1,260 ± 138 nM		K_i = >100,000 nM	K_i = 8,600 ± 1,200 nM		
	5-HT_{2C}	K_i = >10,000 nM		K_i = >100,000 nM	K_i = 114,000 ± 23,000		
Dopaminergic	D2 class	% Inhibition = 54.22		K_i = >100,000 nM	K_i = 14,900 ± 1,800 nM		Boyer et al. (2008); Torralva et al. (2020)
Cytochrome P450 (CYP)	CYP 3A4	K_i = 379,180 nM					Hanapi et al. (2013); Kamble et al. (2020)
		IC_{50} = 11,400->45,000 nM					
	CYP 2D6	K_i = 12,860 nM IC_{50} = 450-2,200 nM					Hanapi et al. (2013); Kamble et al. (2020)

K_i, receptor binding affinity; IC_{50}, median inhibitory concentration

[a]Mitragynine is a competitive antagonist at the κOR and δOR.

Cytotoxicity

259. Saidin et al. (2015) reported that human neuronal cells (SH-SY5Y), embryonic kidney cells (HEK293), and lymphoblastoid cells (MCL-5) showed a time- and dose-dependent decrease in viability following mitragynine exposure in a trypan blue exclusion assay. The authors reported that IC_{50} concentrations for mitragynine were 75, 240, and 80 μM for SH-SY5Y, HEK293, and MCL-5 cells, respectively (Saidin et al. 2015). In MCL-5 cells, chemical inhibitors

indicated that both CYP2E1 and CYP2A6 were involved in cytotoxicity and that this was preceded by cell cycle arrest at the G1 and S phase (Saidin et al. 2015). The authors determined that when coupled with naloxone, the cytotoxicity of mitragynine was partially reduced, suggesting that cytotoxicity partially depends on opioid signaling.

260. Oliveira et al. (2016) examined the cytotoxicity of kratom products (resins, leaf extracts, powdered extracts) using human intestinal (Caco-2) and neuronal (SH-SY5Y) cell lines and an NR incorporation assay (lysosomal membrane integrity assay). Cytotoxicity tests revealed a concentration-dependent decrease in cell viability in both cellular models, with the SH-SY5Y cells being more sensitive to kratom extracts. The authors concluded that "… the cytotoxic effects observed were not only produced by [mitragynine] but rather by other unknown substances present in Kratom products or by interactions among them."

261. Domnic et al. (2021) examined the potential of mitragynine and speciociliatine as chemosensitizers for cisplatin on nasopharyngeal carcinoma (NPC) cells, which can develop resistance against cisplatin in chemotherapy patients over time. This can decrease the effectiveness of the chemotherapeutic regimen to treat targeted cancers. Initial single agent treatment with mitragynine, speciociliatine, paynantheine, and speciogynine was reported to cause weak to moderate inhibition of NPC cell line migration. The authors reported that mitragynine and speciociliatine sensitized NPC cell lines HK-1 and C666-1 to cisplatin, approximately 4- and 5-fold, respectively, via analysis with a 2D monolayer culture. Significantly, this study indicated that kratom and its alkaloids may be potential agents for combination cancer therapy, reducing the doses of chemotherapeutics needed to derive the same effects. Since kratom and mitragynine are not expected to yield adverse outcomes at the chemo effective doses, but could reduce the adverse side effects of cisplatin since lower doses of this drug are required to yield the intended therapeutic endpoints, such results are pharmacologically quite significant.

262. Cytotoxic effects of mitragynine after accelerated solvent extraction (ASE) of M. *speciosa* using HEK293 and HeLa Chang liver cells was examined by Goh et al. (2021). The authors reported a dose-dependent decrease in cell viability observed across both kidney and liver

cell lines. They added that bioactive indole alkaloids and flavonoids mitragynine, paynantheine, quercetin, and rutin were detected in ASE kratom ethanolic extract using UHPLC-ESI-QTOF-MS/MS analysis.

263. While it is difficult to impossible to translate *in vitro* cytotoxicity effects to potential outcomes in humans, this evidence implies that the receptors targeted by kratom are similar to those of many illicit drugs and pharmaceuticals. This has relevance in circumstances of dual- or polydrug use in which binding of one compound, such as kratom, to a receptor could decrease the rate at which other compounds, such as pharmaceuticals or illicit drugs, can bind to these same receptors and subsequently undergo metabolism. The binding by kratom would then decrease the dose at which these other compounds may yield adverse health effects, even though kratom itself is not expected to yield adverse effects under normal dosing procedure.

Mutagenicity and Genotoxicity

264. Saidin et al. (2015) examined DNA damage with a mouse lymphoma thymidine kinase gene mutation assay in L5178 cells. They reported that mitragynine concentrations of below 75 µM did not yield significant DNA damage, with and without metabolic activation at three and 24 hours, respectively. The authors concluded that "[c]ytotoxicity … was substantial at the highest dose [75 µM] … there was a small dose dependent increase in the mutant frequency, but still within the [global evaluation factor] and thus the compound was not considered to be mutagenic."

265. Oliveira et al. (2016) also referenced above, reported that concentration-dependent DNA damage occurred in colorectal adenocarcinoma (Caco-2) cells after exposure to powdered kratom products but not to pure mitragynine. The results were based on the comet assay and analysis 24 hours following exposure. Further, the authors reported that DNA damage occurred at extract concentrations of 2.5 to 5 µg/ml but that such effects were similar despite significant variability in mitragynine content across the extracts (e.g., one extract had 187 mg/g mitragynine, another had 0.768 mg/g). This led them to conclude that other substances may be present in the extracts causing DNA damage, or that interactions between mitragynine and other alkaloids were causing the effect. The Cac-2 cells that these authors used in the comet assay were derived from a

cancer cell line and likely do not not accurately represent normal, healthy cells. The DNA repair mechanisms and responses to DNA damage of these cells may differ from those of non-cancerous cells. Accordingly, results obtained using Caco-2 cells might not be directly extrapolated to other cell types or tissues, limiting the generalizability of these findings

Cardiotoxicity

266. Studies have reported that mitragynine intake may induce dose-dependent prolonged QTc intervals (Leong Bin Abdullah and Singh 2021a). It was reported that in human induced pluripotent stem cell-derived cardiomyocytes (hERG-HEK293), mitragynine and its analogues (paynantheine, speciogynine, and speciociliatine) suppressed the rapid delayed rectifier potassium current by 67% to approximately 84% at doses of 0.1 to 100 μM, with an IC_{50} of 0.91 to 2.47 μM (Lu et al. 2014). At 10 μM, mitragynine was reported to significantly prolong action potential duration at 50% and 90% repolarization, and induced arrhythmia without altering the L-type Ca^{2+} current (Lu et al. 2014). Lu et al. (2014) did not provide sufficient blood concentration data to predict a typical human dose at which cardiotoxicity may occur.

267. Cardiotoxicity was also reported with HEK293-hERG1a/1b recombinant cells, where mitragynine was shown to inhibit the rapid delayed rectifier potassium current with an IC_{50} of 332.70 nM (Tay et al. 2019). Mitragynine was reported to cause a significant reduction of fully glycosylated hERG1a protein expression, but upregulated both core-glycosylated expression and hERG1a-Hsp09 complexes (Tay et al. 2019). In essence, the authors concluded that kratom alkaloids inhibited hERG1A/1B current at low concentrations but upregulated the hERG1a-Hsp90 complex at higher concentrations (Tay et al. 2019). Such *in vitro* effects have not been demonstrated *in vivo* or in humans and would likely have little clinical significance but could potentially translate to cardiac irregularity at certain high doses of mitragynine.

Neurotoxicity

268. Matsumoto et al. (2005) reported that 1 μM mitragynine inhibited neurotransmitter release from the vas deferens nerve endings of mouse neuroblastoma cells (N1E-115), partly through the blocking of Ca^{2+} channel currents. The authors proposed that the Ca^{2+} channel-block

effect was responsible for analgesic and other physiological actions of mitragynine (Matsumoto et al. 2005). This study was a preliminary investigation focused on hypothesis testing and these results should be viewed as potentially explanatory of kratom's analgesic action. That is, the reported effects were mechanistic in nature and did not indicate potential for more general neurotoxicity.

Mitragynine Is Unlikely to Yield Significant Respiratory Depression

269. As summarized by Tabanelli et al. (2023), 7-hydroxymitragynine "… and mitragynine are G-protein-biased agonists of μ-opioid receptors that do not recruit β-arrestin after receptor activation, which has been linked to many undesirable effects of opioids, including constipation, respiratory depression, and dependence." Similarly, Todd et al. (2020) suggested that mitragynine and 7-hydroxymitragynine were partial agonists of the human μ-opioid receptor (μOR), demonstrated functional selectivity for G-protein signaling, and did not measurably recruit β-arrestin in CHO cells. Tanna et al. (2023) reported both mitragynine and 7-hydroxymitragynine selectively bound to the G-protein-coupled receptor to activate the G-protein second messenger, rather than the β-arrestin-2 signaling pathway, which has been shown to be responsible for many of the adverse effects associated with traditional opioids.

270. Varadi et al. (2016) examined pharmacology and structure-activity relationship (SAR) studies both *in vitro* and *in vivo* of mitragynine pseudoindoxyl as a rearrangement product of the corynanthe isomer of mitragynine (corynantheidine). Initial extraction of mitragynine from powdered kratom leaves was further metabolized to corynantheidine and other kratom alkaloids and chemical modification of corynantheidine to produce mitragynine pseudoindoxyl as a therapeutic analog for pain relief. The authors attributed the physiological effects of mitragynine and mitragynine pseudoindoxyl to signaling via μOR agonism and δOR antagonism without canonical β-arrestin 2 signaling, which was noted to reduce the potential for unwanted side effects (such as those of opioids that do activate the β-arrestin pathway and thereby yield respiratory depression).

271. Kruegel et al. (2016) examined mitragynine's *vitro* activity using a human embryonic kidney (HEK293) cell line expressing human opioid receptors. The authors reported that mitragynine was a partial agonist at the μOR in HEK293 cells, as well as competitive antagonists at κ- and δORs Kruegel et al. (2016). More specifically, the authors reported that mitragynine and 7-hydroxymitragynine were G-protein-biased agonists at the μOR that did not recruit β-arrestin following receptor activation. They concluded that molecular docking studies in combination with the observed chemical structure-activity relationship (SAR) suggested that kratom alkaloids had treatment potential for mood disorders.

272. The use of mitragynine and 7-hydroxymitragynine in treating diseases such as cancer and non-cancer pain may present with low to no abuse potential (Tabanelli et al. 2023). Prior administration of mitragynine is reported to decrease subsequent morphine consumption, whereas prior treatment with extracted 7-hydroxymitragynine is reported to increase subsequent morphine consumption. Specifically, studies indicate that "… mitragynine did not lead to dependence or increased self-administration in animal models …" while "… a dependence liability of [7-hydroxymitragynine] … which is ascribable to its reinforcing effects, mediated in part by μ- and δ-opioid receptors" (Tabanelli et al. 2023).

Comparison to Endogenous Endorphins

273. Similar to some kratom alkaloids, endogenous endorphin such as β-endorphin, enkephalins, and dynorphins have substantial binding affinity to ORs. By definition, these compounds are produced naturally within the human body, often in response to pain, stress, diet, or exercise. The receptor effects of such endorphins are similar to, and in some cases stronger than, those of kratom alkaloids. This means that the effects of kratom alkaloids are not outside those associated with compounds every human produces endogenously daily, highlighting kratom's relative safety.

274. Kosterlitz et al. (1989) and Reisine (1995) reported OR binding affinities for several endogenous endorphins. In the former study, the authors reported OR-selective binding affinities in excised tissues, specifically in homogenates of guinea pig brain. For β-endorphin, which is

selective for the μOR, they reported a K_i value of 0.49 nM for the μOR and relative affinities of 0.52 nM, 0.45 nM, and 0.03 nM for the μ-, δ, and κORs, respectively. For dynorphins A and B, which are selective for the κOR, they reported K_i values of 8.5-8.7 nM for the κOR and relative affinities of 0.13-0.14 nM, 0.03-0.04 nM, and 0.83 nM for the μ-, δ, and κORs, respectively (Kosterlitz et al. 1989). In the Reisine (1995) study, the author reported K_i values for several endogenous endorphins, including met-enkephalin (μOR = 0.65 nM, κOR = >1,000 nM, δOR = 1.7 nM), β-endorphin (μOR = 1.0 nM, κOR = 52 nM, δOR = 1.0 nM), and dynorphin A (μOR = 32 nM, κOR = 0.5 nM, δOR = >1,000 nM).

275. The above data show that the OR binding affinities of mitragynine are several-fold lower than those of endogenous endorphins. The OR binding affinities of 7-hydroxymitragynine fit within the range of those expected for endogenous endorphins. Collectively, this information demonstrates that the OR activity of kratom and its alkaloids is no greater than such activity of compounds produced daily by virtually every person. Thus, the alkaloids produce effects within the range of biological norms and do not increase the risk of adverse, OR-associated adverse health effects among typical users. This is especially true given the aforementioned lack of β-arrestin signaling activation seen with exposure to kratom alkaloids, which minimizes potential adverse effects relative to most opioids (natural or synthetic).

Basis of Opinion 6

Epidemiology of Human Kratom Use

276. Data on the effects on the effects of kratom use in humans includes epidemiological evidence, as well as information from case reports and case series. Human studies indicate that kratom use is associated with potential benefits including stimulant effects, pain relief, and as a substitute or withdrawal aid for opioids at doses yielding few to no adverse behavioral or physiological effects. Case data indicate that kratom's alkaloids produce few to no acute adverse health effects when kratom is used alone, but that mitragynine and 7-hydroxymitragynine have been reported along with other compounds in the post-mortem blood and tissues of dual- and polysubstance users.

277. Epidemiological and clinical studies have been conducted to assess kratom-related health outcomes in humans. The endpoints investigated take into consideration the effects of kratom on several organs and bodily systems. It has been well documented that kratom users choose to ingest the substance for a number of reasons, but often for antidepressant, antipsychotic, and pain relief along with the potential to limit opioid withdrawal symptoms (Edinoff et al. 2024).

278. The studies summarized below generally employed small sample sizes and convenience or snowball sampling (i.e., recruiting easily accessible subjects, not persons representative of the general population), limiting their conclusions' strength and generalizability. Inherent bias exists in study designs that rely on self-reported information and designs that employ surveys or interviews (social desirability bias), as the topic of interest carries with it some level of stigma. Nonetheless, kratom studies involving humans, like studies evaluating the effects of e-cigarettes, pose certain ethical and practical limitations, which helps explain why the available studies have such limitations.

279. The following summarized studies reflect what is currently known about the effects of oral use of kratom in humans, but more research with sound methodologies is needed to assess all effects fully.

Evidence of Therapeutic Effects

280. The first known clinical study evaluating mitragynine in humans was conducted by Grewal (1932a). The authors recruited five men to participate in an intervention study to assess the effects of kratom on muscular and mental fatigue. Effects experienced by the participants following oral administration of 50 mg of mitragynine acetate included muscle tension, a reduction in heat sensitivity, improvement in muscular work, increased steadiness, and no effect on blood vessels or range of vision. When 650 or 1,300 mg of powdered leaves were orally administered, it was reported that the participants experienced symptoms like that of 50 mg mitragynine acetate, specifically, a very slight change in heat sensitivity and improvement in muscular work, along with dilatation of the blood vessels (Grewal 1932a; Prevete et al. 2022). The significance of this study was that, for the authors, it reinforced historical thinking that mitragynine could have the

dual benefit of serving as a compound that reduced inflammation and the associated body aches, that it served as a stimulant to promote more physical work with apparently equal effort, and that it had no other adverse effects. In short, it appeared to be a remarkable compound for the worker and the manager.

281. Grewal (1932a) and Prevete et al. (2022) also investigated the effects of mitragynine on mental fatigue in his intervention study, discussed above. It was reported that 50 mg of mitragynine acetate elicited giddiness, haziness, and slight mental fatigue. It was reported that following a 650 or 1,300 mg oral dose of powdered leaves, participants experienced the same effects as those following 50 mg mitragynine as well as an increase in reaction time. In short, the compound seemed to relieve emotional stress at appropriate doses.

282. Vicknasingam et al. (2020) conducted a randomized placebo-controlled, double-blind study to evaluate the effect of kratom on pain tolerance. All participants orally consumed a drink three times over the course of a day. Twenty subjects received a kratom drink (K) or placebo (P) in a randomized and blinded order (KPP, PKP, PPK) and six subjects received three rounds of the placebo only. Each participant was put through a cold pressor task (hand placement into a 2.5 ^{o}C ice bath) to evaluate pain tolerance one hour after consuming their assigned drink. Mitragynine content of administered kratom decoctions was examined with HPLC-UV. The authors found that pain tolerance increased one hour after kratom intake (Vicknasingam et al. 2020; Prevete et al. 2022). Prevete et al. (2023) conducted a review of human kratom studies published within the past five years to investigate the clinical implications of kratom use and stated that the evidence "… supported kratom's therapeutic value as an analgesic … [and] its therapeutic benefit in managing SUD symptoms (e.g., craving) and long-term or acute symptoms (e.g., withdrawal) for alcohol, opioids, and other illicit drugs." These studies indicated two things. First, kratom intake was reported to increase pain tolerance in humans. Second, at certain doses, kratom may have clinical utility in treating substance use disorders or as a means of easing withdrawal symptoms.

283. Saref et al. (2019) and Saref et al. (2020) conducted a series of cross-sectional surveys among illicit drug users with current kratom use to determine the associations between

kratom use and specific behaviors. The authors recruited 293 regular Malay kratom users and employed the Addiction Severity Index. The authors did not find any impairments in social functioning. Both the 2019 (N = 163) and 2020 (N=260) studies assessed kratom use in relation to illicit drug use through interviews and questionnaires. It was reported that kratom use reduced regular use of illicit drugs as well as HIV risk behaviors. Each study suggested kratom had therapeutic potential as a harm reduction tool for opioid addicts. This study suggested that kratom, in some users and if the correct dose was selected, could yield analgesic benefits and help users discontinue use of more dangerous substances, such as traditional opioids (prescription or otherwise). However, given the methodology, it is difficult to determine if the studies by Saref et al. (2019); Saref et al. (2020) demonstrated causality, or if a factor outside of kratom use also influenced outcomes. Prevete et al. (2023) similarly concluded that preliminary clinical data indicated kratom had therapeutic potential as a harm-reduction tool for regular drug users.

284. La-Up et al. (2021) performed blood exams and collected fasting lipid profiles of 285 regular kratom users and 296 controls in Thailand. Potential confounders including BMI, alcohol intake, and cigarette smoking status were controlled for within the study. The authors reported an association between kratom use and elevated HDL (high density lipoprotein) and low TG (triglyceride) levels, and no difference in TC (serum cholesterol) or LDL (low density lipoprotein) levels between kratom users and controls. As is commonly known, HDL is the "good" cholesterol, and a reduction in triglycerides is generally considered a positive effect. Thus, while the real-world relevance of these results is equivocal, the outcome is if anything indicative of a positive effect of kratom use on human blood parameters.

285. Leong Bin Abdullah et al. (2020) conducted an analytical cross-sectional study to determine the fasting lipid profiles among 100 kratom users and 100 controls in Malaysia. It was reported that there were no differences in serum TG and HDL levels among users and controls and that kratom users had lower serum TG and LDL levels than controls. It was additionally reported that there was a slight increase in serum lipids among kratom users who had higher frequencies of daily use.

286. Ramachandram et al. (2023) recruited five regular Malay kratom users to participate in a three-month longitudinal study. Participant blood and vital signs were tested at baseline and after three months. The study had participants orally consume kratom tea/juice packets, which were tested via GC-MS to determine mitragynine content. It was reported that there were no differences between baseline and follow-up blood or vital sign measurements. Participants were reported to consume an average of 1.3 to 1.6 mg/kg-bw mitragynine per day for the study duration. After three months, calcium, total protein, globulin, HDL levels increased, TG levels decreased, and TD and LDL levels remained high relative to clinically defined norms. However, the authors concluded that "… continuous consumption of kratom decoction at a daily mitragynine dose of 76.3-114.8 mg did not appear to alter the safety hematological and biochemical parameters of kratom users."

287. Singh et al. (2018b) performed blood exams on 58 Malay kratom users and 19 controls. The authors reported that kratom users had RBC (red blood cell), hemoglobin, and WBC (white blood cell) counts within normal ranges, elevated HDL cholesterol, and slightly elevated LDL cholesterol levels relative to controls. Effect sizes were linked to duration and quantity of daily kratom use. These effects, while minor, are still not clearly attributable to kratom use, given the significant number of confounding risk factors regarding elevated cholesterol levels.

288. La-Up et al. (2022) conducted blood exams and collected vital signs from 285 regular kratom from 14.7 to 380.7 ng/mL, HDL levels were elevated, and TG levels were decreased. The authors reported that kratom users had higher diastolic blood pressure compared to controls, but no difference in systolic. Analysis of metabolic syndrome (MetS) parameters revealed kratom users had lower odds of MetS, but it was unclear what mechanism drove this protective effect.

289. Prevete et al. (2023) suggested that La-Up et al. (2021), Leong Bin Abdullah et al. (2020), Ramachandram et al. (2023), Singh et al. (2018b), and La-Up et al. (2022) indicated kratom had the therapeutic potential to increase HDL, lower TG, and be protective against MetS.

Collectively, such effects could procure health benefits to users that employ traditional kratom practices.

Potential for Harm Reduction

290. The leaves of *M. speciosa* were first described as a substitute for opium in the early 1800s (Hassan et al. 2013). Prevete et al. (2023) reported that "[k]ratom … has ... been used as a substitute in nonmedical settings for dangerous drugs (I.e., methamphetamine, benzodiazepines, or heroin) or to mitigate the withdrawal from opioids, alcohol, and other illicit substances, especially among opioid poly-drug users." Preclinical evidence has suggested that kratom use has the potential to treat and or mitigate alcohol use disorder, withdrawal, and seeking behavior; dependence and withdrawal from opioids; and craving and addiction from methamphetamine (Prevete et al. 2023). Additional preliminary clinical data demonstrated the harm reduction potential of kratom use, as it reduced regular and illicit drug use, opioid adverse effects, and HIV risk behaviors among opiate and illicit drug users (Prevete et al. 2023). In essence, kratom use has been shown to help reduce withdrawal symptoms in illicit drug and alcohol addiction and may prove to be a helpful harm-reduction tool.

Evidence of Adverse Effects

291. Across two studies, Leong Bin Abdullah et al. (2019a) and Leong Bin Abdullah et al. (2019b) investigated psychosis and kratom use among 150 regular male kratom users in Malaysia in a cross-sectional clinical survey. The results, based on the Mini International Neuropsychiatric Interview and Brief Psychiatric Rating Scale, indicated that six users experienced mild to severe psychotic symptoms, but these were not linked to duration, quantity, or daily frequency of kratom use.

292. Singh et al. (2019) recruited 70 regular male kratom users and 25 controls to participate in an observational study in Malaysia to assess the effects of long-term kratom use on cognitive functioning. Participants were asked to complete a series of tasks including paired associated learning, motor screening, delayed matching to sample, spatial working memory, reaction time, and attention switching tasks and data was collected using the Cambridge

Neuropsychological Test Automated Battery. It was reported that impairment was detected in visual episodic memory/new memory learning, though there were no differences in terms of kratom use, and there were no alterations for any of the other task areas. Though subjective measures of kratom use were reported (e.g., number of glasses of kratom tea or decoction per day), study methodology precluded derivation of doses at which effects occurred.

293. (Jasim et al. 2022) compared the urinary protein profile of 88 Malay kratom users and 83 control subjects. Urine protein analysis, creatinine assay analysis, and toxicology tests were performed. They reported that kratom users had higher concentrations of urinary protein, protein creatine ratio (PCR), and albumin compared to controls. However, urinary creatine concentrations were reported to be within a healthy range. (Prevete et al. 2023) reported that the clinical implications of this research included early-stage renal injury among kratom users.

294. Leong Abdullah et al. (2021) recruited 100 Malay kratom users and 100 controls to compare prevalence of ECG abnormalities. Resting ECG demonstrated sinus tachycardia in kratom users, though there were no significant differences in other ECG abnormalities. Additionally, the authors reported borderline QTc interval (a measurement of heart health) showed no association with kratom use. (Leong Bin Abdullah and Singh 2021b) also assessed cardiovascular functioning among Malay regular kratom users (N=9) by collecting resting ECG, transthoracic echocardiogram, and blood exams. Contrary to their previous study, they identified a dose-dependent prolonged QTc interval.

295. (Saengmolee et al. 2022) compared heart rate variability (HRV) indices of cardiac autonomic function in 31 regular kratom users and 19 controls in Thailand. Consumption among users occurred via oral chewing of kratom leaves. The ECG acquisition and HRV analyses revealed ultra-short HRV with normalized low frequency (LFn) and high frequency (HFn) indices in kratom users. Additionally, the authors reported changes in cardiac autonomic function with parasympathetic dominance for kratom users and that there was no significant association between HRV indices and kratom use.

296. A study conducted by Singh et al. (2018c) that involved 19 Malay regular kratom users, found that there were no alterations in testosterone levels among long-term users relative to ranges expected in the general population. The authors reported that users consumed an average of 76.23 to 94.15 mg of mitragynine daily via oral decoction. Even at the highest intake level (> 3 glasses of decoction per day), testosterone levels remained within normal ranges.

297. Singh et al. (2018a) conducted a retrospective study on 125 regular kratom users from northern Malaysia. The authors employed the Constipation Assessment Scale to assess constipation prevalence during kratom use. It was reported that users consumed kratom via oral decoction, with many participants consuming more than two to three glasses of kratom tea per day. The authors reported limited constipation problems among participants, including those with the highest daily kratom consumption.

298. Several previously discussed studies reported that liver parameters were within normal ranges among long-term kratom users, relative to non-users (Singh et al. 2018a; Singh et al. 2018c; Leong Bin Abdullah et al. 2020). Schimmel and Dart (2020) conducted a search for epidemiologic studies assessing kratom and liver injury. The authors described the results as limited. A Malaysian study of 58 regular users (consuming kratom twice daily for ≥ 2 years) and 19 controls was cited and reported no difference in transaminase levels between the groups. The study excluded those with diabetes, a history of liver or heart disease, and substance use, but utilized a snowball sampling method. The authors concluded that liver injury related to kratom use was not common and that most users would not experience it. They noted that it was not clear for which subset of users the risk exists or was elevated. They highlighted that a limitation of their review was that the human evidence that they relied on was of low-quality (Schimmel and Dart 2020).

299. Post et al. (2019) reviewed kratom exposure reported to the United States National Poison Data System from 2011 to 2017. It was reported that 1,807 reports were made to U.S. Poison Control Centers (PCCs) during the examined period, with 65% of these cases representing single-substance use and the remaining 35% representing multi-substance or poly drug use.

Approximately 70.8% of reports were for males and the annual number of exposures increased from 13 in 2011 to 682 in 2017, representing a 52.5-fold increase. More than 80% of reports concerned ingestion, with "unknown" route of administration, nasal inhalation, and "other" representing 7%, 3.6%, and 1.4% of reports, respectively. The authors concluded that "[m]ultiple-substance exposures were associated with greater odds of admission to a [health care facility] (OR: 2.80; 95% CI: 2.21–3.55) and a serious medical outcome (OR: 2.25; 95% CI: 1.77–2.85) compared with single-substance exposure" (Post et al. 2019). The critical point of information is the note that multiple substance exposures were associated with greater odds of hospital admission, as it shows that users with polysubstance use are at higher risk of adverse health effects, rather than users of kratom alone. Additionally, instances of single-substance kratom use may be skewed by the weakness of interviews in determining substance use in a health care setting.

300. Olsen et al. (2019) reviewed data on 27,338 United States overdose deaths between July 2016 and December 2017 that were reported to the State Unintentional Drug Overdose Reporting System (SUDORS), with 152 (0.56%) decedents testing positive for kratom, classified as "kratom-positive," following postmortem toxicology. Kratom was reported to be the cause of death, classified as "kratom-involved," for 91 (59.9%) of the 152 kratom-positive decedents. The authors stated that, for seven cases, "… kratom was the only substance to test positive on post-mortem toxicology, although the presence of additional substances cannot be ruled out" (Olsen et al. 2019). A history of substance misuse was found in approximately 80% of kratom-positive and kratom-involved deaths. It was reported that fentanyl was listed as the cause of death for 65.1% of kratom-positive decedents and 56.0% of kratom-involved decedents. Heroin was listed the cause of death for 32.9% of kratom-positive decedents, benzodiazepines for 22.4%, prescription opioids for 19.7%, and cocaine for 18.4% (Olsen et al. 2019). Many users of kratom simultaneously use other substances, including illicit drugs. As a result, deaths attributed to kratom are much more likely to be attributed to other substances, such as fentanyl, rather than kratom use.

301. In evaluating causality in kratom-related liver injury, Schimmel and Dart (2020) reviewed 26 case reports identified through literature search as well as cases reported in the Drug-

Induced Liver Injury Network, on FDA databases, and on internet forums. The cases reported in internet forums lacked sufficient information to make causal assumptions. Across all case reports included in their review, twelve included human liver biopsies. Cholestasis and inflammation were frequently noted, though dose and frequency of use for these cases were not described. Histological analyses demonstrated predominantly cholestatic injury, however, biochemical parameters indicated liver injury was heterogeneous among those affected.

302. The 26 case reports compiled through literature search captured the effects of multiple kratom formulations, including powder, tea, capsules, crushed leaves, and a considerable number of unknown forms. On average, cases experienced onset of symptoms 21 days (range: 2-49) following the start of use. Reported adverse liver symptoms included abdominal discomfort, jaundice, pruritis, dark urine, chills, and light-colored stools. Insufficient reporting of dose and frequency of use prohibited dose-response observations. Case specifics varied considerably based on information, or lack thereof, provided; history of substance use; concomitant drug use; and existing infection, creating a considerable challenge in assessing to what extent kratom use was responsible for liver injury.

303. In the Schimmel and Dart (2020), study, seven cases reported in the Drug-Induced Liver Injury Network were included. Causal association was determined by expert opinion. The median time to symptom onset was 22 days following use. Symptoms were not consistent across cases and included jaundice (5), itching (6), abdominal pain (5), and fever (3). All patients reported ethanol use and all recovered. The FDA Adverse Event Reporting System (FAERS) was also queried for cases of hepatotoxicity. The authors selected 10 cases from 408 reports that mentioned mitragynine or herbals but acknowledged that FAERS included incomplete reports and unverified information and that their selection may have contained cases unlikely to be caused by kratom. Given that all patients reported ethanol use, incomplete reports, and unverified information, it is difficult to determine the potential of kratom to induce such livery injuries.

304. In a 2019 study comparing the risk of death associated with kratom use relative to opioid use, Henningfield et al. stated that "… there is currently no correlation between MG

[mitragynine] blood levels and observed behavioral, adverse, or lethal effects. It is therefore unclear whether MG serves as a good indicator for reporting kratom intoxication and especially kratom fatality" (Henningfield et al. 2019). Given this, the estimates provided by Post et al. (2019) and Olsen et al. (2019) may be questioned.

Dependence, Abuse, and Withdrawal

305. Serotonergic and dopaminergic systems, which are fundamental to habit-forming, are activated by well-known addictive drugs (e.g., heroin, cocaine, methamphetamines) and when active, these systems can inform human reward systems/perceptions and potentially contribute to dependence or addiction (Hassan et al. 2013).

306. It was reported that chronic exposure to kratom preparations could yield withdrawal symptoms in humans, including hostility, aggression, inability to work, excessive tearing, aching of muscle and bones, jerky limb movements, dry mouth, frequent urination, and constipation (Hassan et al. 2013).

307. According to Stanciu et al. (2019), medical facilities in the United States have recently seen more individuals presenting with symptoms after "... abrupt ..." discontinuation of long-term kratom use (Stanciu et al. 2019). The European Monitoring Center for Drugs and Drug Addiction have reported that symptoms are first seen 12 to 24 hours from last use and can have a duration of up to seven days (Stanciu et al. 2019). Some physiological elements may include mydriasis, nausea, sweating, chills, muscle and body aches, tremors, and twitches as well as cravings and psychological symptoms that may include insomnia, anxiety, fatigue, irritability, hostility, mood disturbance, and hallucinations. Reports have noted that the intensity of withdrawal symptoms are positively correlated to daily consumption, duration, and frequency of kratom use (Stanciu et al. 2019). Chronic use of kratom may result in withdrawal symptoms, but it appears that such outcomes depend on duration and frequency of use.

308. As reported by Eggleston et al. (2019), a total of 2,312 kratom exposures were reported to the National Poison Data System between January 2011 and July 2018. This included 935 single substance exposures, with 86.2% cases reporting oral ingestion for kratom use and

61.6% reporting intentional abuse or misuse. The authors concluded that 1,377 exposures to kratom involved at least one other substance (Eggleston et al. 2019).

309. In a cross-sectional survey of 293 regular kratom users in Malaysia, it was reported that 32% of users had increased their kratom intake over time, 64% experienced withdrawals within one to three days following cessation, and 89% had attempted at least once to abstain from use in the past (Singh et al. 2014; Anand and Hosanagar 2022). The intensity of cravings varied, with 23% of users reporting high cravings and 77% of users reporting low cravings. Additionally, more than half of the regular users "… developed severe kratom dependence problems, while 45% showed a moderate kratom dependence" (Singh et al. 2014; Anand and Hosanagar 2022).

310. In another study, prevalence and correlates of past year kratom use were estimated using data from the 2019 National Survey on Drug Use and Health, which comprised a nationally representative probability sample of non-institutionalized individuals aged 12 years or older in the United States (N=56,136) (Palamar 2021). Results showed that an estimated 0.7% of United States individuals had used kratom in the past year, and "[p]ast-year proxy diagnosis of prescription opioid use disorder was associated with increased odds for kratom use (AOR=3.20, 95% CI=1.38, 7.41), with 10.4% … of those with use disorder reporting use" (Palamar 2021). They stated that "[t]hose reporting past-year cannabis use both with (AOR=4.33, 95% CI=2.61, 7.19) and without (AOR=4.57, 95% CI=3.29, 6.35) use disorder and those reporting past-year cocaine use (AOR=1.69, 95% CI=1.06, 2.69) and prescription stimulant misuse (AOR=2.10, 95% CI=1.44, 3.05) not accompanied with use disorder were at higher odds for kratom use" (Palamar 2021).

311. Schimmel et al. (2021) analyzed a cross-sectional survey of non-medical use of prescription drugs program (NMURx) in the third quarter of 2018 and first quarter of 2019 among 59,714 respondents aged 18 years or older. The authors estimated that the prevalence of past year kratom use was 0.8%. They reported that among those with past year kratom use, 36.7% used prescription opioids non-medically, 21.7% used illicit opioids, 54.4% used another illicit drug, and 67.1% used cannabis.

312. These papers indicated that there was a notable overlap between people who used kratom and people who used prescription opioids (non-medically), cannabis, or illicit drugs. While firm conclusions cannot be drawn from these studies alone, these papers may clarify the potential use of kratom to help reduce symptoms from other opioid use among users, or potentially a similarity in addictive behaviors among users. These associations should be considered when reviewing the available epidemiology concerning kratom use.

Case Reports and Series

313. Many case reports and series describing individuals' clinical presentation following kratom use have been published (Afzal et al. 2020; Botejue et al. 2021; Halim et al. 2021; Lei et al. 2021; Thewjitcharoen et al. 2022; Ahmed et al. 2023; Arhin et al. 2023; Bachu et al. 2023; Haider et al. 2023; Kiyokawa et al. 2023; Peran et al. 2023; Roma et al. 2023; Smith et al. 2023; Shi and Shea 2024). Prevete et al. (2023) reported that kratom users self-reported analgesic and stimulatory effects and that they tended to lessen their self-administered kratom dose over time (Smith et al. 2023). Kratom users also reported the belief that kratom use was safe and beneficial at low to moderate doses and did not carry the risks associated with prescription or illicit drugs (Smith et al. 2023). Kratom users did describe tolerance to kratom over time and that they experienced jitteriness and vision impairment when a dose higher than intended was consumed (Smith et al. 2023).

Reports Confounded by Polysubstance Use

314. As reported by Prevete et al. (2023):

> "While in Asia kratom has been used with a limited number of associated health hazards, most of the safety issues have been reported in the West, mainly among users ingesting kratom on a daily basis in combination with other drugs, such as prescription medicines (e.g., antipsychotic, antidepressant, and hypnotic drugs, among others) and psychoactive substances (e.g., amphetamine, methamphetamine, cyclopropyl fentanyl, opioids, ethanol)" (Prevete et al. 2023).

315. Put simply, the few adverse health effects reported among kratom users in Western countries are generally associated with the simultaneous use of other, more hazardous substances, prescription or otherwise. These adverse effects are rare to nonexistent when kratom is used in a traditional capacity. Additionally, toxicity associated with kratom use has been attributed to contaminants including unscheduled substances, microbes, and toxic metals (Prevete et al. 2023). For instance, Smith et al. (2022) reported mental and psychological distress among kratom users, specifically those who combined kratom with nonmedical opioids and methamphetamine. Authors have hypothesized that the use of mitragynine and other substances may have combined effects at endogenous receptors, specifically the adrenergic and estrogenic receptors, resulting in nervous system depression (Kronstrand et al. 2011; McIntyre et al. 2015). It is difficult to attribute any adverse health effects to kratom use alone in many studies due to the likelihood of users simultaneously using other substances, such as illicit drugs or alcohol, or even contaminants within certain kratom products.

316. Afzal et al. (2020) and Halim et al. (2021) documented cases of kratom-induced tonic-clonic seizures in individuals who consumed three to four 8 mL bottles of kratom daily for 1.5 years and up to >200 mL of kratom drink eight times per month for 84 months; however, it was reported that these cases exhibited polysubstance use behaviors and had histories of mental illness and substance use disorders (Afzal et al. 2020; Halim et al. 2021). Ten cases of kratom-associated mania and psychosis were reported by Bachu et al. (2023). The duration of use among these cases ranged from two weeks to 15 years, and a significant association between kratom use

and worsening mania and psychosis was reported. No concentration data was provided from these reports, making it difficult to generalize the documented effects to other kratom users.

317. Botejue et al., 2021, Thewjitcharoen et al., 2022, Haider at al., 2023, and Roma et al., 2023, described cases of kratom-associated liver injury (Botejue et al. 2021; Thewjitcharoen et al. 2022; Haider et al. 2023; Roma et al. 2023), Symptoms included abdominal pain, back pain, itching, unintentional weight loss, fever, nausea, fatigue, jaundice, dark urine, constipation, and loss of appetite, though clinical presentation was not consistent across cases. It was reported that diagnoses included cholestatic hepatitis consistent with drug-induced liver injury (DILI), elevated liver enzymes, moderate extrahepatic biliary ductal dilation, and general cholestatic injury. Evidence of polysubstance use, including prescription, recreational, and or illicit drugs, was revealed via testing in all but one case. Use parameters were not discussed in full, though, in cases for which dosage and duration of use were provided, kratom use varied.

318. Peran et al. (2023) reported that among 10 cases of kratom poisoning, the overdose toxidrome appeared opioid-like in that individuals were reported to have experienced hypertension, tachycardia, mydriasis, miosis, vomiting, bradycardia, and impaired consciousness. Cases reported using kratom tea, tablets, or powder orally and overdose symptoms manifested 1 to 24 hours following use. All cases were young adults using kratom recreationally, and dosage was not provided. Additionally, urine toxicology screening demonstrated that 70% of cases tested positive for other substances.

319. Several case reports and series suggested that kratom-induced hypoxia (following the consumption of eight 400 mg kratom capsules and a bottle of extra-strength kratom extract) and withdrawals (following ingestion of 35 g of kratom daily for an unspecified period of time) can be treated with buprenorphine/naloxone (Ahmed et al. 2023). Notably, kratom dependence, when coupled with polysubstance use, may necessitate elevated medication dosing (Lei et al. 2021; Arhin et al. 2023; Kiyokawa et al. 2023).

320. Several publications have reported deaths among polysubstance users who used kratom (Holler et al. 2011; Neerman et al. 2013; Karinen et al. 2014; Domingo et al. 2017;

Chodchoy and Sinchai 2020). (Shi and Shea 2024) described a fatal overdose in a 44-year-old male with a history of schizoaffective disorder, bipolar type, abdominal pain managed by hydromorphone, the dose of which had recently been increased, and loss of appetite. Toxicological testing revealed hydromorphone (79 ng/mL), mitragynine (560 ng/mL), and olanzapine (240 ng/mL) were present post-mortem, and autopsy showed that the decedent had abdominal distention with >4 L ascites (Shi and Shea 2024). McIntyre et al. (2015) reported the autopsy results of a deceased 24-year-old male with a history of alcohol abuse, depression, accidental overdose, and pharmaceutical suicide attempts. Samples were collected ~30 hours post-mortem, stored at 4°C, and analyzed via GC-MA. Mitragynine (0.23 mg/l in peripheral blood, 0.19 mg/l in central blood, 0.43 mg/l in liver, <0.05 mg/l in vitreous humor, and 0.37 mg/l in urine, with no detection in gastric fluids), Venlafaxine (1.1 mg/l), O-desmethylvenlafaxine (1.6 g/l), diphenylhydramine (0.45 g/l), mirtzapine (0.24 g/l), and ethanol (0.02 g/l) were detected. Kronstrand et al. (2011) reported nine mortalities wherein blood mitragynine concentrations ranged from 0.02 to 0.18 µg/g and O-desmethyltramadol blood concentrations ranged from 0.4 to 4.3 µg/g. Two cases were female, and seven were male. The mean age was 28 years. The cases had histories of drug abuse, and autopsies revealed lung edema and congestion, hepatitis, liver steatosis, and brain edema. A case of a 56-year-old woman with a history of COPD and one-day post-mortem blood concentrations of 2.500 ng/ml mitragynine, 63 ± 5 ng/ml lorazepam, and 0.19 ± 0.01 mg/l oxycodone was described by Wang and Walker (2018). The death was attributed to acute mitragynine toxicity; however, the oxycodone blood concentration neared the lethal reference value (>0.21 mg/l). Jittasopa and Srisont (2021) reported that a deceased 43-year-old man had a blood mitragynine concentration of 3,600 ng/ml and that death was attributed to an asthma attack (Jittasopa and Srisont 2021).

321. (Ahmad et al. 2021)investigated cases of suspected kratom-induced liver injury in registered the United States Drug-Induced Liver Injury Network. Of 404 cases of liver injury associated with herbal and dietary supplements enrolled in the network between 2003 and 2019, 11 cases were attributed to kratom at varying levels of certainty. It was noted that the frequency of these reports was not consistent over the 16-year time period. Four patients reported a history of

alcohol abuse. The authors reported that ten patients had initial bilirubin > 2.5 mg/dL (median peak total bilirubin = 11.7 mg/dL), initial median serum alanine aminotransferase (ALT) was 362 U/L, and alkaline phosphatase (AlkP) was 255 U/L. All patients developed jaundice, while only some presented with itching and mixed or cholestatic pattern of injury as well, and recovered (10 by 6 months, 1 by 1 year). Duration of use and dose was not reported. This study showed that it is quite difficult, if not impossible, to analyze kratom's health effects in users as in many instances any adverse effects are caused by other substances the user may have ingested, such as illicit drugs or alcohol.

322. Gershman et al. (2019) reviewed Colorado death certificates to identify deaths related to kratom between 1999 and 2017, finding 15 total. The authors compiled the autopsy reports for the 15 kratom-related deaths and determined 11 cases involved multidrug ingestion (2-6 drugs), eight of which contained positive test results for opioids. The remaining four deaths, for which fatality was attributed to mitragynine toxicity only, were investigated further. They reviewed police records and tested available blood samples with high-performance liquid chromatography with tandem mass spectrometry. The authors concluded that 14 out of 15 kratom-related deaths involved multiple drugs, with one case of mitragynine toxicity lacking residual blood for confirmatory testing. The mitragynine levels for these cases ranged from 16 to 4,800 ng/mL. In all cases where information was available, deaths related to kratom were in actuality attributable to multiple drug use, and in a majority of cases, opioid use was noted. The authors did not find a death clearly attributable to kratom use alone.

Mitragynine-Only Reports

323. Behonick et al. (2022) reported two fatalities in which mitragynine was detected at high concentrations, and the deaths were ultimately attributed to acute mitragynine intoxication. A 37-year-old man was found dead after a bicycle ride, and it was reported that he had a blood mitragynine concentration of 2,325 ng/ml and lung congestion. While no prescription and illicit drugs were detected in urine, the case had a history of traumatic brain injury, alcohol use, cigarette smoking, and withdrawal seizure disorder. The second case was a 33-year-old man who passed

away after a low-speed car crash. It was reported that the individual had a blood mitragynine concentration of 3,809 ng/ml and pulmonary edema. Again, no drugs were detected via urine screen, but the case had a history of anxiety, depression, and use of cocaine, marijuana, kratom, and ethanol (Behonick et al. 2022). Mata and Andera (2020) documented three fatality cases (mean age = 33 years) with blood mitragynine concentrations of 1,250 to 4,310 ng/ml (central) and 1,590 to 3,420 ng/ml (peripheral), and no confounding substances detected (Mata and Andera 2020). Mata and Chang (2023) reported that a 32-year-old male with a history of alcohol abuse, drug use, and suicide attempt had post-mortem blood mitragynine concentrations of 7,500 ng/ml (cardiac) and 3,300 ng/ml (femoral) with no confounding substances detected (Mata and Chang 2023).

324. Case reports and series are useful in generating hypotheses and describing disease profiles. They alert all of us to the potential for a relationship that may be harmful to those exposed. However, we acknowledge that these study designs are not suitable for producing generalizable conclusions regarding causation (that is, for demonstrating whether a given kratom dose will cause an effect across a population or just a segment of the population). Moreover, recent evidence indicates that genetic differences across persons can significantly influence response to kratom and mitragynine. That is, one person may note substantial effects (positive or negative) at the same kratom or mitragynine dose, and another person may note zero effects. This is likely related to genetic variability in both the tissue-specific prevalence and binding affinities of opioid receptors, along with genetically based variability in CYP enzyme expression (which could modulate metabolism of mitragynine to 7-hydroxymitragynine).

Biomarkers for Kratom Alkaloids in Humans

325. Currently, mitragynine concentrations are determined via blood or urine testing. Studies that present concentrations established by these testing methods are limited in that they rarely report the kratom or mitragynine dose. Consequently, it is difficult to determine the magnitude or timing of kratom use when analyzing alkaloid detection in blood or urine.

Blood Testing

326. Post-mortem case reports and retrospective analysis of autopsy findings are the source of most data of human blood concentrations of kratom alkaloids (Domingo et al. 2017; Papsun et al. 2019; Chodchoy and Sinchai 2020; Mata and Andera 2020; Mata and Chang 2023).

327. Table 7 below summarizes the reported blood mitragynine concentrations of living and deceased kratom users. Notably, the studies summarized below with no pharmacological confounders are the most informative for human risk assessment due to the lack of documented polysubstance use. Fatal or injury cases with evidence of multiple substance use prior to the event present a challenge in deciphering which substance the effect could be attributed to and generate questions pertaining to drug-drug interactions. However, these studies are limited by an inability to assess differing metabolic phenotypes in users.

Table 7. Human blood mitragynine concentrations among fatal cases with few to no confounding compounds reported post-mortem.

Number of Samples	**Blood Concentration (ng/ml)**	**Confounding Substances**	**Study**
1 [b]	2,325	No	Behonick et al. (2022)
1 [b]	3,809	No	Behonick et al. (2022)
1[c]	7,500 (cardiac)	No	Mata and Chang (2023)
1 [c]	3,300 (femoral)	No	Mata and Chang (2023)
3 [b]	1,250-4,310 (central)	No	Mata and Andera (2020)
3 [b]	1,590-3,420 (peripheral)	No	Mata and Andera (2020)
41 [b]	2.98-1,554.23	Not reported	Chodchoy and Sinchai (2020)
1,001 [a b]	5.6-29,000 [d]	Not reported	Papsun et al. (2019)

[a]Live subjects.

[b] Post-mortem samples.

[c] Unspecified vital status.

[d] The peak 29,000ng/ml finding was determined to be an outlier from a post-mortem case. Adjusted mean = 381 ng/ml.

Urine Testing

328. While ELISA methods for detecting mitragynine in urine may be the future of cheap, rapid tests for providing evidence of kratom use, they are limited by an inability to determine alkaloid concentrations (Lei et al. 2021; Jasim et al. 2022).

329. Fu et al. (2015) reported an observed <±3% variance in concentrations of mitragynine and 7-hydroxymitragynine after 14 days of sample storage. Le et al. (2012) stated that mitragynine remains stable in urine for five days when refrigerated (4ºC) and frozen (-20ºC). Table 8 below summarizes the reported urine concentrations of kratom alkaloids across the published literature.

Table 8. Urine concentrations of kratom alkaloids obtained via urinalysis.

Alkaloid	**Number of Samples**	**Urine Concentration (ng/ml)**	**Relative Yield (%)**	**Study**
Mitragynine	50 [a]	1,580-12,510		Le et al. (2012)
	1 [c]	51.25		Fu et al. (2015)
	NA [c]	500-9800		Lee et al. (2020)
	1 [b]	<10		Domingo et al. (2017)
	1 [b]	>400		(Domingo et al. 2017)
	26 [b]	288.10		Chodchoy and Sinchai (2020)
	26 [a]	273.65		Chodchoy and Sinchai (2020)
	16 [b]	26-1,987		Basiliere and Kerrigan (2020b)
	29 [c]	11-10,079		Helander and Rylski (2023)
7-hydroxymitragynine	50 [a]		0.1-2.4	Le et al. (2012)
	1 [c]	8.72		Fu et al. (2015)
	16 [b]	<LOD-295		Basiliere and Kerrigan (2020b)

5-desmethylmitragynine	50 [a]		0.0-9.0	Le et al. (2012)
17-hydroxymitragynine	50 [a]		0.0-26.7	Le et al. (2012)
Speciociliatine	16 [b]	23-3,309		Basiliere and Kerrigan (2020b)
	29 [c]	19-14,332		Helander and Rylski (2023)
Speciogynine	16 [b]	59-1,684		Basiliere and Kerrigan (2020b)
	29 [c]	4.0-3,700		Helander and Rylski (2023)
Paynantheine	16 [b]	2-317		Basiliere and Kerrigan (2020b)
Mitraciliatine	29 [c]	55-12,039		Helander and Rylski (2023)

[a]Live subjects.

[b]Post-mortem samples.

[c]Unspecified vital status.

FDA Study to Identify a Safe Dose For Kratom(2024)

330. In 2024, the FDA reported some results from its single ascending dose study, a Phase I safety trial, which was conducted to assess the safe dose range for kratom and identify any serious adverse events following dosing (Reissig et al. 2024).

331. They recruited 40 non-dependent, recreational polydrug users with opioid experience. Specially, inclusion criteria stated that the participants had used opioids recreationally at least 10 times over their lives, had more than one use in the past 12 weeks, and had used two or more perception altering drugs or stimulants on 5 or more occasion over their lives. The authors noted that "[s]ubjects were otherwise healthy with exclusions for significant diseases and a history of substance use disorder" (Reissig et al. 2024).

332. Cohorts consisted of eight subjects each, with 2 randomly selected placebos per cohort. For those that did not receive the placebo, doses were 1, 3, 8, 10, and 12g in capsule form (500 mg/capsule). Subjects received a standardized high fat meal prior to dosing. Pharmacodynamic effects were assessed using Visual Analog Scales (VAS) and blood samples (Reissig et al. 2024)

333. All subjects completed the study. No subjects reached stopping criteria, which was defined as a kratom-related serious adverse event occurring in a cohort or moderate or severe adverse event in at least 50% of the cohort. The mitragynine content in each capsule was 5.07 ± 0.71 mg, and 7-hydroxymitragynine was below the limit of quantification (Reissig et al. 2024).

334. The authors reported that:

> "No deaths or serious adverse events (SAEs) occurred. An SAE was defined as any untoward medical occurrence that results in death, is life-threatening, requires inpatient hospitalization or prolongation of existing hospitalization, results in persistent or significant disability or incapacity (defined as a substantial disruption of a person's ability to conduct normal life functions), is a congenital anomaly or birth defect, is an important medical event (including development of drug dependence or drug abuse) that may jeopardize the subject or require intervention to prevent one of the other outcomes listed above (according to medical judgment of an Investigator). In addition, no stopping criteria were reached, defined as one kratom-related SAE occurring in a cohort or moderate or severe AEs in 50% or more subjects in a cohort" (Reissig et al. 2024)

335. Adverse events were grouped by System Organ Class when total kratom events reported were greater than placebo. It was reported that 5 subjects who received kratom and 2 placebos experienced gastrointestinal disorders, 5 kratom group members and 6 placebos experienced nervous system disorders, 3 kratom group members and 1 placebo experienced psychiatric disorders, 2 kratom group members and no placebos experienced skin and subcutaneous disorders, and 2 kratom group members and no placebos experienced general

disorders. The authors did not provide further information on the kind of adverse events (referred to by the authors as "preferred term") summarized above; however, they noted that vomiting was the only event for which the frequency among those who received kratom exceeded that in the placebo members. Specifically, vomiting was observed in 2 subjects who received 8 g of kratom, 1 subject who received 10 g, and two subjects who received 12 g. No vomiting was reported at doses under 8g (Reissig et al. 2024).

336. They reported that circulating mitragynine blood concentrations increased with dose. Tmax was reported to be variable and range from 2 to 4 hours. They noted that at doses >1g, mild pupillary constriction was observed in a dose-related manner. The highest pupillary constriction (shrinking of the pupils) was observed among the 12g group. Pupillary constriction is a known side-effect of opioid use and was likely studied in comparison to such substances. The authors stated that pupillary constriction is consistent with mu-opioid effects but, for kratom, was "relatively mild" in comparison to morphine or oxycodone (Reissig et al. 2024).

337. The authors added that no dose-related effects were observed "on numerous study endpoints," mentioning "drug liking" (traditionally defined as a subjective measurement to the question" Do you like the drug effect you are feeling now?") but they did not discuss these other endpoints in detail. They added that even at the highest dose, changes from baseline did not differ significantly from placebo. Analyses for overall drug liking and likelihood of taking drug again were not observed to be dose-related, while non-key secondary endpoints (i.e., "high" and "feeling drunk") were highest among the highest dose group. The authors stated that "Cmax values following 12 g appeared to be greater than those after the 10g dose, suggesting dose-related PK effects" (Reissig et al. 2024).

338. While no other information on the study or its findings have been released by the FDA at this time, the AKA reported that the FDA announced in January 2024 that it plans to conduct a Human Abuse Potential study to assess kratom dependency and addiction liability potential (American Kratom Association 2024).

What the Human Data Tells Us

339. The existing literature investigated the effects of kratom on multiple endpoints, including muscular, sensory, behavioral, mental, renal, biochemical, cardiovascular, endocrine, gastrointestinal, hepatic, and dependence, which encompasses most bodily systems. Together, the literature does not indicate that kratom use increases the risk of death or acute injury. While different users may experience different effects, less is known about factors that influence one's response to oral use of kratom, such as metabolic phenotype and preexisting conditions. Additionally, the documented fatal cases involving kratom overwhelmingly exhibit confounding by polysubstance use. Prescription or illicit drug interactions with kratom may present a risk as the effects of combining kratom with other substances are not currently understood. The studies discussing adverse health outcomes following kratom use do not present generalizable evidence that these effects are likely to be seen in the broader kratom-using population. However, there is evidence of the beneficial effects of kratom at low to moderate doses, including pain relief. Namely, it has been suggested that kratom has therapeutic potential to mitigate withdrawals following the cessation of use of other, usually more harmful, substances.

Basis of Opinion 7

340. Kratom has not been shown to increase the risk of adverse cardiovascular effects in humans, nor pose significant cardiovascular risks from chronic use at any dose evaluated. If Ms. Moller experienced a cardiovascular effect which led to her death, it would have been unrelated to her kratom use. To support my opinion, I have performed a literature search on the cardiovascular effects of kratom use.

Plaintiffs' Experts Opinions Regarding Kratom and Cardiovascular Health

341. Several of the Plaintiff Experts in this case have opined regarding the potential cardiovascular health effects of kratom use. It should be noted that, in the cases in which these experts provided citations for their opinions, they overwhelmingly relied upon case reports which were often insufficient to draw the conclusions that they claimed or opined regarding the ability of kratom to cause adverse cardiovascular health effects.

342. Dr. Thundiyil opined that cardiac arrhythmias were a documented symptom of kratom use and that kratom-related deaths involve clinical findings consistent with cardiorespiratory arrest (Thundiyil: pg. 9). He added that:

> "[k]ratom also acts as a stimulant. Stimulants like kratom have the potential to cause enlargement of the heart. An enlarged heart is a heart that is electrically susceptible to arrythmias. The stimulant effects of kratom increase the likelihood of electrical instability and sudden cardiac death. The stimulant effect of kratom has been associated to occur at lower doses" (Thundiyil: pg. 9-10).

343. Dr. Thundiyil specifically opined that "[t]he blood concentration of mitragynine (150 ng/ml) found in Ms. H. Moller's blood was sufficient to cause adverse events" (Thundiyil: pg. 10). Dr. Thundiyil did not specify which papers supported his opinions, however in his reliance list he generally noted that he relied, among others, on papers by Leong Bin Abdullah et al. (2021a,b) and Krantz et al. (2023), which are discussed in detail below.

344. Dr. Wharton opined adverse cardiac events can be caused by exogenous substances and stated that a review of opioid-associated arrhythmia in North America published in 2023 "… demonstrated a 'disproportionate' association of 'life threatening arrhythmias in North America' with mitragynine" (Wharton: pg. 2). Based on his reliance list, the paper Dr. Wharton relied upon to make this claim is Krantz et al. (2023), which is discussed below.

345. Dr. Williams opined that, according to a 2023 FAERS study, "… MG was associated with a greater incidence of ventricular arrhythmia/cardiac arrest than any other drug tested… In 39% of those cases, MG exposure was indicated in the absence of concurrent QTc prolonging drugs, thereby suggesting that MG was the primary driver of cardiotoxicity" (Williams: pg. 2-3).

346. Dr. Williams also relied upon the Krantz et al. (2023) paper to draw his conclusions.

Cardiovascular Health and Kratom

In Vitro Studies

347. Studies have reported that mitragynine intake may induce dose-dependent prolonged QTc intervals (Leong Bin Abdullah and Singh 2021a). It was reported that in human induced pluripotent stem cell-derived cardiomyocytes (hERG-HEK293), mitragynine and its analogues (paynantheine, speciogynine, and speciociliatine) suppressed the rapid delayed rectifier potassium current by 67% to approximately 84% at doses of 0.1 to 100 μM, with an IC_{50} of 0.91 to 2.47 μM (Lu et al., 2014). At 10 μM, mitragynine was reported to significantly prolong action potential duration at 50% and 90% repolarization, and induce arrhythmia without altering the L-type Ca^{2+} current (Lu et al. 2014). Lu et al. (2014) did not provide sufficient blood concentration data to predict a typical human dose at which cardiotoxicity may occur.

348. In response to Lu et al. (2014), Eckhardt and Nickel (2023) stated:

> "In vitro study human stem-cell derived cardiomyocytes demonstrated a dose-dependent blockade of I_{kr}, prolonging the action potential, suggesting an hERG-dependent mechanism here as well. It is notable, though, that these experiments were carried out with concentrations considerably higher than serum levels seen in overdose. There was no attempt to compensate for the lack of I_{k1} in human stem-cell derived cardiomyocytes, which is known to exaggerate the effect of hERG blockade" (Eckhardt and Nickel 2023)

349. Cardiotoxicity was also reported with HEK293-hERG1a/1b recombinant cells, where mitragynine was shown to inhibit the rapid delayed rectifier potassium current with an IC_{50} of 332.70 nM (Tay et al., 2019). Mitragynine was reported to cause a significant reduction of fully glycosylated hERG1a protein expression, but upregulated both core-glycosylated expression and hERG1a-Hsp09 complexes (Tay et al. 2019). In essence, the authors concluded that kratom alkaloids inhibited hERG1A/1B current at low concentrations but upregulated the hERG1a-Hsp90 complex at higher concentrations (Tay et al. 2019). Such *in vitro* effects have not been demonstrated *in vivo* or in humans and would likely have little clinical significance, but could potentially translate to cardiac irregularity at certain high doses of mitragynine.

350. Nawaka et al. (2025) reported investigating the effects of kratom leaf extracts on hypolipidaemia via the expression of LDLR and PCSK9 in HepG2 cells. Real-time RT-PCR, Western blotting, flow cytometry, and BODIPY-LDL uptake assays were reportedly performed, and it was determined that red-vein and white-vein kratom leaf extracts increased LDLR protein expression and decreased PCSK9 expression by downregulating SREBP2 and HNF-1α. In addition, it was reported that both strains of kratom leaf extracts increased LDL uptake in HepG2 cells, and that mitragynine significantly increased LDLR protein expression while decreasing PCSK9 expression. The authors concluded that red-vein and white-vein kratom leaf extracts exhibited hypolipidemic effects and could act as a potential supplement to improve hypercholesterolemia.

351. Tay et al. (2016) reported investigating the effects of mitragynine on the current, mRNA, and protein expression of the human ether-a-go-go-related gene (hERG) channel in hERG-transfected HEK293 cells and *Xenopus* oocytes. In addition, the effects of mitragynine on Kir2.1 and GIRK channel currents were analyzed in the oocytes. It was reported that mitragynine inhibited the hERG tail currents following depolarization pulses with IC_{50} values of 1.62 uM and 1.15 uM in the transfected cell line and oocytes, respectively. It was noted that mitragynine likely interacts with high-affinity drug-binding sites in the hERG channel pore cavity because the Y652A and F656A S6 point mutations reduced the inhibitor effects of mitragynine. It was reported that mitragynine was found to inhibit hERG protein expression but not affect hERG expression at the transcriptional level. Additionally, the authors reported that mitragynine inhibited the GIRK channel. The authors indicated that additive cardiotoxicity risks may result from the blocking of both hERG and GIRK channels.

In Vivo Studies

352. In a study robust for its time, Grewal (1932b) performed *in vivo* and *ex vivo* analyses of mitragynine in frogs, mice, rabbits, and cats, along with presenting some information on human-specific effects. The author evaluated the effects of mitragynine fumarate and mitragynine acetate salts. In an analysis of repeated-dose toxicity, Grewal (1932b) administered 15 mg/kg-bw

mitragynine to rabbits for "several" weeks. This was reported to yield "… a progressive loss of weight …" and coat deterioration and balding. The author reported that the lowest lethal dose (LD_{Lo}) of mitragynine administered intravenously in frogs and rabbits was 0.13 g/kg for both species. In mice, the LD_{Lo} was reported to be 0.37 g/kg via subcutaneous injection and 0.20 g/kg via intraperitoneal injection. In frogs, the author stated that acute effects included paralysis and respiratory cessation, with death observed after eight hours. In mice and rabbits, symptoms of acute mitragynine toxicity included "… exaggerated reflexes, tremors, muscular incoordination … convulsions, and … respiratory failure." Grewal (1932b) added that humans dosed orally with 50 mg mitragynine showed "… motor excitement, followed by giddiness, Rombergism, and tremors of the extremities, face, and tongue." In short, mitragynine demonstrated *in vivo* acute toxicity that could be described as moderate (Hodge and Sterner 1949).

353. Decebrate (immobilized) cats dosed intravenously with 15 to 20 mg mitragynine were examined for intestinal movement to determine the effects of mitragynine on smooth muscle fibers. It was reported that the tested mitragynine doses reduced the intestinal threshold for stimulation associated with the vagus nerve. Administration of atropine was reported to eliminate the effects of mitragynine, prompting the author to conclude that mitragynine was "… not likely to act directly on muscle fibers" (Grewal 1932b).

354. In examining the effects of mitragynine on striped muscle fibers, Grewal (1932b) added that unspecified doses of mitragynine elicited "… a progressive fall in the power of muscle contraction" in unspecified frog species. The excitability was reported to decrease continuously over time, with frog muscle fatigue increasing at an expedited rate in the presence of mitragynine relative to its absence. It was reported that mitragynine depressed cardiac muscle contraction in the hearts of frogs and rabbits while simultaneously increasing coronary vessel outflow. Similarly, intravenous administration of 2.5 mg mitragynine in anesthetized frogs, rabbits, and cats demonstrated that mitragynine "… had the general effect of dilating the vessels … caus[ing] a marked fall in blood pressure, which recover[ed] quickly after a single dose." Repeated administration of mitragynine elicited a continued depression of blood pressure and increased

cardiac output associated with the aforementioned vasodilation. However, the author concluded that these effects were "… not due to any direct action of [mitragynine] on the heart …" but due to "… dilating blood vessels" (Grewal 1932b).

355. Collectively, the results reported by Grewal (1932b) are a helpful introduction to the *in vivo* effects of mitragynine and kratom. Despite this, the dated approach of the study and poorly specified methods make generalization to current products and exposure scenarios difficult. Moreover, though Grewal (1932b) failed to specify doses for many examined endpoints; those doses specified are, in light of modern toxicological and pharmacological knowledge, relatively high and unrepresentative of typical use scenarios for kratom products. Furthermore, the results reported specifically for muscle activity in cats, rabbits, and frogs, while informative, do not provide detailed mechanistic insight into mitragynine's activity, in contrast to more recent literature. They indicate that mitragynine has few direct cardiotoxic effects, but that it does act as a vasodilator.

356. In another early study, Macko et al. (1972) evaluated the antinociceptive, antitussive, nalorphine antagonism, cardiotoxicity, and acute and subacute toxicity of mitragynine in Sprague-Dawley rats, cats, and dogs. The reported antinociceptive oral ED_{50} for mitragynine salts in rats ranged from 16.8 to 20.2 mg/kg-bw based on the methods of D'Amour and Smith (1941) and Randall and Selitto (1957). Analgesic effects were also reported in dogs at 2.0, 4.0, 8.0, 12.0, 16.0, and 24.0 mg/kg-bw mitragynine via oral administration, although dose dependence was unclear or absent, and adverse side effects, including shivering and decreased spontaneous motor activity, were also observed across doses. In the hot plate test in mice (54.5°C), 92 mg/kg-bw mitragynine yielded 100% analgesia via oral administration, but no analgesia was reported for a similar dose administered subcutaneously. In dogs, the antitussive oral ED_{50} for mitragynine HCl and mitragynine ethane disulfonate salts were reported to be 2.3 and 1.8 mg/kg-bw, respectively. The authors stated that intraperitoneal (36.8 mg/kg-bw) and oral (55.2 mg/kg-bw) mitragynine salts failed to inhibit gastrointestinal activity in dogs based on the charcoal meal test.

357. Cardiovascular-specific results reported by Macko et al. (1972) were mixed. In anesthetized dogs, intravenous mitragynine salt doses of 0.092 to 9.2 mg/kg-bw were reported to cause no adverse effects on blood pressure. In anesthetized cats administered intravenous mitragynine salts, weak hypotension was reported at 0.46 to 0.92 mg/kg-bw in some animals and only at 4.6 mg/kg-bw in other animals. Intravenous mitragynine salts at 0.46 to 2.3 mg/kg-bw yielded "… a transient lowering of mean arterial blood pressure" in one cat, which later died due to respiratory failure at 4.6 mg/kg-bw.

358. Chichagi et al. (2024) identified three rat studies in their review of the literature. They reported that two of the rat studies observed no significant differences in or effects on cholesterol and triglyceride levels when compared to the control group or baseline (pre-administration of mitragynine) (Sabetghadam et al. 2013b; Ilmie et al. 2015).

359. Ilmie et al. (2015) orally administered 100, 200, and 500 mg/kg mitragynine to 24 adult malperane Sprague-Dawley rats for 28 days. They identified no significant differences in cholesterol levels when the dosed animals were compared to controls. In contrast, (Harizal et al. 2010) observed a rise in blood pressure (1 hour after dosing) as well as an increase in triglyceride and cholesterol following oral administration of 100, 500, and 1000 mg/kg mitragynine to rats for 14 days (Harizal et al. 2010)

360. Sabetghadam et al. (2013b) treated male and female Sprague-Dawley rats orally with 1, 10, and 100 mg/kg-day mitragynine dissolved in Tween-20 solution (total volume of 5 ml/kg) for 28 days. The authors reported that "[t]he lowest dose corresponded to the average daily intake of ketum users in Malaysia" and the highest dose was derived as one tenth of the LD_{50} from Sabetghadam et al. (2013b), which was 1,098 mg/kg-bw via intraperitoneal administration. Protocols were conducted in accordance with OECD guideline No. 407. The authors stated that no overt signs of toxicity were observed among animals at any dose, though piloerection was reported in male and female rats in the 10 mg/kg-bw and 100 mg/kg-bw/day groups "… during the last week of treatment." They added that mild ptosis was observed in both sexes at 100 mg/kg-bw/day. With respect to hematology, red blood cells and white blood cells were decreased in animals dosed

with 100 mg/kg-bw/day, neutrophils were increased in those dosed with 10 mg/kg-bw/day, and platelet count was significantly decreased in animals at all doses relative to controls. A significant increase in liver weight was reported in the 100 mg/kg-bw/day animals relative to controls, though no changes were reported for the kidneys, brain, lungs, spleen, and heart. Following histopathological analysis, adverse effects were noted in the liver and brain at the highest dose (Sabetghadam et al. 2013b)

361. Given the lack of overt signs of toxicity, the study by Sabetghadam et al. (2013b) implied that few adverse effects are expected with repeated doses of mitragynine that exceeded those of human kratom users by up to 10-fold. While some hematologic and hepatic effects were noted at 10 and 100 mg/kg-bw/day, the clinical significance of these changes (especially for blood parameters) is minimal to absent. Further, such changes only occurred at doses several-fold above those expected in human kratom users.

Human Studies

362. La-Up et al. (2021) performed blood exams and collected fasting lipid profiles of 285 regular kratom users and 296 controls in Thailand. Potential confounders, including BMI, alcohol intake, and cigarette smoking status, were controlled for within the study. The authors reported an association between kratom use and elevated HDL (high-density lipoprotein) and low TG (triglyceride) levels, and no difference in TC (serum cholesterol) or LDL (low-density lipoprotein) levels between kratom users and controls. As is commonly known, HDL is considered the "good" cholesterol, and a reduction in triglycerides is generally regarded as a positive effect. Thus, while the real-world relevance of these results is equivocal, the outcome is, if anything, indicative of a positive effect of kratom use on human blood parameters.

363. Leong Abdullah et al. (2021) recruited 100 Malay kratom users and 100 controls to compare the prevalence of ECG abnormalities. Resting ECG demonstrated sinus tachycardia in kratom users, though there were no significant differences in other ECG abnormalities. Additionally, the authors reported that borderline QTc interval (a measurement of heart health) showed no association with kratom use. Leong Bin Abdullah and Singh (2021b) also assessed

cardiovascular functioning among Malay regular kratom users (N=9) by collecting resting ECG, transthoracic echocardiogram, and blood exams. Contrary to their previous study, they identified a dose-dependent prolonged QTc interval.

364. Saengmolee et al. (2022) compared heart rate variability (HRV) indices of cardiac autonomic function in 31 regular kratom users and 19 controls in Thailand. Consumption among users occurred via oral chewing of kratom leaves. The ECG acquisition and HRV analyses revealed ultra-short HRV with normalized low frequency (LFn) and high frequency (HFn) indices in kratom users. Additionally, the authors reported changes in cardiac autonomic function with parasympathetic dominance for kratom users and that there was no significant association between HRV indices and kratom use.

Retrospective Analyses (Krantz et al (2023) and others)

365. Krantz et al. (2023) (relied upon by Drs. Thundiyil, Wharton, and Williams in this case) reported analyzing data provided by the U.S. Food and Drug Administration Adverse Reporting System (FAERS) to assess whether mitragynine was disproportionately associated with reports of ventricular arrhythmia.

366. The authors explained that they queried the FAERS database for simultaneous reports of mitragynine exposure and ventricular arrhythmia and cardiac arrest. Additionally, secondary endpoints included QTc-prolongation and torsade de pointes (a specific form of ventricular tachycardia, or fast heart rhythm) "given the mechanistic potential for proarrhythmia to result from blockade of the human ether-a-go-go (hERG) ion channel, responsible for the delayed rectifier potassium current" (Krantz et al. 2023).

367. It was reported that the authors calculated proportionate reporting ratios to detect signals of disproportionate reporting for arrhythmia events. In doing so, they compared the ratio of arrhythmia events associated with mitragynine and all events associated with mitragynine to that of arrhythmia events associated with all substances in the database and all events associated with the other substances.

368. The authors reported that there were 46 reports of ventricular arrhythmia/cardiac arrest and mitragynine exposure (mitragynine as primary suspect = 16; mitragynine as secondary suspect = 30). Reports of ventricular arrhythmia/cardiac arrest and methadone [prescription opioid, full agonist; positive control], loperamide [nonprescription antidiarrheal agent], buprenorphine [opioid medication used for opioid use disorder, partial agonist, negative control], and naltrexone [prescription medication used for alcohol and opioid use disorders, negative control] totaled to 1,163, 1,008, 429, and 42, respectively.

369. The authors reported that mitragynine exhibited the highest signal for disproportionate reporting of ventricular arrhythmia/cardiac arrest (PRR: 8.9, 95% CI: 6.7-11.7) of the substances analyzed.

370. Conversely, there were only 2 reports of QTc-prolongation/torsade de pointes and mitragynine exposure (mitragynine as primary suspect = 1; mitragynine as secondary suspect = 1). For the other substances of interest, reports of QTc-prolongation/torsade de pointes were 608 for methadone, 755 for loperamide, 96 for buprenorphine, and 22 for naltrexone.

371. Regarding the lack of disproportionate reporting for mitragynine and torsade de pointes, the authors offered two explanations:

> "First, mitragynine is highly protein bound, with variable binding to plasma proteins. Applying the proposed free plasma concentration rather than total results in a substantially greater safety margin … A second possibility is that mitragynine's arrhythmia mechanism might not solely involve QTc-prolongation. This might be analogous to that of propoxyphene (Darvon), which was removed from the market for ventricular arrhythmia despite only 1 case of torsade de pointes in the literature. Propoxyphene is a potent inhibitor of voltage-gated sodium channels, which can promote monomorphic rather than polymorphic ventricular tachycardia. The importance of looking beyond hERG blockade when evaluating opioid proarrhythmic potential has been recently investigated: in addition to inhibiting hERG, methadone equipotently blocked Ik1, the ion channel responsible for

terminal repolarization and U-waves on surface electrocardiography, likely amplifying its risk. Finally, the cardiac ion-channel effects of kratom are likely complex because the available product represents an admixture of > 50 bioactive alkaloids rather than a single pharmacologic entity" (Krantz et al. 2023).

372. Proportional reporting ratios (PPRs) are not risk estimates, such as odds ratios or relative risks, and should be interpreted with caution. A PRR:

"measures a reporting relationship between a medicinal product P and an adverse event R on the basis of a relative increase of the proportion of individual cases related to an adverse event. This does not necessarily imply a causal relationship between the administered medicinal product P and the occurrence of the adverse event R. Such statistical disproportionality may reflect one or more of a number of biases and artefacts inherent in pharmacovigilance data as well as 'statistical noise.' Consequently, there is a scientific consensus that [signals of disproportionate reporting] identified with quantitative methods should always be medically assessed" (European Medicines Agency (EMA) 2006).

373. Additionally, it should be noted that pharmacovigilance databases present their own challenges as a data source. According to the FAERS database disclaimer:

"The FDA Adverse Event Reporting System (FAERS) database houses reports submitted to the FDA by drug manufacturers (who are required to submit these reports to FDA) and other such as health care professionals and consumers. Submission of a safety report does not constitute an admission that medical personnel, user facility, importer, distributor, manufacturer or product caused or contributed to the event. Although these reports are a valuable source of information, this surveillance system has limitations, including the potential submission of incomplete, inaccurate, untimely and/or unverified information. In addition, the incidence or prevalence of an event cannot be determined from this reporting system along due to potential under-reporting of events and lack of

> information about frequency of use. Because of this, FAERS data comprise only one part of the FDA's important post-market surveillance data and the information on this website does not confirm a causal relationship between the drug product and the reported adverse event" (FAERS).

374. Due to the limitations of the study (i.e., data source, statistical method), Krantz et al. (2023) is not relevant to the evaluation of kratom's cardiotoxic potential. Concluding that kratom has the potential to cause adverse cardiovascular effects, as Drs. Thundiyil, Wharton, and Williams did in their opinion letters, is not possible based on the results of this study.

375. Graves et al. (2021) reported conducting a retrospective analysis of data provided by the American Association of Poison Control Center's National Poison Data System and collected from 2014 to 2019. Analyses of cardiological abnormalities associated with kratom use were limited to the calculation of prevalence measures, and no risk estimates were presented. The authors reported that 44.8% of persons aged 18 to 59 years, 36.8% of persons aged 60 to 69 years, and 51.9% of persons aged 70 years or greater experienced cardiovascular clinical effects such as hypertension and tachycardia. No additional information was provided regarding the risk for cardiovascular injury associated with kratom use among the study sample.

376. The retrospective review by Eggleston et al. (2019) highlighted kratom-related toxicities reported to the National Poison Data System (NPDS) and a review of kratom-related fatalities from a county medical examiner's office in New York. A total of 2,312 kratom exposures were reported from 2011 to 2018, increasing from 18 cases in 2011 to 357 cases in the first 7 months of 2018. After excluding multiple substance exposures, 935 cases involving kratom alone were analyzed for toxic effects. The most common adverse effects were agitation (18.6%), tachycardia (16.9%), and drowsiness (13.6%), while some of the most severe adverse effects were seizures (6.1%), withdrawal symptoms (6.1%), and coma (2.3%) (Eggleston et al. 2019). The NPDS data, while useful for identifying clinical effects, did not provide details surrounding kratom use, such as the dosage taken, the duration of use, the motivations for using kratom (e.g., self-treatment of pain, opioid withdrawal, or recreational use), or medical history.

Case Reports

377. Several case reports or series were identified that reported cardiovascular events following kratom use.

378. Dodulik et al. (2024) described the case of an 18-year-old man who collapsed at football practice and was observed to convulse and gasp. They reported that cardiopulmonary resuscitation was performed before the patient with intubated and transferred to the hospital. It was reported that the patient was not taking medication and had no family history of cardiovascular disease. Laboratory testing revealed an elevation of high-sensitivity troponin I (230 ng/L). Toxicology screening returned positive results for caffeine and kratom; the mitragynine concentration was reported to be 98 ng/mL, and no caffeine concentration was provided. It was reported that the patient confirmed consuming a 250 mL energy drink and 2g of kratom powder before the football practice. It was reported that the patient had good left and right ventricular function, no dilatation of the right-sided ventricles, no significant valvulopathy, and no evidence of coronary heart disease, coronary artery anomaly, or structural cardiac involvement. Additionally, no pathogenic sequence variants in genes associated with familial ventricular fibrillation were found; however, a sequence variant was identified in MYOM1 in a heterozygous state, but its clinical significance is unknown. Once discharged, it was reported that the patient stopped taking kratom and had no recurrence of ventricular arrhythmia.

379. The authors concluded that "it is reasonable to assume that, in this particular case, the ventricular fibrillation developed as a result of the combination of kratom, a caffeinated energy drink, and physical exertion." They added that "[t]here is insufficient scientific evidence linking kratom alone to ventricular fibrillation" (Dodulik et al. 2024).

380. Holton et al. (2024) described a case of a 36-year-old man with a history of alcohol abuse, tobacco use (three packs a day; 18-year pack history), opioid use disorder, depression, bipolar disorder, and seizure disorder, who visited the emergency department with chest pain. It was reported that the chest pain, which was described as sharp, intermittent, and radiating from the left shoulder, was accompanied by lightheadedness, palpitations, and syncope. The symptoms

were reported to occur multiple times a day, increasing in duration and frequency over the four weeks preceding presentation. It was reported that the patient had been non-adherent to his lamotrigine prescription for more than three months due to a lack of insurance. The patient reported consuming ~100g of white vein kratom powder a day (4 uses per day) for the past three years to manage withdrawals and cravings. It was reported that the patient had declined Suboxone and had no personal or family history of cardiac dysrhythmia or sudden cardiac death.

381. Upon admission, hypertension and tachycardia were noted; the patient was not in acute distress and was alert, and the physical examination was remarkable for left-sided chest pain that radiated to the clavicle and neck. EKG revealed "tachycardia with a normal QTc and coved ST segments followed by T wave inversions in leads V1 and V2 consistent with type 1 Brugada pattern". Blood testing revealed leukocytosis. The authors concluded that the case had acquired type 1 Brugada syndrome induced by chronic high-dose kratom use. They identified three cases of Brugada syndrome in the setting of frequent high-dose kratom use and stated that "[g]iven the frequency and similarities between the case reports, the link between frequent, chronic, and high-dose kratom use and Brugada syndrome is convincing." They provided no mechanism of action for how kratom use could induce Brugada syndrome, a familial autosomal channelopathy that affects the transmembrane ion current in cardiac cells and increases risk for ventricular arrhythmias and sudden cardiac death, nor how "chronic" and "high-dose" kratom use would be defined (Holton et al. 2024).

382. Miller et al. (2025) provided a case report of a 25-year-old male with anxiety and attention-deficit/hyperactivity disorder who self-medicated with kratom. It was reported that the patient had been ingesting kratom for 4 years [patient's dosing over time was not disclosed], and his estimated daily dosage of kratom prior to presentation was 84 to 100 g. It was reported that the patient presented to an academic tertiary care emergency department after experiencing seizure-like activity, which was preceded by his consumption of kratom by approximately 30 minutes. The dose the patient administered prior to the adverse event was not reported. Previous medical history indicated that the patient vaped menthol nicotine from electronic cigarettes and denied use of

recreational drugs. The authors reported that there was no family history of significant cardiac disease, unexplained drownings, unexpected sudden death, or dysrhythmias.

383. The patient's initial electrocardiogram demonstrated a type-1 Brugada pattern with QRS of 160ms and QTc of 654ms. Briefly, Brugada syndrome (BrS) is "… characterized by idiopathic ventricular fibrillation [abnormal heart rhythm of unknown cause] in association with right ventricular electrocardiogram (ECG) abnormalities without structural heart disease. This disorder is also genetically heterogeneous, although it is most frequently linked to mutations in the Na^+ channel α-subunit, SCN5A" (Kasper et al. 2012).

384. It was reported that his potassium and calcium levels were 3.2 mmol/L and 8.4mg/dL, respectively. According to Harrison's Principles of Internal Medicine, the patient's potassium and calcium levels were low relative to clinical laboratory values (3.5 – 5 mmol/L and 8.7 – 10.2 mg/dL, respectively) (Kasper et al. 2012). The authors reported that there was no evidence of toxidrome, and his neurologic and cardiac exams were normal. It was reported that the patient's kratom was analyzed by gas chromatography mass spectroscopy and liquid chromatography quadrupole time-of-flight, which only detected kratom alkaloids.

385. (Miller et al. 2025) concluded that:

> "[K]ratom was the cause of our patient's cardiotoxicity based on the patient's negative family history of sudden cardiac death, previously unremarkable electrocardiogram, his presenting electrocardiogram, normalization of his electrocardiogram with kratom abstinence, lack of significant electrolyte derangements to explain his electrocardiogram abnormalities, and no structural abnormalities on echocardiography. In addition, there were no contaminants detected in the kratom sample provided by the patient and the urine drug screen was negative" (Miller et al. 2025)

386. However, notably, no blood mitragynine level was reported for the patient described by Miller et al., and the authors did not discuss dose-response, despite the patient's reported dose being very high and the condition being very rare. The patient was also reportedly a

regular kratom user, with a four-year use history. The authors concluded "[k]ratom can cause significant cardiac conduction and repolarizations in a patient without structural anomalies or additional confounding xenobiotics" even though a case report cannot establish causality. While Miller et al. (2025)'s description of the patient's presentation and treatment is a good addition to the literature, it is not sufficient to assert a causal relationship between kratom use and cardiotoxicity.

387. Jain and Lloyd stated that "[s]everal clinical factors, including fever, tricyclic antidepressants, lithium, antihistamines, cocaine, calcium channel blockers, and anesthetics like propofol and bupivacaine, can precipitate spontaneous type 1 BrS ECG changes in individuals with normal baseline."

388. However, Jain and Lloyd interpreted Miller et al. as support of the hypothesis that kratom may additionally precipitate these changes. They further hypothesized that kratom "… disrupts the balance of inward and transient outward currents in the right ventricular outflow tract, which trigger the characteristic surface ECG pattern of BrS, and in the worst case, result in phase 2 reentry and sudden death," before reminding the reader that causality has not been established (p.1). Additionally, Jain and Lloyd introduced the likelihood of a genetic predisposition or subclinical BrS genotype in individuals who exhibit the described herb-induced phenotype.

389. Similar to Miller et al. (2025), Jain and Lloyd's commentary lacks any discussion of a dose-response relationship between kratom use and alleged subsequent cardiac injury apart from one sentence: "[kratom] is relatively mild in potency, which encourages larger doses, thereby increasing the risk of nontarget organ effects" (Jain and Lloyd 2025). Jain and Lloyd do not mention that the patient discussed in Miller et al. (2025) was estimated to take approximately 84 to 100 g of kratom daily. Surely, the authors acknowledge that this magnitude of kratom use is outside the realm of what is generalizable to the general population, even from an economic perspective. Additionally, Miller et al. (2025) stated that the patient's neurologic and cardiac exams were normal, with no evidence of ongoing toxidrome.

390. Chinnappan et al. (2023) described a case of a 36-year-old male, smoker with a history of polysubstance use who presented to the emergency department "… with a 3-day history of progressive pleuritic chest pain and shortness of breath" which the patient reported was a "… sharp pain across his chest with an associated non-productive cough." It was reported that the comprehensive medical history was negative for intravenous drug use, inhalational exposure, hunting or animal exposure, sick contacts, a history of a lymphoproliferative disorder or malignancy, chronic medication use, a recent history of choking or aspiration events, or alcohol use. The authors stated that a chest radiography demonstrated a bilateral infiltrate, and a CT angiography showed no evidence of pulmonary embolism but revealed extensive, bilateral, multifocal consolidative changes.

391. The authors reported that discussions with the family revealed the patient used kratom to alleviate his opioid cravings. It was reported that:

> "Given the rapidly progressive dyspnoea and respiratory failure requiring mechanical ventilation, with no other triggering events identified, negative infectious and cardiology work-up, and imaging findings of worsening bilateral lung opacities, with a PaO2:FiO2 ratio <300 on a PEEP of >5 mmHg, a final diagnosis of kratom-induced acute respiratory distress syndrome (ARDS) was made" (Chinnappan et al. 2023)

392. Chinnappan et al. (2023) did not report any toxicology screening results for the patient, so 1. The presence of mitragynine was not confirmed, and 2. The presence of any substances that could lead to ARDS was not confirmed. Additionally, the patient's kratom use parameters were not described. The patient's treatment plan included intubation, respiratory aid, and supplemental oxygen; however, the authors did not report cessation of kratom use. Furthermore, although the patient was reported to be a current smoker with a 7.5 pack-year history, the authors did not address the role of tobacco as a risk factor for acute respiratory distress syndrome.

393. Torres-Ortiz et al. (2022) documented the case of a 61-year-old male with a history of degenerative lumbar disc disease and hyperlipidemia [high levels of fats in blood] who presented to a nephrology clinic for unexplained persistent hyperkalemia that was noted to be asymptomatic. It was reported that he was prescribed rosuvastatin [a statin medication], and he denied nonsteroidal anti-inflammatory drug, antibiotic, and illicit substance use as well as smoking and drinking alcohol.

394. It was reported that when the patient visited the referring clinic two months prior to visiting the nephrology clinic, his serum potassium levels were between 5.6 and 5.8 mmol/L. At the time of his visit to the referred clinic, he had been on a strict low potassium diet, and his serum potassium level had dropped to 5.3 mmol/L. The authors reported that the reference range for potassium was 3.5-5.1 mmol/L, showing that while his serum potassium level was still elevated from normal, it had decreased since his previous visit. It was reported that his other laboratory results were normal, except for a mild increase in liver enzymes.

395. The authors reported that:

> "Upon re-evaluation, the patient reported using kratom for recreational purposes almost on a daily basis for the past four months. One month after kratom discontinuation, his blood chemistry revealed normalization of the potassium level" (Torres-Ortiz et al. 2022).

396. However, the authors reported that "[h]is serum potassium level improved slightly on a strict low-potassium diet as instructed before his presentation to our clinic," which is evidenced in the reduction described above (Torres-Ortiz et al. 2022).

397. Torres-Ortiz et al. (2022) suggested that kratom was the cause of the patient's hyperkalemia but never measured the levels of mitragynine in the patient's blood. Additionally, since the patient's serum potassium levels began to lower with adherence to the low potassium diet while he was presumably still taking kratom, it is not possible to know if continuing use would have had any effect on his potassium level given the first intervention. Kratom dosing, including in the context of a dose-response relationship, was not discussed by the authors.

398. Patel et al. (2021) reported the case of a 28-year-old male with a history of alcohol and opiate abuse and "… questionable seizures" who presented to the emergency department after being found unresponsive. It was reported that the patient had no significant surgical history, allergies, or family history, and his only medication was pantoprazole for reflux.

399. According to the authors, the patient received Narcan, intranasal and intraosseous, which he did not respond to, so he was intubated in the field. It was reported that upon emergency room admission, a head CT showed no acute intracranial bleeding. The physical exam was notable for myoclonic jerking, but the patient's pupils, heart sounds, rate, and rhythm, and lungs were normal, and there was no distention of the abdomen or edema in the extremities observed.

400. It was reported that urinalysis revealed "amber, turbid urine with large blood and 100 mg/dL protein" and that the urine toxicology results were negative for amphetamines, barbiturates, benzodiazepines, cocaine, cannabinoids, and opiates. According to Patel et al. (2021), the patient was admitted to the intensive care unit for ventilator-dependent respiratory failure, acute renal failure (ARF), severe metabolic acidosis, rhabdomyolysis, and shock liver.

401. The authors described the patient's complicated clinical course, which included placing an emergent central line to address hypotension resulting in an iatrogenic pneumothorax and chest tube placement, worsening renal failure and persistent hyperkalemia, elevated creatine kinase and liver enzymes, and hemodialysis to manage ARF from acute tubular necrosis secondary to severe rhabdomyolysis. It was reported that a transthoracic echocardiogram showed severely reduced left ventricular systolic function with an ejection fraction of 31-35% and a brain MRI showed multifocal acute infarcts involving the posterior parietal lobes bilaterally and left basal ganglia. A transesophageal echocardiogram was reported to have shown no cardiac source of emboli. Additionally, it was reported that the patient experienced methicillin-resistant Staphylococcus aureus (MRSA) pneumonia, which was addressed with intravenous fluids and antibiotics. It was reported that the results of a LexiScan nuclear stress test demonstrated normal cardiac contractility and ejection fraction, and that the patient's cardiac function had been restored.

Following several rounds of dialysis, it was reported that the patient's mental status resolved, but his renal function did not, and a permanent hemodialysis catheter was placed.

402. The authors stated:

> "Further inquiry from his girlfriend and family revealed that the patient had been abstinent from drugs for the past 10 years; however, he relapsed one week ago after his father passed away. Patient had started using kratom one week prior to presentation… Subsequent urine test results several days later confirmed the presence of high levels of mitragynine" (Patel et al. 2021).

403. The authors did not provide the quantitative value of mitragynine detected in the patient's urine. They also did not provide any further details regarding the patient's kratom use (i.e., dose) or how kratom could cause this type of clinical presentation; however, they concluded that the "… patient had transient nonischemic reversible cardiomyopathy due to the toxicity of kratom."

404. Abubaker et al. (2025) presented a case report of a 27-year-old male with a history of opioid abuse who experienced a tonic-clonic seizure following kratom ingestion. They reported that the seizure occurred approximately one to two minutes after ingestion of 6 kratom extract tablets (of unspecified mitragynine concentration), and the patient had an estimated daily kratom usage of 120-140 grams per day. It was reported that an ECG showed a Brugada pattern, which was reportedly not present one year prior, and the patient denied any family history of cardiac death or previous seizures. In addition, the authors noted that the dose the patient consumed "… was approximately 3 times his usual intake …" (Abubaker et al. 2025). As with other case reports, a full medical history of the patient was not available.

405. Sangani et al. (2021) reported a case of a 45-year-old female with a history of Crohn's disease, breast cancer (status post chemotherapy), radiation one year prior, bilateral mastectomy with reconstruction two months prior, and chronic pain who was brought to the emergency room following an episode of syncope. It was reported that she suffered from a burn injury due to heating pad use for postoperative pain, for which she reported taking kratom one

month prior to presentation. Her initial daily intake of kratom was reported to be 6 capsules [size unknown], increasing to more than 10 pills per day over the course of two weeks. Two days prior to admission, it was reported that the patient took "… extra kratom pills" while experiencing body aches, specifically on the right side, fatigue, and weakness. It was reported that the patient took "… 2 more extra pills" eight hours prior to admission, but the total dose consumed on the day of admission was not reported. The authors stated that the patient passed out for six hours, after which she was drowsy, had worsening right lower extremity pain and swelling, difficulty walking, and hearing loss, so she called Emergency Medical Services.

406. In the emergency room, the patient was reported to have significant swelling of the lower right extremity with tenderness, weakness on the right side, and decreased right-sided hearing. It was reported that a brain CT scan showed no acute findings, a neck CT scan revealed a nonspecific inflammatory change in the sternocleidomastoid muscle, and a CT scan of the thorax, abdomen, and pelvis showed pneumonia and probable colitis. The authors stated that the patient was diagnosed with compartment syndrome of the right lower extremity and developed hypoxia with gross cyanosis of the upper body and severe rhabdomyolysis, which caused acute kidney injury from acute tubular necrosis complicated with severe hyperkalemia. Following dialysis and multiple debridements, the patient was reportedly discharged with home health care.

407. Sangani et al. (2021) stated that "[s]tudies show the adverse effects of kratom are heavily dose-dependent," yet they did not attempt to estimate the dose of kratom self-administered by the patient in the week or day of hospital admission. They also did not describe any toxicology testing outside of a nondescript urine drug screen, which was stated to be negative. No blood mitragynine concentration was reported.

408. Nonetheless, the authors hypothesized that kratom alkaloids and metabolites "… can cause muscle injury by acting through the α-adrenergic receptors as this pathway can cause vasoconstriction leading to muscle ischemia and rhabdomyolysis," citing Maduhusoodanan et al. 2004. Madhusoodanan et al. (2004) did not discuss kratom or its alkaloids and instead described a

case of rhabdomyolysis following intravenous heroin use with evidence of illicit polypharmacy, undermining their postulation.

409. Sheikh et al. (2021) documented a case of a 44-year-old male with a history of hypertension and hyperlipidemia, both reportedly controlled with medical therapy, who presented to the emergency room following a cardiac arrest. According to the authors, the patient's family reported that he had recently started taking "Premium Maeng Da Kratom" in addition to his "… chronic …" ingestion of 2 to 8 Monster energy drinks daily. Based on this information, the patient's total daily caffeine intake would equate to 172 mg to 688 mg. No kratom use parameters or blood mitragynine levels were reported, so no dose preceding the adverse event could be ascertained, and the presence of mitragynine in the patient was not toxicologically confirmed.

410. It was reported that in the emergency room, the patient's potassium was below normal ranges, urine toxicology was positive for ethanol, X-ray showed pulmonary vascular congestion, no acute pathology was found on head CT, and his coronary arteries were normal. According to the authors, the patient was intubated due to hypoxic respiratory failure. It was reported that an ECG was performed, which revealed a prolonged QT interval, intraventricular conduction block, and normal global left ventricular function without dilation or valvular abnormalities.

411. The patient's differential diagnoses reportedly included myocardial infarction with non-obstructive coronary arteries, idiopathic ventricular arrhythmia, and cardiomyopathy secondary to infiltrative disease (hemochromatosis, amyloidosis). It was reported that the patient was treated with amiodarone for heart rhythm control and antibiotics for aspiration pneumonia.

412. Even though very little information regarding the patient's kratom use was discussed by Sheikh et al. (2021), the authors concluded that "[w]hile [kratom] has opiate-like properties, there is a potential for QT prolongation which can pre-dispose healthy individuals to ventricular arrhythmias". Regarding the contribution of the patient's chronic and heavy consumption of energy drinks, the authors stated that a "… definitive causal relationship [between energy drinks and cardiac events] has yet to be identified" (Sheikh et al. 2021). Causal attribution

of kratom to ventricular fibrillation remains weak, particularly in the presence of confounding by excessive caffeine intake.

413. Abdullah et al. (2019) presented the case of a 35-year-old male with a past history of substance abuse who was found unresponsive and not breathing at his residence. It was reported that cardiopulmonary resuscitation (CPR) was performed, return of spontaneous circulation occurred after 2 cycles of CPR and epinephrine, and the patient was intubated in the field. It was noted that his substance use history included alcohol, benzodiazepines, opioid pain medications, marijuana, and methamphetamine; however, the patient was reported to have been through incarceration and rehabilitation and denied using illicit substances prior to his admission.

414. Following admission, the patient was reported to have had abnormal laboratory test results, left ventricular systolic dysfunction with a left ventricular ejection fraction of 20%, which the authors attributed to his recent cardiac arrest, and a negative urine drug screen.

415. According to the authors:

> "[T]he EMS and police reported they had found a large amount of kratom on the patient. After extubation, the patient [admitted] to using kratom for his opioid dependence. He obtained a large quantity of kratom in the form of powder from a local Vape store. The powder was used to make tea, and he drank it numerous times a day" (Abdullah et al. 2019).

416. No additional information regarding the patient's kratom use was provided, and no blood mitragynine concentration was reported.

417. Over 48 hours of observation in the intensive care unit, the patient's symptoms of hypotension, shock, acute kidney injury, acidosis/lactic acidosis, and rhabdomyolysis were reported to have resolved. The authors stated, "[i]n our case, it was more likely that the opioid-like properties of kratom led to respiratory depression and cardiac arrest"; however, mitragynine has not been shown to recruit β-arrestin, part of the β-arrestin 2 signaling pathway that is responsible for the respiratory depressive effects of traditional opioids (Abdullah et al. 2019; Heywood et al. 2024).

418. Aggarwal et al. (2018) described the case of a 26-year-old male who presented to the emergency department in cardiorespiratory arrest, having taken an unknown quantity of kratom 24 hours before presentation. According to the authors, the arrest was primarily characterized by pulseless electrical activity, with a brief period of ventricular arrhythmia noted. It was reported that the patient had no medical history or regularly prescribed medications prior to admission. Urine toxicology was negative for the compounds analyzed, except for codeine, which the authors stated was taken at a standard dose prior to his admission. The arrest was reportedly treated following the standard protocol, sodium bicarbonate was administered for metabolic acidosis, and naloxone was given per the advice of the National Poisons Information Service. It was reported that return of spontaneous circulation occurred after one hour with significant inotropic and vasopressor support, and a large ventilatory shunt was noted. It was reported that a CT scan showed imminent cerebral herniation. The authors stated that the patient underwent hemodiafiltration for metabolic acidosis and received lipid emulsion therapy, following which his cardiovascular and respiratory failure improved for about one hour. It was reported that despite multi-organ treatment, the patient's cardiorespiratory function deteriorated once again, the patient's family decided to withdraw active treatment, and he passed away 12 hours after the initial return of spontaneous circulation was achieved due to cardiorespiratory failure and hypoxic brain damage.

419. Aggarwal et al. (2018) did not indicate that mitragynine was toxicologically screened for, and the dose taken prior to admission appeared to be unknown. However, the authors reported that the cardiorespiratory arrest was attributed to kratom ingestion by the clinical team and coroner. Because the dose of intralipid yielded a positive effect, allowing medical personnel to reduce the dose of inopressor by 30%, and there was improvement in oxygenation and ventilation observed, the authors postulated, "that a proportion of the cardiovascular collapse was therefore a result of cardiotoxicity of the Kratom … [and] that at least a proportion of the compounds are cardiotoxic".

420. Wolfer et al. (2020) described the case of a 20-year-old man who was reported to have abruptly experienced seizures followed by developing bradysystolic cardiac arrest. It was

reported that the patient had a history of ADHD and was attending a state-mandated drug monitoring program for weeks. The authors stated that the patient reported using "a commercially prepared kratom tincture for intoxication without triggering a drug screen," self-administering one bottle per day. It was reported that the patient had consumed a bottle of the kratom tincture within an hour before seizing. According to toxicology testing, the patient was reported to have a mitragynine blood concentration of 560 ng/mL (urine: 2300 ng/mL). Additionally detected were amphetamine, caffeine, acetaminophen, ibuprofen, and cotinine, all of which were determined not to explain the patient's presentation.

421. The authors concluded:

> "Though other explanations such as undiagnosed seizure disorder, inhalants, or withdrawal from benzodiazepines or alcohol are possible, these are unlikely given the patient's medical history and prolonged abstinence from routinely tested drugs. Our case details the moments leading up to cardiac arrest, and implies that a possible etiology of kratom-related death may be status epilepticus and its associated respiratory failure with metabolic acidosis" (Wolfer et al. 2020)

422. Peran et al. (2023) reported that among 10 cases of kratom poisoning, the overdose toxidrome appeared opioid-like in that individuals were reported to have experienced hypertension, tachycardia, mydriasis, miosis, vomiting, bradycardia, and impaired consciousness. Cases reported using kratom tea, tablets, or powder orally and overdose symptoms manifested 1 to 24 hours following use. All cases were young adults using kratom recreationally, and dosage was not provided. Importantly, urine toxicology screening demonstrated that 70% of cases tested positive for other substances; therefore, the contribution of kratom to the patients' symptoms is unknown.

Review Papers

423. Begum et al. (2024) reported conducting a review of the literature to compile toxicity data on kratom from *in vitro*, *in vivo,* case studies, and surveys. All studies relevant to

cardiotoxicity cited by Begum et al. have been discussed independently above. Based on their review, the authors concluded:

"[T]he numerous scientific studies indication that through kratom … has several ethnopharmacological usages and pharmacological potential, its toxicity profile remains insufficiently understood. The gaps in our understanding persist due to the limited number of toxicological studies available which indicate potential risks, including hepatotoxicity, neurotoxicity and cardiotoxicity, specifically with high-dose or long-term of its consumption" (Begum et al. 2024)

424. Grundmann et al. (2023) conducted a review to summarize the history, pharmacology, user trends, adverse effects, and potential benefits of kratom. Regarding non-opioid pharmacology, Grundmann et al. (2023) reported that mitragynine exhibits low binding affinity at 5-HT1A (Ki = 5.88 uM) and 5-HT2b (Ki = 1.26 uM) and suggested that modulation of 5-HT2B receptors may lead to abnormal cardiovascular activity; however, the authors did not evaluate the ability of mitragynine to modulate this serotonergic receptor. Grundmann et al. (2023) reported that in reviewing case reports, kratom appeared to be associated with monomorphic or polymorphic ventricular dysrhythmias. They stated:

> "Polymorphic ventricular tachycardia may be related to kratom's effects on the QTc interval. A prolonged QTc interval has been reported in several cases of kratom exposure. Mitragynine and several other alkaloids present in kratom inhibit the outward delated rectifier potassium current (I_{kr}) in a dose dependent manner. Although regular users of kratom do not have higher rates of prolonged QT than control groups, the risk of QT prolongation is strongly correlated with recent use, higher daily doses, and elevated serum mitragynine concentrations, suggesting a dose-dependent effect even in chronic regular users. The prolongation of QTc had been theorized, though not proven, to the be the cause of polymorphic ventricular tachydysrhythmias (Torsade de Point) and cardiac arrest" (Grundmann et al. 2023)

425. Chichagi et al. (2024) conducted a review of the literature to assess the cardiovascular effects, including cardiotoxic potential, of mitragynine in humans. The authors compiled 11 case reports, four cross-sectional studies, two case-control studies, one case series, three retrospective studies, and one cohort study.

426. They reported that the most prevalent cardiovascular symptoms experienced by kratom users were tachycardia, hypertension, and abnormal lipid profiles, and that they identified case reports of arrhythmias, cardiac arrests, and heart failure among kratom users. The review was unable to assert an association between kratom use and cardiovascular events.

427. The authors concluded that "… kratom consumption might have adverse effects on the cardiovascular system at higher blood levels. However, low doses seem to be safe. Additionally, there is no consensus on the safe consumption duration, or threshold dose, threatening the cardiovascular system" (Chichagi et al. 2024).

428. This review did not present a risk estimate for users or define use parameters (i.e., dose, duration, frequency) that may increase a kratom user's risk of cardiac injuries. Additionally, the review is limited by the study designs included, such as cross-sectional studies, which are unable to establish temporality between exposure and outcome, and case reports, which lack a control group for comparison.

Conclusions on Cardiovascular Toxicity

429. Based on the available literature, most instances of kratom-related cardiac injury were only published in case reports. The majority of these case reports presented limited information regarding the patient's kratom use (i.e., no dose estimation), did not toxicologically confirm the presence of mitragynine in their respective patients, and concluded that kratom was the cause of the various injuries without providing sufficient evidence.

430. It is my opinion that, based on the available literature I have reviewed, kratom has not been shown to increase the risk of serious cardiovascular events in humans. Additionally, if Ms. Moller is found to have had a heart attack or other cardiovascular event, then it would not have been related to her use of kratom.

Basis of Opinion 8

431. Based on the case materials reviewed, there is no available information regarding Ms. Moller's kratom usage or dose parameters; however, based on the Coroner's Report, Ms. Moller's postmortem blood mitragynine concentration was 150 ng/mL (Coroner's Report: pg. 16). While it is impossible to know whether that concentration of mitragynine was representative of her antemortem levels, 150 ng/mL is a blood concentration that has been observed in human volunteers ingesting dose of kratom that are accepted to be well-tolerated in humans.

432. As previously discussed at length, Huestis et al. (2024) found that human volunteers given a single dose of mitragynine (via kratom) of 53.2 mg showed an average maximum mitragynine blood concentration of 125 ng/mL (with a standard deviation of 51.8, indicating that several volunteers had blood concentrations greater than 150 ng/mL). As previously discussed, a study conducted by the FDA found that mitragynine doses of approximately 80 mg were well-tolerated in humans, with no adverse effects, and that doses of up to 120 mg of mitragynine were also well-tolerated, except for some cases of transient nausea (Reissig et al. 2024).

433. While it is impossible to know what exact dose of kratom Ms. Moller used in the 24 hours prior to her passing, or in the previous months or years, the blood concentration of mitragynine found in the Coroner's Report is comparable to those found in human volunteers following ingestion of kratom in doses documented to be well-tolerated in humans with no serious adverse effects. In fact, it is well documented in the literature that typical kratom doses are many times lower than thresholds expected to cause adverse health effects in humans.

Estimated Alkaloid Doses from Kratom Products

Kratom Use Data

434. Smith et al. (2022) assessed the current state of kratom dosing, changes in routines (based on kratom use), and perceived effects, including onset timing, duration, and variability of beneficial and adverse outcomes from use and cessation. The survey study was a follow-up to a prior online survey that evaluated substance use and social conditions. The authors recontacted all

surveyors who self-reported lifetime kratom use (n = 289) and obtained additional information related to kratom during a one-month period from April 15 to May 15, 2021 (Smith et al. 2022).

435. Typical dose was measured by asking respondents to identify the kratom formulation they most frequently consumed (e.g., capsule, gram, spoonful, tablespoon, cups of tea, etc.), the number of said uses per day, the number of products per use, and the length of time for which the respondent consumed that typical daily amount in weeks, months, or years (Smith et al. 2022). It was determined that respondents most frequently reported consuming kratom via capsules (n = 47), then grams (n = 37), spoonfuls (n = 25), tablespoons (n = 11), and finally cups of tea (n = 8). The average amount of kratom used per dose was 5.4 ± 4.8 capsules, 4.6 ± 3.6 grams, 2.5 ± 2.7 spoonfuls, 2.1 ± 1.0 tablespoons, or 1.6 ± 1.1 cups of tea. The average number of doses taken per day was 2.6 ± 2.4, and the typical dosing routine was stable for 65.0 ± 112.9 weeks (Smith et al. 2022).

Methods

436. Based on the data from Smith et al. (2022) and the assumption of a 70 kg human weight, we estimated potential doses among typical United States kratom users using the formula below:

437. In employing a conservative approach (likely to overestimate the true risk), upper-end estimates of mitragynine concentrations reported in the literature were relied on in the following calculations.

Estimated Doses

Leaves

438. It was assumed that mitragynine content ranged from 0.8 to 26.6 mg per gram of dry leaf based on data from Kikura-Hanajiri et al. (2009) (commercial products) and Leksungnoen et al. (2022) (leaves cut in Thailand), respectively.

439. The average amount of kratom used per dose was assumed to be 4.6 grams based on Smith et al. (2022)

Low-end intake per day (Estimate)

$$= \mathbf{0.14} - \boldsymbol{bw/day}$$

High-end intake per day

$$= \mathbf{4.54} - \boldsymbol{bw/day}$$

Teas/Juices/Drinks

440. A mitragynine content of 0.006% to 0.74% by weight was assumed for 360 ml containers of kratom tea based on the results of Singh et al. (2018b) for tea derived from fresh leaves and Sharma et al. (2019)for lyophilized tea, respectively.

441. Based on the above, the mitragynine concentration range was assumed to be 0.06 to 7.4 mg/g.

442. The average dose of kratom tea was assumed to be 1.6 cups based on Smith et al. (2022), translating to 576 grams of product per use (1.6 servings x 360 ml glasses).

Fresh Leaf Estimate

$$= \mathbf{1.28} - \boldsymbol{bw/day}$$

Dry Leaf Estimate

$$= \mathbf{158} - \boldsymbol{bw/day}$$

Powders

443. A mitragynine content of 0.088 to 21.40 mg/g was assumed based on Kikura-Hanajiri et al. (2009); Fowble and Musah (2019); Prozialeck et al. (2020) and Fleming et al. (2023).

444. The average amount of kratom used per dose was assumed to be 4.6 grams based on Smith et al. (2022).

Low-End Estimate

$$= \mathbf{0.015} - \boldsymbol{bw/day}$$

High-End Estimate

$$= \mathbf{3.66} - \boldsymbol{bw/day}$$

Capsules

445. 500 mg capsules were assumed to contain from 3.35 mg/g mitragynine to 38.74% mitragynine by weight (i.e., 387.4 mg/g) based on Prozialeck et al. (2020) and Sharma et al. (2019), respectively.

446. The average amount of kratom used per dose was assumed to be 5.4 capsules based on Smith et al. (2022).

447. Assuming that an average capsule is 500 mg, the average mass consumed per use was 2.7 grams (5.4 x 0.5 grams).

Low-End Estimate

$$= \mathbf{0.34} - \boldsymbol{bw/day}$$

High-End Estimate

$$= \mathbf{38.85} - \boldsymbol{bw/day}$$

Liquid Extracts

448. As illustrated below, various marketed kratom extract products advertised different dosing procedures, along with varying mitragynine concentrations. The range of approximate mitragynine concentration was estimated to be 2.03 to 650 mg/g, and the serving size ranged from approximately 0.05 to 29.5 grams.

- Based on manufacturing data from CBD Kratom Extract, it was assumed that mitragynine content was 13.2 mg/g (33 mg/2.5 g serving).
    - Based on manufacturing data from CBD Kratom Extract, an average extract serving was assumed to be approximately 2.5 grams (15 ml, or 6 servings, per container) (CBD Kratom Cannabis Dispensary 2024).
- Other websites advertised kratom products containing 60 mg mitragynine per serving. The products were advertised to hold 59 ml and 2 servings.
    - Based on this information, it was assumed that an average extract serving was approximately 29.5 ml. Therefore, the mitragynine concentration was approximately 2.03 mg/g (Super Speciosa Raw Leaf 2024b).
- Another manufacturer website detailed that the "MIT45 GO" product, which was a gel pouch, contained 150 mg of extract with a 65% mitragynine concentration. The listed products were reported to have 3 servings in each shot (Croxford 2024).
    - Based on the above information, it was assumed that each serving contained 50 mg total and 32.5 mg mitragynine (650 mg/g).
- Another product sold, known as an "MIT45 shot," which was a liquid, contained 250 mg of extract with a 45% mitragynine concentration. The listed products were reported to have 3 servings in each shot (Croxford 2024).
    - Based on the above information, it was assumed that each serving contained 83.33 mg total and 37.5 mg mitragynine (450 mg/g).
- Collectively, the above data mean that, on a per serving basis, the MIT45 GO product contained the least mitragynine while the Super Speciosa Kratom Extract contained the most mitragynine. This is reflected in the estimates below.

Low-End Estimate

$$= \mathbf{1.21} - \boldsymbol{bw/day}$$

High-End Estimate

$$= 2.22 - bw/day$$

Comparing the Estimated Dose to the Toxicological Data

449. The estimated doses for all consumption types ranged from 0.015 to 158 mg/kg-bw/day, where the highest dose was achieved through the consumption of kratom tea. The lowest lethal dose observed in single- and repeated-dose *in vivo* studies in mice using subcutaneous, intraperitoneal, and oral routes of administration ranged from 200 to 1,098 mg/kg-bw (Grewal 1932b; Macko et al. 1972; Sabetghadam et al. 2013a; Singh et al. 2019; Chakraborty et al. 2021).

450. Taking a conservative approach by applying the lowest observed lethal dose (200 mg/kg-bw), the estimated doses are between 1.27- and 13,333-fold lower than expected lethal doses. If the upper estimate of observed lethal dose is considered, the estimated values are between 6.95- and 73,200-fold lower than expected lethal doses. Specific to the oral route of administration, which is the most relevant, these estimates are approximately 3- to 50,000-fold below the LD_{50} values for mitragynine *in vivo.*

451. Our estimates (0.015 to 158 mg/kg-bw/day) generally fell within the range of mitragynine doses reported to yield few to no adverse effects in single- and repeated-dose *in vivo* studies in mice, rats, and dogs (Macko et al. 1972; Sabetghadam et al. 2013a; Sabetghadam et al. 2013b). Macko et al. (1972) reported few to no adverse effects following oral administration of mitragynine at 5 to 50 mg/kg-bw/day, and Sabetghadam et al. (2013b) reported no overt signs of oral toxicity at 1, 10, and 100 mg/kg-bw/day, apart from piloerection and increased liver weight at 10 and 100 mg/kg-bw/day. This indicates that some margin of safety should be present between mitragynine concentrations ingested by the majority of United States kratom consumers and mitragynine concentrations expected to yield adverse effects with acute or chronic use. While it is unknown how much kratom Ms. Moller ingested in the day or days prior to her passing, typical kratom doses would not result in adverse health effects, including cardiovascular health effects.

CLOSING COMMENTS

452. I submit these opinions to a reasonable degree of scientific certainty and am prepared to support them in both deposition and/or courtroom testimony. I may supplement this report if additional information becomes available or if I am asked to address other issues.

/s/ Dennis J. Paustenbach December 18, 2025

Dennis J. Paustenbach, Ph.D., DABT, CIH Date

Chief Scientist, TRC Strategic Health Sciences



DENNIS J. PAUSTENBACH, PH.D., C.I.H., D.A.B.T.

EDUCATION

Ph.D., Environmental Toxicology, Purdue University, 1982
M.S., Counseling Psychology, Indiana State University, 1978
M.S., Industrial Hygiene (minor in toxicology), University of Michigan, 1977
B.S.Ch.E., Chemical Engineering, Rose-Hulman Institute of Technology, 1974

- Post-Doctoral Research (in absentia), Pharmacokinetics, Wright-Patterson Air Force Base, Dayton, OH; 1985-1987 (part-time) (collaborated with Dr. Melvin Andersen).
- Visiting Scientist at the Harvard School of Public Health; Center for Risk Analysis; Boston, MA (1996-1997). A one-year appointment that included 10 weeks in residence at the college. Worked with Dr. John Graham.
- Dr. Eng., (Honorary), Rose-Hulman Institute of Technology, 2007
- Sc.D., (Honorary), Purdue University, 2006

PROFESSIONAL REGISTRATIONS/CERTIFICATIONS

- Certified Industrial Hygienist, #1815 (re-certified 1987, 1993, 1998, 2003, 2008, 2013, and 2018). Recertification occurred in 2023.
- Certified Safety Professional, #5954 (re-certified 1987, 1996, 2005, 2012, 2018, 2024) Registered Professional Engineer-in-Training (1974)
- Diplomate of the American Board of Toxicology, 1984, #253 (re-certified 1992, 1996, 2000, 2005, 2010, 2016 and 2022)
- Certified Environmental Assessor, California, 1991, REA #01264 (re-certified 1995, 2000 and 2008; allowed certificate to lapse)
- Fellow of the Academy of Toxicological Sciences (1993; re-certified in 1998, 2003, 2008, 2013, 2018, 2020, and 2025)

AREAS OF EXPERTISE

Dr. Dennis J. Paustenbach, Ph.D., C.I.H., D.A.B.T., has program management experience, significant subject matter expertise, and has testified in court as an expert in the following general areas:

- Toxicology
- Health Risk Assessment
- Industrial Hygiene
- Exposure Science
- Chemical Engineering
- Classic Industrial Safety
- Environmental Regulatory Affairs

REPRESENTATIVE EXPERIENCE

Dr. Dennis Paustenbach is a board-certified toxicologist and industrial hygienist with nearly 40 years of experience in risk assessment, environmental engineering, toxicology, safety and occupational health. Since August of 2024, he has served as the section head of the risk sciences division of TRC Engineering, a global engineering firm of 9,000 persons.

From April 2019 until July 2024, he was the President and senior consultant of Paustenbach and Associates. The firm was devoted to Dennis's historical interests in toxicology, occupational hygiene, regulatory affairs, engineering, and risk assessment. The firm had about 22 staff scientists located in three geographical locations.

In 1985, he founded ChemRisk, which ultimately became the largest human and ecological risk assessment consulting group in the U.S. They specialized in the risk analysis of chemicals and radionuclides in air, water, soil, sediments, consumer products, pharmaceuticals, and medical devices from 1985- 2019 (except for the years at Exponent).

Initially, in 1987, ChemRisk was a joint venture with McLaren-Hart Environmental Engineering. In 1995, Dennis became President and Chief Executive Officer (CEO) of McLaren-Hart, a nationwide consulting firm of 600 persons (number 100 in the US at the time). He was the youngest professional (age 43) in the U.S. to head a national environmental firm, which at that time had $700M (revenue).

In 1997, Dennis joined Exponent and was a Group President responsible for leading the Health Sciences division (approximately 140 professionals). In 2003, he left Exponent and he restarted ChemRisk as a separate enterprise.

In 2015, the management team sold the firm to Cardno, an Australian based environmental services firm. He was President of Cardno-ChemRisk from 2015 until he left the firm in April 2019. ChemRisk was the largest provider of risk assessment services in the U.S and Australia.

Dr. Paustenbach has provided expert witness testimonies in public meetings, and as many as 700 depositions and six dozen trials concerning the health effects of chemicals in sediments, air, soil, consumer products, foods, groundwater, and the workplace. He has been an adjunct professor at six universities.

He has been an invited peer reviewer for prominent journals and for proposed regulations. Dennis has published approximately 300 peer-reviewed articles and has written more than 50 book chapters. His three textbooks on risk assessment are among the most popular that have been published in this field, and they have been adopted by a number of universities in various countries. The third edition, entitled, Human and Ecological Risk Assessment: Theory and Practice, was released in April 2024.

He recently served as the lead editor of the 7th edition of Patty's Toxicology (2024); the classic seven volume reference text (nearly 8,000 pages). His publications have been cited 3,000 times by other researchers.

PROFESSIONAL HONORS/AWARDS

- In 2025, he was awarded the Herb Stokinger award for lifetime contributions to the field of occupational toxicology by the Amer Conf of Governmental Industrial Hygienists (ACGIH). The lecture was delivered on Oct. 29, 2025, to several thousand members.
- In 2024, he was given the Shubik Award by the Toxicology Forum. This is awarded to persons who have significantly impacted the toxicology profession during their lifetimes.
- In 2024, he was ranked in the top 1% of the most productive environmental scientists in America by Stanford University
- Was recognized as one of the top five consulting professionals within Cardno (a firm of 5,000 employees) (2018)
- Gave the Dr. John Doull Annual Lecture at the Medical School at the University of Kansas for "How risk assessment can help inform decision making: Four case studies," Kansas City, MO, September 2017, since then, I have been an adjunct professor of medicine in that department.
- Awarded in 2016 the Best Published Paper for "A preliminary evaluation of immune stimulation following exposure to metal particles and ions using the mouse popliteal lymph node assay," (Tox Appl Pharm. 308:77-90). Medical Device and Combination Product Specialty Section – Society of Toxicology Annual Meeting, Baltimore, MD, March 2017.
- Awarded in 2014 the Best Published Paper Regarding Medical Devices for "Toxicology - based cancer causation analysis of CoCr-containing hip implants: a quantitative assessment of genotoxicity and tumorigenicity studies" (J Appl Tox. 34(9): 939-967. Medical Device and Combination Product Specialty Section – Society of Toxicology Annual Meeting, San Diego, CA, March 2015).
- Honored by Purdue School of Health Sciences as one of the most accomplished graduates in the history of the program (2015)
- Elected a "Fellow" in the Society for Risk Analysis (2012)

- Awarded the Henry Smythe Award for contributions in toxicology to the field of occupational health, American Conference of Governmental Industrial Hygienists (ACGIH) (2010)
- Awarded the Ed Baier Award for significant contributions to the field of Industrial Hygiene, American Industrial Hygiene Association (AIHA) (2009)
- Invited to lecture at the International Conference on Risk Assessment, Lugano, Switzerland (2009)
- Awarded the title of "Fellow" by the American Industrial Hygiene Assoc (2009)
- Honorary Doctor of Engineering (2007)
- Honorary Doctor of Science (2006)
- Society of Toxicology, awarded the Best Published Paper Award in Risk Assessment (2002)
- Society of Toxicology, awarded the Arnold J. Lehman Award for Contributions to the Practice of Risk Assessment and Toxicology (2002)
- Awarded the Purdue University Distinguished Alumnus Award, School of Health Sciences (2000)
- Society of Toxicology, awarded the Best Platform Presentation in Risk Assessment Award at the 39th Annual Meeting (2000)
- Society for Risk Analysis Outstanding Risk Practitioner Award (1997)
- Society of Toxicology, Best Published Paper Award in the Field of Risk Assessment (1995)
- Rose-Hulman Institute of Technology, Distinguished Alumni Award (1994)
- AIHA Kusnetz Award for "distinguished contributions to the field by a person under 40 years of age" (1992)
- American Men and Women in Science (1993–present)
- Who's Who in America (1993–present)
- Who's Who in the Health Sciences (1994–present)
- Health Sciences Honorary (Alpha Chi Omega) (1980)
- Chemical Engineering Honorary (Omega Chi Epsilon) (1974)

GOVERNMENT GRANTS AWARDED

- 1982: NIOSH grant to study low volume/high velocity ventilation systems. Award: $60K (Purdue University).
- 1981: Grant to write an industrial hygiene engineering course for NIOSH. Award: $60K (Purdue University). Was used by NIOSH for almost 20 years.
- 1980: NIOSH grant to develop teaching modules for use in undergraduate engineering classes. Award: $80K (Purdue University)

SECURITY CLEARANCE

- Q-level Security Clearance issued by the U.S. Department of Energy for 1991-1993 [Rocky Flats Nuclear Weapons Facility and for 2005-2010 (Los Alamos)].
- Re-issued in 2011 under the new and more rigorous program.
- Current status is unknown.

PROFESSIONAL-RELATED ACTIVITIES

- Textbook reviewer for Wiley Interscience (1985-2012)
- Textbook reviewer for MacMillan Publishing Company (1986-1992)

MEMBERSHIP AND SERVICE TO PROFESSIONAL SOCIETIES

- Society for Risk Analysis (SRA)
  - Council Member for National Organization (1998- 2000)
  - Council Member of Northern California chapter (1995- 1996)
- American Industrial Hygiene Association (AIHA)
  - Member of the Risk Assessment Committee (1995–1997)
  - Liaison representative between AIHA and the SRA (1991–1994)
  - Member of the Toxicology Committee (1982–1984)
  - Member of the Workplace Exposure Limits Committee (1982–1984)
  - Co-Chair of Committee on Unusual Work Shifts (1981–1984)
  - President of Indiana Section (1981)
  - Member of Hygienic Guides Committee (1976)
- American Conference of Governmental Industrial Hygienists (ACGIH)
- Society of Toxicology (SOT)
  - President, Risk Assessment Specialty Section (2000)
  - Vice President elect, Risk Assessment Specialty Section (1998–2000)
  - Member of FAST 50th Anniversary Committee of SOT (2008-2011) [responsible for preparing documents, films, the book on SOT history and the party which was to celebrate the profession].
  - Assistant editor of the SOT 50th anniversary book describing the history of the Society of Toxicology (2010)
- Society of Environmental Toxicology and Chemistry (SETAC) Associate Chairman of Organization Committee (2000)
- International Society of Exposure Analysis (ISEA)
  - Member of Awards Committee (1999-2002)
  - Member of Executive Council (2002-2005)
  - Served as associate editor of the journal for nearly 15 years
- International Society of Regulatory Toxicology and Pharmacology (ISRTP)
  - Vice-president of the Society (2018-2024)
  - Chair of the Publications and Conferences committee (2016-2025)
- Air and Waste Management Association (AWMA)
- New York Academy of Science (NYAS) (Member, 1989)
- Sigma Xi (Stanford Chapter)

ADJUNCT PROFESSORSHIPS

- Adjunct Professor of Toxicology, Kansas University Medical Center (KUMC) (2017- 2027)
- Adjunct Professor of Toxicology, University of Michigan (2004-2012).
- Adjunct Clinical Professor of Medicine, University of California at Irvine (1996-2005)
- Visiting Scholar at Harvard School of Public Health (1996-1997)
- Adjunct Professor in Toxicology, University of Massachusetts (1993- 2006)
- Adjunct Professor, University of Texas, School of Public Health in Houston (1985- 1989)
- Adjunct Professor, Purdue University, Department of Toxicology/Pharmacology (1983-1984)
- Visiting Professor, University of Bridgeport, Department of Mechanical Engineering (1983)
- Graduate Instructor and Adjunct Professor of Industrial Hygiene and Industrial Engineering, Purdue University (1978-1982). Taught all undergraduate and graduate courses in industrial hygiene and industrial toxicology. Had the largest undergraduate program in industrial hygiene in the United States in 1982. He taught almost 300 undergraduate and graduate students in the School of Health Sciences and the College of Engineering.

PEER REVIEWER FOR THE FOLLOWING JOURNALS

- American Industrial Hygiene Association Journal
- Annals of Occupational Hygiene
- Applied Environmental and Occupational Hygiene
- Archives of Environmental Health
- British Journal of Ind Medicine
- British Medical Journal
- Canadian Journal of Dermatology
- Chemical Engineering Education
- Chemical and Biological Interactions
- Clinical Toxicology
- Critical Reviews in Toxicology
- Dose-Response
- Drug Metabolism and Disposition
- Environmental Health Perspectives
- Environmental Science and Technology
- Food and Chemical Toxicology
- Forensic Toxicology
- Human and Ecological Risk Assessment (HERA)
- Industrial Engineering
- International Journal of Work and Environmental Health
- Journal of Chemical/Biological Interactions
- Journal of Air and Waste Management
- Journal of Dose-Response
- Journal of Toxicology and Env Health

- Journal of Toxicology and Env Health Part II (reviews)
- Journal of Society for Exposure Assessment
- Journal of the American Medical Association (JAMA)
- Journal of Regulatory Toxicology and Environmental Policy
- Journal of Toxicological Sciences
- Lancet
- Mechanical Engineering
- New England Journal of Medicine (NEJM)
- Nature
- Occupational and Environmental Medicine (British)
- Professional Issues in Engineering
- Regulatory Toxicology and Pharmacology
- Risk Analysis
- Scandinavian Journal of Work, Environment & Health
- Science
- Toxicology
- Toxicology Letters
- Toxicology and Applied Pharmacology
- Toxicology and Industrial Health

EXPERIENCE SUMMARY (PROFESSIONAL CAREER)

TRC Companies
Chief Scientist
August 2024 – Present

- In August 2024, Paustenbach and Associates was purchased by TRC Companies with the purpose of developing a foothold in the high end risk assessment space (with a specialty in toxic tort litigation)
- Consultant in toxicology, industrial hygiene, risk assessment, state of the art, safety, and some aspects of chemical engineering.
- Strategic Health Sciences group leader. A team of nearly 20 persons who specialize in solving environmental problems using risk assessment methods.
    - Subject matter expertise in asbestos, PFAS, talc, benzene, chromium, airborne particulates, statistics, epidemiology, diacetyl, dioxins/furans, dioxins, PCBs, silica, engineered stone, formaldehyde, lead and mercury (and various other metal)s, formaldehyde, e-cigarettes (vaping), methylene chloride, side-stream exposure to cigarettes, the chlorinated solvents, carbon monoxide, radionuclides, and a host of other chemicals.

Paustenbach and Associates
President and Chief Toxicologist
April 2019 – July 2024

- Consultant in toxicology, industrial hygiene, risk assessment, state of the art, safety, and some aspects of chemical engineering.
- Our team can address epidemiology, toxicology, occupational health, ecotoxicology, environmental toxicology (over a very wide range of issues) and carcinogen risk assessment challenges facing the private sector or government entities.
- Experts in asbestos, talc, benzene, chromium, airborne particulates, statistics, epidemiology, diacetyl, dioxins/furans, dioxins, lead and various other metals, formaldehyde, e-cigarettes (vaping), side-stream exposure to cigarettes, the chlorinated solvents, carbon monoxide, radionuclides, and a host of other chemicals.
- Skilled at assessing the risks of exposure to nearly any chemical in air (direct and bystander exposure in the workplace), water, groundwater, soil, sediments, and consumer products)
- Remain active in the range of topics which he has studied for nearly 40 years.
- We had 3 offices, 17 full-time professionals and several "at will" professionals.

Cardno-ChemRisk
President Emeritus and Founder
December 2015 – April 2019

- Former President and Founder for a 125-person consulting firm. Description of the historical firm is shown below. Served as a Global Senior Principal consultant

ChemRisk
President
June 2003 – December 2015

- Chief scientist and manager for a 110-person consulting firm which provided state-of-the- art toxicology, industrial hygiene, exposure assessment, health sciences (e.g., pharmaceutical) and risk assessment services to clients who addressed a variety of public health and environmental health concerns. Offices were located in San Francisco, Pittsburgh, Boulder, Brooklyn, Cincinnati, Chicago, Los Angeles, Washington (DC), Houston, Portland (Oregon), and Jackson Hole.
- Continued to conduct research in exposure assessment, industrial hygiene, toxicology, epidemiology, simulation studies, epidemiology, dose reconstruction and risk assessment.

- Over the past 30 years, has been involved in directing (or co-directing) more than $60M in original research, which has been published, or will be published, in peer-reviewed literature.
- Provided expert witness testimony in public meetings and court hearings concerning the health effects of chemicals in sediments, air, soil, and groundwater.
- Nearly 35 years of experience in the fields of chemical/environmental engineering, risk assessment, toxicology, and industrial hygiene.
- Published nearly 300 peer-reviewed publications, more than 50 book chapters and about 350 conference abstracts in the fields of exposure and risk assessment, toxicology, engineering, industrial hygiene, and safety.
- Served as an adjunct professor in toxicology and risk assessment at the University of Michigan from 2005-2012 and continues to give lectures annually at various colleges.
- Has participated on more than 40 science advisory panels.

Exponent, Inc. Menlo Park, CA
Vice President
October 1998 - June 2003

- Member of the Corporate Management Committee.
- Most senior scientist and consultant within the Environmental Group.
- Responsible for the operations and management of Environmental and Epidemiology groups from October 1998 to January 2000. The group had about 140 employees and generated $19M a year in revenue.
- Editor of a college textbook and reference text entitled Human and Ecological Risk Assessment: Theory and Practice (2002).
- Author of a college textbook and reference text, The Risk Assessment of Environmental Hazards: A Textbook of Case Studies (1989), which is among the most frequently used textbook in undergraduate and graduate health risk assessment courses (5,000 copies sold).
- Specialized in the areas of industrial and environmental toxicology, pharmacokinetics, air pollution, historical state-of-knowledge regarding environmental issues, and ecological and human risk assessments.
- Provided expert witness testimony in public meetings and court hearings concerning the health effects of waste discharges and chemicals in sediments, air, soil, and groundwater.

McLaren/Hart Environmental Engineering Corporation, Alameda, CA
Senior Consulting Toxicologist (of counsel)
September 1997 - October 1998

- Assisted the company in moving through the process of merging and selling the firm.
- Senior scientist in the firm.
- Maintained relationships with various clients and served as Principal-in-Charge on selected projects.

McLaren/Hart Environmental Engineering Corporation, Alameda, CA
President and Chief Executive Officer
June 1996 - September 1997

- Responsible for the profit/loss, growth, and vision for the firm.
- Gave strategic direction to McLaren/Hart Environmental Inc., a 525-person nationwide firm that provided comprehensive environmental consulting services. Responsible for oversight of many of the technical/consulting aspects of the firm. Annual revenues of $100M.
- Directed the Consulting Operations Managers for 16 offices nationwide.
- Served on the Board of Directors.
- Maintained an active consulting and applied research practice in the areas of risk assessment, environmental engineering, toxicology, and occupational health.

McLaren/Hart/ChemRisk®, Alameda, CA
Vice President, McLaren/Hart Environmental Engineering Corporation Chief Technical Officer
January 1994 - June 1996

- Second ranking officer in the company.
- Responsible for revenue production for U.S. consulting operations (600 persons).
- Responsible for directing the technical/scientific efforts of this major (top 100) environmental consulting company.
- Responsible for directing the technical/scientific efforts of the company and for providing leadership to the four professional disciplines within 15 offices.
- Jointly responsible with the CEO/President for developing an international business presence. Focused on the development of business in Australia and China, as well as coordinating joint work efforts in England, Spain, Switzerland, and Germany. Became premier human and ecological risk assessment contractor in Australia and Switzerland.
- Responsible for starting, developing, and managing new offices in Sydney and Melbourne, Australia (March 1994 - March 1995). Presented a series of six lectures (two to four hours each) for the New South Wales and Victoria Environmental Protection Agency.

- Directed/conducted more than two hundred relatively important risk assessments of contaminated soils, sediments, and air emissions between 1985-1996 and was the project manager on numerous major contracts.

McLaren-Hart/ChemRisk®; Alameda, CA
Vice President, McLaren/Hart Environmental Engineering Corp. Chief Technical Officer
October 1991 - December 1993

- Responsible for providing direction to the technical/scientific portions of the company and for providing leadership to the five professional disciplines within all 15 offices.
- Responsible for coordinating corporate efforts when bidding on and executing major projects. Responsible for managing the National Business Programs in Remediation, Regulatory Compliance Management, Risk Assessment, Government Programs, Environmental Chemistry, Sales/Business Development, and Marketing. Responsible for the budget associated with these efforts.
- Responsible for day-to-day management of the ChemRisk® Division. In various polls by trade associations, it was ranked the most respected risk assessment group in the U.S. in 1993, it performed about $14M in risk assessment consulting.
- Provided guidance on operations in Alameda, CA; Burbank, CA; Irvine, CA; Rancho Cordova, CA; Chicago, IL; Portland, ME; Southfield, MI; Springfield, MO; St. Louis, MO; Warren, NJ; Albany, NY; Cleveland, OH; Philadelphia, PA; Pittsburgh, PA; and Houston, TX.
- Coordinated scientific and business aspects of international activities (primarily in Europe). In particular, gave scientific advice regarding work performed in former East Germany (Jena); Basel, Switzerland; and Budapest, Hungary.

ChemRisk Division; Alameda, CA
Vice President, McLaren/Hart Environmental Engineering Corp. Founder and National Director
November 1987 - October 1991

- Founder of the ChemRisk Division of McLaren/Hart Environmental Engineering. Managed and directed the joint venture involving the risk assessment component of the firm. The ChemRisk group grew from one person to nearly 100 professionals within four to five years. Annual revenues for 1991 were about $10M. The group had five offices nationwide (Alameda, CA; Irvine, CA; Cleveland, OH; Warren, NJ; and Portland, ME), which provided ecological and human health risk assessment services. ChemRisk was ranked as one of the 10 most successful environmental consulting groups in the industry for three consecutive years.

- Directed consulting activities in the areas of risk assessment, environmental and occupational toxicology, and the scientific aspects of toxic tort cases. Provided advice on air toxics and industrial hygiene issues, as well as remedial investigation/feasibility studies.
- Provided expertise in the areas of industrial and environmental toxicology, pharmacokinetics, environmental fate and transport, air pollution, odor abatement, and ventilation engineering.
- Routinely involved in the evaluation of health hazards posed by hazardous waste sites, landfills, contaminated soil, gaseous emissions, water, the workplace, and incinerators, as well as the control of in-plant gaseous releases, local exhaust ventilation, treatment of point-source gaseous emissions, contaminated soil and run-off, pesticides, and manufacturing processes.
- Managed projects involving the assessment of the health risks and significance of chemical contaminants present in water, sediment, ambient air, food, soil, the occupational environment, and in consumer products.
- Frequently interacted with local, regional, state, and federal regulatory agencies. Participated in negotiations to resolve issues involving clean-up standards for air, water, soil, and reusable items (e.g., contaminated vehicles and equipment). Risk communication experience included coordination of the scientific and medical aspects of complex toxic tort cases, representation of clients at public hearings, presentation of scientific data in professional and public forums, and development of opinion letters for use in litigation.
- Participated in the scientific debate regarding acceptable levels of emissions of tetrachlorodibenzo-p-dioxin (TCDD) in water from pulp and paper mills (GA, NC, and WA; 1990). Also identified acceptable levels of TCDD, benzene, formaldehyde, and chromium in air and soil.

Industrial and Environmental Toxicology Syntex Corporation; Palo Alto, CA
Manager
August 1984 - November 1987

- Managed a group of PhD toxicologists responsible for evaluating the corporation's occupational and environmental health hazards. The group also served as a scientific resource for the industrial hygiene and environmental engineering departments at both the corporate headquarters and remote manufacturing facilities.
- My group was responsible for setting all occupational exposure limits for many highly potent pharmaceuticals and for writing the "safe work practice" guidelines.
- Conducted complex risk assessments of three Superfund waste sites, including Times Beach, MO. Other significant sites include the Lyons Landfill (Denver, CO) and a site in the Bahamas.

- Other activities included the assessment of health risks of hazardous waste sites, the coordination of toxicity testing of all manufactured intermediates, the establishment of guidelines for laboratory use and manufacture of super-potent pharmaceuticals, setting of corporate occupational exposure limits, and directing of applied toxicology research programs.
- Participated in face-to-face negotiations with federal and state regulatory agencies regarding the evaluation, remediation, and closure of hazardous waste sites; reaching agreements on joint research efforts (e.g., USEPA's mobile incinerator); and defining compliance with air and water pollution regulations. Many discussions involved dioxins and various metals.
- Developed and directed the scientific and medical aspects of the litigation associated with the remediation of Times Beach, MO, and, peripherally, 43 other dioxin-contaminated sites.
- Wrote six 200-page evaluations of proposed federal environmental standards which were then submitted to the USEPA. Was influential in the development of USEPA and CDC positions on the health hazards posed by dioxin in soil, air, fly ash, water, sediment, dust, and other media.

Major Contributions

- Participated in USEPA's scientific re-evaluation of the health hazards posed by low-level exposure to dioxin (Region VII and at headquarters).
- Conducted a risk assessment of an USEPA Superfund site listed under the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA) outside Boulder, CO, and the Lowry Landfill in Denver, CO.

Stauffer Chemical Company; Westport, CT
Risk Assessment Scientist
February 1982 - August 1984

- Toxicologist responsible for the risk assessment aspects of environmental regulatory affairs. Primary responsibility was to evaluate the safety of company products through the design of toxicology studies and the conduct of health risk assessments.
- Responsible for risk assessment aspects of obtaining and maintaining the registration of agricultural and specialty chemicals. Conducted scientific assessment of company products alleged to present environmental or human health hazards. Responsible for developing and awarding contract research involving radionuclides, as well as the environmental fate and toxicology of pesticides and non-target wildlife.
- Responsible for assessing the potential hazards posed by municipal waste sites, groundwater contamination, water pollutants, new product uses, indoor and ambient air pollutants, and the manufacture and use of new products.

- Jointly responsible with the Industrial Health and Occupational Medicine groups for the development of the firm's OELs. Provided toxicology support for establishing Preliminary Manufacturing Notifications.
- Interacted with environmental groups such as the Sierra Club, Environmental Defense Fund, Audubon Society, etc. Interacted with both state and federal regulatory agencies including Occupational Safety and Health Administration (OSHA), USEPA and the Consumer Product Safety Commission (CPSC) as well as the California Department of Health Services, Louisiana Department of Health, and Texas Department of Agriculture. Worked as many as two days per week in Washington, DC, interacting with the above-mentioned groups, as well as USEPA and OSHA.
- Represented the regulated community, which consisted of the American Industrial Health Council (AIHC), Chemical Manufacturers Association (CMA), and Chemical Industry Council of California (CICC) at three public hearings regarding the proposed California Cancer Policy (1983).

Major Contributions

- Responsible for developing many of the environmental research programs and the risk assessment needed to obtain a USEPA registration for the first "biorational" pesticide to be approved by the USEPA. Pro-Drone, a pesticide for fire ants, was an insect juvenile hormone mimic. Designed and supervised field studies to demonstrate the efficacy of the chemical.
- Prepared risk assessments that were instrumental in registering three major agricultural chemicals. Facilitated and streamlined the registration and approval of major pesticides that had complex toxicology profiles by using risk assessment methodologies.
- Participated in the USEPA Science Advisory Board hearings on radionuclide emissions from phosphorous processing facilities (Section 112 of Clean Air Act). Participated in the development of the risk assessment used in developing the proposed rule (worked with Mr. Don Scroggins, Attorney at Law).

Purdue University; West Lafayette, IN
Adjunct Professor/Graduate Instructor
September 1978 - February 1982

- Prior to and during the pursuit of a doctoral degree, held an appointment in the School of Health Sciences and in the Department of Industrial Engineering.
- Developed and directed the entire undergraduate and graduate program and curriculum in industrial hygiene in the School of Health Sciences. Over four years, the Purdue undergraduate industrial hygiene program became the largest in the nation with nearly 60 students graduating per year.

- Developed and taught graduate level courses in environmental and occupational health, which included Industrial Hygiene and Toxicology, Industrial Hygiene Engineering, Occupational Health Law, and Occupational Safety and Health Engineering.
- Under a research grant from NIOSH, wrote a comprehensive (500-page) detailed outline for a textbook(s) and graduate course in Occupational Health Engineering. This textbook was used by many engineering schools to incorporate an awareness of safety and health principles into their design courses.

Eli Lilly and Company, Lafayette, IN
Industrial Hygiene Engineer
July 1977 - February 1980

- Participated in a wide variety of corporate projects involving environmental and occupational health at the firm's three major production facilities in Lafayette, Clinton, and Indianapolis, IN.
- Established the company's industrial hygiene program, including employee training and a trace analytical chemistry lab at these plants.
- Worked on numerous corporate projects in occupational health in the U.S., Canada, and internationally.
- Responsible for establishing programs to ensure the safe laboratory use and manufacture of toxic chemicals and sensitizing agents.

Major Contributions

- Involved in the design of new production facilities ($200M program) with the goal of ensuring that good environmental and occupational health engineering practices were implemented.
- Participated in developing the original USEPA protocol to assess the potential human hazards associated with the field application of herbicides and pesticides.
- Played a significant role in obtaining a favorable closure of a USEPA refutable presumption against registration of this firm's major product (Treflan®) that had been found to contain nitrosamines.
- Primary contact with NIOSH on health hazard evaluation studies of workplaces that handled or manufactured diethylstilbestrol (DES), pesticides, nitroglycerin, and products containing nitrosamines.

Eli Lilly and Company, Clinton, IN
Process Engineer/Chemist
May 1974 - September 1976

- Served as a chemical engineer, chemist, and industrial hygienist. Duties included improvement of production yields and installing process equipment in the facilities that manufactured antibiotics (including Keflex® and Cechlor®) and fermentation products (including monensin and cyclovine). Designed and installed several million dollars in process equipment.
- Played a significant role in several research and development projects that brought about major improvements in profitability.
- Was a member of primary engineering team responsible for the "start-up" of new automated broth processing unit for world's largest fermentation operation. Also, a member of technical service group responsible for the daily operation of two large solvent recovery units.
- Involved in establishing procedures for complying with Food and Drug Administration (FDA), OSHA, and USEPA regulations. Established this facility's industrial hygiene program. Wrote nearly all the relevant MSDS and warning statements for internal products, intermediates, and some purchased chemicals.
- His experience as a Kemper scholar for Kemper Insurance Company prepared Dr. Paustenbach to enter the fields of occupational and environmental health, as well as, become a fire protection engineer-in-training (1972). During those internships in 1971-1973, he worked in the Boston, NJ, New York, and Chicago offices.

KEY PROJECTS (PARTIAL LIST)

1. **Engineered Stone and Respirable Crystalline Silica Exposure:** Retained to evaluate the claims that fabricating engineering stone slabs were the cause of acute silicosis in some workers. This included a toxicological risk assessment and an evaluation of the effectiveness of engineering controls. (Summer and Fall of 2025).

2. **Formaldehyde in Airline Attendant Uniforms:** Retained to assess the inhalation and dermal hazards of the presence of formaldehyde as a crease stiffing agent in the uniforms of airline flight attendants. (Summer and Fall of 2025).

3. **Glyphosate**. Was contacted regarding the most recent claims about the adverse health effects of glyphosate. It is unclear whether the plaintiff claims will survive a MSJ. The scientific basis of the claims was weak. Fall of 2025.

4. **Hexavalent chromium and NHL**. A worker in the aerospace industry claimed that the inhalation of chromium cause his cancer. Case is in the early stages. Fall of 2025.

5. **Isocyanate exposure and hypersensitivity pneumonitis**: Retained to evaluate the hazards of TDI and MDI to workers at a repackaging facility. This case involved an employee who claimed permanent disability due to MDI causing his hypersensitivity pneumonitis. (Summer and Fall of 2025).

6. **PFAS in Astro Turf**: Retained to assess the dermal hazard due to PFAS in astro turf used for sports events. Original research was conducted and it was published in a peer reviewed paper (Spring of 2025).

7. **Heavy Metals**: Was asked to investigate the health hazards of the presence of up to 12 metals in the filler material within tampons. Original research was conducted to evaluate the transfer through vaginal tissue. (Spring of 2025).

8. **PFAS in Winter Gloves**: Retained to evaluate the alleged hazards of PFAS in winter gloves. The claim was that enough PFAS penetrated the skin to create a systemic toxicity hazard. Our research showed that this was not true. Indeed, very little, if any, measurable quantity of PFAS will penetrate the skin. (Spring of 2025).

9. **Retained to evaluate the health hazard to men from the use of talc during barbering:** Retained to assess the alleged hazards associated with trace concentrations of serpentine asbestos (most likely non-asbestiform) in talc products used after a hair cut. (Spring 2025).

10. **Retained to evaluate the alleged hazard of nitrous oxides released from a gas stove:** There has been much debate about the hazards of using gas stove vs electric stoves in unventilated kitchens (which don't have hoods). Retained in the spring of 2025, deposed in fall of 2025 and the case is to go to trial in 2026.

11. **Retained to evaluate a consumer product which contains both kratom and kava:** A new formulation was proposed, and we were asked to evaluate whether it posed any health hazards to potential consumers (Spring of 2025).

12. **Retained to investigate a case involving gaseous emissions from a landfill:** As is often the case, a landfill experienced an event that allegedly caused the release of large quantities of odorous gases. We were asked to determine whether a health hazard existed for the surrounding community. (Spring of 2025)

13. **Retained to assess the cause of death in a case involving kratom ingestion:** A person was reported to have died after consuming kratom. However, they also ingested other substances and had a number of other serious medical conditions. We were asked to determine whether kratom was the most likely cause of death. (Spring of 2025)

14. **Retained to evaluate the possible residual risks of aerial emissions from a fire at a metals processing facility:** In Philadelphia, a large industrial fire existed for about two days, and it released some unknown quantity of airborne particulate. We conducted residential soil and in the home r wipe sampling to evaluate whether significant quantities of the particulate had entered the community and posed any type of health hazard. (Spring of 2025).

15. **Assess risks of exposure to asbestos of several sheet metal workers:** Was retained to evaluate the likelihood that exposures to asbestos in the 1960s were causal of mesothelioma. (Spring of 2025).

16. **Retained to understand the dioxin and metals risks in the LA neighborhoods affected by the fires of the winter of 2025:** An insurance company wanted to know if the ash presented special risks. (Spring of 2025).

17. **Assess the health risks associated with alleged misuse of fire doors:** Was retained to characterize whether there was a human health risk associated with cutting a door (which should never occur) which may have contained Libby vermiculite. Retention was not finalized (Spring of 2025).

18. **Assess the health risks associated with the alleged presence of endotoxins on the surface of a medical implant:** Retained by the makers of a medical device to assess if endotoxins could be responsible for the alleged adverse health effects. (Spring of 2025).

19. **Investigation of alleged PFAS river contamination:** We were retained to evaluate if a chemical distributor purchased, sold or shipped any chemicals that could be classified as PFAS or were PFAS precursors to clients that were situated upstream of several towns along the Coosa River (January 2025)

20. **Newly Synthesized PFAS:** Conducted a risk assessment on a newly synthesized PFAS intended for use in the military and aerospace industry (Summer-Winter of 2024 to present).

21. **Kratom:** Conducted original research evaluating the acute toxicity of this herbal when taken in conjunction with certain pharmaceutical agents (Fall of 2024 to present).

22. **Retained to Evaluate a Novel Fluorinated Compound For Use in Data Centers:** A manufacturer asked us to evaluate the toxicology data regarding a dielectric fluid used to cool server components in data centers, which would significantly reduce cooling costs and energy expenditures (Fall of 2024)

23. **Juul e-cigarettes:** Conducted an analysis of the likelihood that adults might find use of this product to be a gateway drug for smoking. Our analysis of the literature indicated that vaping does not appear to be converting vapers to smokers (Fall of 2024).

24. **Analyzed impact of a large fire on warehoused paper products:** We were retained to determine whether gases and smoke from the fire that had occurred more than one mile away had affected the quality of more than one million boxes, intended for food use.. This was a complex risk assessment involving the collection of more than 500 wipe samples and bulk samples including the analysis for PAH, chlorine, furans, dioxins and a few other chemicals/metals (Fall of 2024).

25. **PFAS Regulatory Advocacy:** At the request of some chemical and semiconductor firms, we carefully analyzed various alternative approaches to regulating PFOA/PFOS and the four similar chemicals under the Clean Water Act. EPA and OMB were in the process of promulgating an MCL for these chemicals. Our analyses were shared with representatives of various states, several trade associations, two or more not-for-profit groups, and numerous elected officials. This resulted in two published papers about the hazards posed by PFOA/PFOS in drinking water (Fall of 2023-Spring of 2024)

26. **Interacted with representatives of the Pentagon:** A group within the military requested assistance on how to conduct a risk assessment of a few PFAS containing materials and some chrysotile containing materials which they considered important for national security (Spring of 2024)

27. **Contaminated garlic food seasoning:** We were asked to evaluate the safety to sensitized persons associated with the presence of a certain Asian pepper in garlic food seasoning. In some countries, up to 3-4% of consumers are allergic to certain types of peppers (Summer of 2024).

28. **Hazards of fire associated with batteries used in e-vehicles:** At the request of an auto maker, we conducted a screening level assessment of the off-gases and combustion products which were released from a fire involving batteries when an e- vehicle is in an accident (Summer of 2024).

29. **Formaldehyde:** Testified before EPA on the reasonableness of their proposed occupational limit for formaldehyde which was based on sensory irritation (Summer of 2024). Provided comments on behalf of the American Chemistry Council (ACC). Pro bono.

30. **Proposed occupational exposure limits for various commercial forms of asbestos:** Invested several months, without financial support, conducting research to propose OELs for chrysotile, amosite and crocidolite based on the recent work by Darnton (July 2024). This resulted in a published paper in the summer of 2025.

31. **Conducted a human and ecological assessment of a former paper mill (now a community park):** This assessment focused on a park in Northern California which was alleged to be a continuing source of dioxins (Spring of 2024-Summer of 2026).

32. **Daubert challenge on paper by Wiliams et al:** In a New Orleans court, I testified about the scientific strength of this paper on craftsman. It has been challenged previously. The outcome of the challenge was that the judge concluded that I had followed all the necessary requirements to be able to testify as an expert (July 2024).

33. **Prop 65 case:** A large apartment building in Los Angeles was alleged to have contained asbestos in the ceiling and trial lawyers filed suit against the landlords for not posting a Prop 65 warning. We conducted sampling and showed that the building has airborne concentrations below the "safe harbor" (they were not measurably present). (Spring of 2024). Deposition occurred in early 2025 and courtroom testimony was in summer of 2025.

34. **Worker exposures to asbestos in the steel industry:** Was retained by several steel makers to evaluate their industry hygiene data and testify in court about the possible exposures in the 1980-2010 era. A published paper was also generated (2023-2024)

35. **Evaluate off-spec chili powder:** A client had purchased a ton or more of chili powder from a 3rd world country. The ground powder had an earthy smell and there was a question about whether it posed a health risk. This was resolved after adequate study. Spring of 2024.

36. **PFOA/PFOS in Drinking Water Risk Assessment:** Conducted a risk assessment of the health hazards posed by these chemicals in drinking water as compared to the contribution to diet via seafood. Seafood was found to be a much more important source (Spring of 2024). This resulted in a very popular published paper on PFAS.

37. **Evaluation of comments submitted by the public on the proposed EPA MCL for PFOA/PFOS in drinking water:** Due to a likely upcoming legal challenge, we assembled all of the comments, evaluated them for merit and then identified how many of the comments had an impact on the final rule issued on April 10, 2024. Our analysis was published in a peer reviewed journal in the spring of 2025.

38. **Benzene contaminated soil:** Many trucks containing benzene contaminated soil were moved to a secure landfill. The drivers believed that the vapors caused both acute and latent effects. The client was an oil company. (Winter of 2023)

39. **Material Safety Data Sheet:** A client had a new drug which they needed to ship across the nation and the globe. Due to its toxicity when undiluted, we needed to write a data sheet which was comprehensive and met all appropriate regulatory guidelines (Winter of 2023)

40. **Contaminated spices:** Was retained to characterize the risk of various contaminants in certain hot peppers that were farmed in Asia and then shipped to the U.S. (Fall of 2023)

41. **Take home exposure regarding asbestos in a tire manufacturing facility :** Was retained to assess the probability that exposure of a quality control employee who walked through the plant could expose his son to fibers on his clothing (a take home claim) sufficient to cause peritoneal mesothelioma. (2022 and 2023)

42. **Retained to evaluate take home exposure regarding a pipefitter who worked in a Navy Shipyard:** It was alleged that a pipefitter in the 1960s was exposed to amosite in sufficient concentrations to bring home enough asbestos on his clothing to cause mesothelioma in his spouse. Puget Sound Navy Shipyard. A deposition and trial occurred in 2024 (2023- 2024)

43. **Health risks of ingesting Kratom:** In recent years, thousands of persons have chosen to purchase Kratom (a plant product) and use it as an aid for reducing systemic pain. Some have used it as a hallucinogenic agent while others use it to wean themselves off opioids. Was retained to work on several claims that were brought to the courts about alleged deaths due to overdosing on tea derived from the leaves. (Fall of 2023)

44. **Contaminated spice product:** Was retained by a firm to characterize the possible health risks (or lack thereof) associated with oregano that had been contaminated with mouse droppings. The risk analysis was informed by classic toxicology principles and historical FDA guidance. (Fall of 2023)

45. **East Palestine Fire (OHIO):** Retained to evaluate the health risks to the community associated with the combustion products from a railcar involved in East Palestine fire, as well as, the risk to groundwater (Fall of 2023). This retention did not move forward.

46. **Nitrosamines in a new pharmaceutical drug:** Was retained to do a quantitative assessment of a novel nitrosamine in a drug that hopes to come to market. This resulted in a comprehensive risk assessment that was published in a peer reviewed journal (Spring of 2023).

47. **Derive an occupational exposure limit for a new and novel pharmaceutical agent:** A manufacturer wanted to identify an OEL before attempting to bring it into large production so that engineering or administrative controls could be identified and implemented. (Spring of 2023)

48. **Evaluated the health hazards of potentially contaminated beverages:** Was asked to evaluate the impact of nine railcars being involved, to some degree, in the train wreck in East Palestine, Ohio. The evaluation involved whether nearly 1,000,000 bottles of beer needed to be destroyed due to proximity to the fire. (Spring of 2023)

49. **Evaluate the possible hazards of NO, NOx and PM2.5 from gas stoves:** Was retained to design a sampling plan and then conduct a health risk assessment of the emissions from a gas stove. This came into national prominence in 2022 and 2023 (Summer of 2023)

50. **Evaluate the risks posed by asbestos in the steel industry:** Questions continue to be raised about this topic. We gathered much of the data collected across the industry for the past couple decades and analyzed it. The data was originally collected by the steel industry trade assoc.

51. **Evaluated the health risks of vermiculite packing:** A claim was made that there was an asbestos hazard due to the historical use of vermiculite as a packing material for shipping tritium and other radionuclides. (2023)

52. **Conducted a radiological risk assessment of changing out a "pig" used to descale gas pipes:** A claim was made that persons who remove the descaling device that travels through gas pipelines (called a pig) are potentially exposed to excessive amounts of radionuclides when they change out the filter which contains scale. We evaluated all existing literature and case-specific information, then issued an opinion letter. It was a class action in Massachusetts. (2023). It was resolved in the spring or summer of 2023.

53. **Conducted an assessment of the potential cancer hazard to users of ENDs products (e.g., vaping):** There were concerns raised about this hazard. We collected all the relevant data, assessed the risk and our analysis was published in 2024. There were numerous trials about the alleged health hazards to users.

54. **Conducted a risk assessment of exposure to airborne benzene at a truck filling station (due to gasoline and diesel):** Several employees were concerned that they had developed NHL as a result of working at a facility that either pumped gasoline or diesel fuel into various trucks. We evaluated the published data and, fortunately, they had industrial hygiene data for this facility. (2022-2023)

55. **Evaluated the hazard of a unique nitrosamine in various products:** Between 2018-2022, a number of products were identified by FDA as being contaminated with various forms of nitrosamines. We conducted health risk assessments on several of them. We expect the work to be ongoing until the FDA establishes a guideline for how to characterize the risk of nitrosamines in products, which often represent less than 1/100th to 1/10,000 the dose of nitrosamines formed endogenously.

56. **Evaluated the effectiveness of a particular respirator for protecting against exposure to asbestos:** A major manufacturer had been drawn into litigation where claims were made that their device failed to adequately protect workers during the era in which it was sold. Our engagement started in 2022, and it is still ongoing.

57. **Evaluated 20 years of industrial hygiene data collected by a Steel Manufacturer:** Claims have been made over the years that asbestos exposures in Steel Mills during the 1960-1990 era might have been appreciable. That firm asked us to evaluate all their industrial hygiene data and to interpret it. Spring of 2022.

58. **Conducted a comprehensive risk assessment of a former Navy Shipyard used to restore vessels after WW II:** There had been a remediation of a fairly large navy shipyard that occurred over the past 15 years. After the remediation was completed, the city planned to put offices and homes on the former site. The community was not convinced that it had been properly cleaned so they filed various legal actions. We conducted a health risk assessment and published the analysis in a peer reviewed journal (Critical Reviews in Toxicology) Deposition was in 2025 and trial is scheduled for 2026 (2019-2026).

59. **Presence of methylglyoxal (MGO) in the gases from e-cigarettes:** It was reported that MGO was being emitted from some brands of electronic nicotine delivery (ENDs) devices and inhaled by users. The chemical was alleged to be a possible strong irritant to the lungs of users. The work began in 2022 and ended in 2025.

60. **Assess the likelihood of kidney disease due to working in a diluted sewage pumping station:** A claim was made that an employee who worked in a pumping station developed acute kidney failure due to breathing gases in the building. Began in 2022 and was settled in early 2023.

61. **Evaluation of a fire associated with ash from a wood to energy plant:** A pit where wood ash had been stored went exothermic and burned down the storage facility also moved into a nearby forest. We were asked to make suggestions regarding how to prevent such an incident in the future and to conduct a companywide safety assessment. Began in 2022 and still ongoing.

62. **Asbestos hazard associated due to its presence in laboratory equipment:** A firm who sold muffle furnaces, gauze pads for Bunsen burners, and asbestos blankets asked us to conduct a risk assessment of the possible hazard to high school and college students who used these products from about 1940-1990. Work began in 2021 and is still ongoing.

63. **Contaminated bottled water incident:** It was alleged that a firm that sold processed water had distributed a product which was alleged to cause liver damage in select consumers. We were part of an investigative team retained by the client to evaluate the validity of the claims and to try to identify the contaminant (if the claims were valid). (2022 and still ongoing)

64. **Contaminated foods incident:** It was found that more than one hundred pounds of liquid ammonia were released in a warehouse full of vegetables (millions of pounds). We were asked to conduct a safety assessment using classic risk assessment techniques, as well as review FDA guidelines, to determine whether some or all of the products were safe to eat. (2022)

65. **Asbestos disease potential posed by Vermiculite fireproofing:** Due to continued asbestos litigation, we were asked to assess a particular type of fireproofing to determine if the vermiculite was from the Libby mines. We were then asked to evaluate thousands of pieces of air sampling and bulk sampling data to determine if there was a health risk associated with reoccupying the building. Gave testimony in a higher court in Calgary in Dec. 2022. The case involved a nearly $1B rental agreement. We were told that the claims about adverse health effects were dismissed.

66. **Nitrosamines (NDMA) in several consumer products:** We were asked to evaluate the likelihood that nitrosamines were being formed in a pharmaceutical agent due to storage at warehouses or drug stores. We were also asked to determine if the nitrosamines that were present posed a health risk to consumers. The key product was a blood pressure medicine. Case was dismissed after declaration was submitted to the courts in Canada. (2022).

67. **Airborne toxicants from a petroleum fire:** Was retained to evaluate the validity of claims that BTEX vapors which were released from a fire near a ship that was in a waterway in Texas has caused various lung ailments in those on the ship. (2022).

68. **Recall of consumer products due to trace benzene in propellant:** In 2020-2021, there were a number of products recalled due to the presence of trace amounts of benzene in the propellant. We evaluated the theoretical increased risk associated with the concentrations of airborne benzene with each use. In all cases, the concentrations were too low to warrant the concern of the public or regulatory agencies.

69. **Contaminated grain:** Was retained by a major processor of grains to evaluate an unusual scenario. The firm had transported 20 truckloads of wheat to a barge. The transport company failed to wash out the truck which has previously hauled farm fertilizer. Parts per billion concentrations of fertilizer were in the grain. A risk assessment was required to decide what should be done with the grain. (Fall of 2021).

70. **Contaminated hair dye:** A firm was alleged to have sold a hair dye containing a carcinogen (4-amini biphenyl, 4-ABP) that was sometimes measured in hair dyes in the 1950s-1970s (but it was 2021). The plaintiff claimed that their cancer was due to this trace contaminant which could well be present in most dyes at the ppb level today. We conducted a risk assessment which estimated the absorbed dose. (Fall of 2021).

71. **Covid:** There were a number of claims that persons developed Covid and died due to workplace exposure. We were retained by several employers to conduct an analysis of the most likely cause of the workers disease (usually airway disease) and the likelihood that Covid was responsible. (2021-2022). The first case went to trial in New Jersey in Oct. 2023.

72. **Toxicity of herbal supplements:** A claim was made that use of an over-the- counter vitamin supplement had caused acute liver failure. We conducted an evaluation of the toxicology of each of the components, the doses taken each day, and the various other factors that can cause liver failure. We concluded that the plaintiff's ingestion of nearly one- half bottle of whiskey per day was the more likely cause of the liver injury. (2021)

73. **Contaminated beverage recall:** Performed a fault tree analysis and field study to evaluate whether a beverage manufacturer, as alleged, performed a number of production runs which were contaminated with an agent that caused acute viral hepatitis in some consumers. This evaluation began with virtually no data, and we carefully examined the process, as well as the products, from two separate facilities. A report and risk assessment were issued. (Winter of 2021 through 2022; litigation continues).

74. **Nitrosamines in pharmaceuticals:** Provided advice to a large pharmaceutical company regarding trace contamination by nitrosodimethyamine (NDMA) and nitrosdiethylamine (NDEA) in their products. In around 2017, it was found that some over the counter and prescription drugs contained this chemical which was found to be a rodent carcinogen many years ago. We were asked to conduct a health risk assessment on those persons who ingested these drugs for up to three years during which the contamination was present in Canada. Initial work was performed in the Fall and Winter of 2021.

75. **Accidental death of two chemical workers:** We were retained to evaluate an incident where two employees were exposed to some airborne agent for a relatively short period of time and, sadly, eventually died of pulmonary edema. We were part of a team to try to identify the likely chemicals (including PFIB) that were responsible for the incident which occurred in the production of PTEE (perfluoro tetra-fluorinated ethylene). It appears that we identified a previously unknown occupational hazard that is unique to some portions of the chemical manufacturing industry (Fall of 2021-2025).

76. **State of the art defense regarding crocidolite in transmissions:** A firm which once manufactured automatic transmissions in the 1945-1970 era was sued when workers developed mesothelioma. We were asked to conduct a state-of-the-art analysis to determine whether the firm had acted responsibly given the information available in publicly accessible information of the time. (Winter of 2021 through 2022)

77. **Retained by a large manufacturer to evaluate an insurance claim regarding COVID:** A major manufacturer in the United States believed that they have had suffered a loss of $4B due to inability to produce goods due to COVID. Like many other firms, they believed their business interruption insurance should cover some of those claims. We were retained to evaluate whether their protective measures were state of the art and that they had done all that they could to control the spread of the virus. (Winter of 2021-Winter of 2023)

78. **Provided expert testimony on exposures during railroad work:** A plaintiff believed that they had developed several diseases due to exposure to diesel exhaust, creosote, mineral spirits, carbon monoxide and airborne particulates while being an engineer on a railway. (Spring of 2021)

79. **Retained to offer opinions about the likely magnitude of exposure to asbestos in the steel industry:** Contrary to what is commonly believed in the field of industrial hygiene, there was never significant exposure to asbestos for the vast majority of jobs in this industry from 1950 to the current era. A comprehensive analysis of 40 years of industrial hygiene data was conducted and the data supported that view. (Spring of 2021)

80. **Retained to study exposures due to asbestos gloves in the steel, glassmaking and laboratory industry:** A comprehensive analysis of published and unpublished data was conducted. Expert testimony on the topic was offered in Spring of 2021.

81. **Retained to evaluate the various ways that persons who looked in labs in the 1940s-1990s were exposed to asbestos:** A major supplier retained Dr. Paustenbach to evaluate this matter and offer expert testimony in Winter of 2020.

82. **Allegations of exposure to trace benzene in calibration fluids:** Claims were made that trace concentrations of benzene were present in naphtha fluids used in an industry related to mining and gas. Even though there are 20-30 years of knowledge that virtually ND concentrations of benzene are in these fluids, claims that some persons developed AML were made in Winter of 2020.

83. **Retained to evaluate exposures to beryllium in golf club manufacturing:** It was claimed that an employee who used a grinder in a golf club factory developed CBD. We were retained to evaluate the exposures and the probability that his disease, if accurately diagnosed, was due to these exposures. (Winter of 2020).

84. **Asbestos in laboratory products (1950-2010):** Was retained by lawyers representing a laboratory equipment manufacturer. Claims had been made about unacceptable levels of exposure associated with using a tong with asbestos sleeves, gauze pads containing chrysotile asbestos, asbestos containing gloves, and other equipment. (Winter of 2020).

85. **NDMA in pharmaceuticals:** Was contacted by a manufacturer who was receiving claims that their product contained trace concentrations of nitrosamines. This is an issue that has been raised in the 1970s and 1980s, when our team gave advice to FDA on how to set acceptable contaminant levels for over the counter and prescription drugs. (Fall of 2020).

86. **Evaluated the risk of an asbestos related disease due to fiber drift at a shipyard:** Lawyers for the plaintiffs claimed that persons who worked very far away from a work area were overexposed. In this case, the sailor was allegedly exposed outside a ship within 200 yards of an exhaust duct. (Summer of 2020).

87. **Retained as a toxicology/pharmacology expert regarding nicotine:** Due to litigation associated with e-cigarettes, there was a need for someone who had studied nicotine pharmacology to get involved in claims regarding adverse health effects in persons of varying ages. Was retained by a major manufacturer. (Summer of 2020-Fall of 2022).

88. **Evaluated the likelihood of permanent brain damage due to chronic low-level exposure to carbon monoxide:** It was alleged that ten men worked in an area where the airborne concentrations in the winter were in the vicinity of 10-50 ppm for several hours per day. The question to be addressed was the likelihood that these doses would produce short term or permanent memory loss and learning impairment. It was also alleged that the exposures caused white dots in certain portions of the brain. (Fall of 2020).

89. **Served as a PMK witness for a firm that went out of business around 1991:** Our firm attempted to learn as much as possible about the history of our client and the totality of the coverage and testify as a 30b6 witness. (2020-2022)

90. **Respirators and their limitations for protecting workers:** Was retained by a major manufacturer who sold disposable (sometimes single use) respirators intended to protect workers from various dusts. The claim was that they often did not provide a protection factor of 5 or 10 and, as a result, did not always adequately protect persons who worked with asbestos in the 1970s and 1980s. (Summer of 2020).

91. **Evaluated the plausible health hazard of asbestos emissions to a community 2-3 miles from the point source:** Was retained to estimate the ambient concentrations of crocidolite and chrysotile far from the plant which had inadequate air cleaning devices, and which manufactured transite pipe, transite wallboard, and cements. This required the use of original research conducted by our firm on fiber transport which was then incorporated into classic air dispersion models. (Summer/Fall of 2020)

92. **Evaluated claims regarding talc:** Due to allegations regarding the hazards of using cosmetic talc in two settings, we evaluated the maximum plausible airborne concentrations that might have occurred and the duration. At this time, we have not been retained as experts. (Summer-Winter of 2020).

93. **Conducted exposure assessment of Pressmen who worked with benzene in the 1950- 1970:** It was not unusual for workers in press shops to remove ink from printing machines using a cleaning fluid which might contain 0.5-10% benzene. The allegations involved a worker who developed multiple myeloma. (Summer of 2020).

94. **Evaluated the likely mechanisms of initiation of certain cancers, including leukemias:** The question brought to us was “when can one say that a person has experienced the onset of a disease” based not on the date of first exposure, which is not initiation of disease, but rather when the disease process was initiated. Retained as a consulting expert. This was a novel scientific and legal question. (Summer of 2020).

95. **Retained to evaluate the risks to mechanics of working with asbestos- containing gaskets on motorcycles:** The number of claims against motorcycle manufacturers has increased in recent years. We were retained to give opinions about the magnitude of exposure and the likelihood of developing an asbestos related disease. (Lasted all of 2020).

96. **Evaluated the possible health risks of aluminum in potable water for an RV manufacturer:** Reviewed available literature on aluminum leaching and possible toxicity to consumers due to certain concentrations being present in hot water heaters from potable water tanks. Found that for the concentrations measured there was a no health risk to consumers. Both EPA and NHTSA adopted our risk assessment. (Fall of 2019).

97. **Was retained to evaluate the possibility of developing a hypersensitivity pneumonitis as a result of exposure to food flavorings while working in a Chinese or Vietnamese restaurant:** Due to the interest in alleged hazards due to exposure to diacetyl in consumer products (like popcorn), there were a number of cases filed in the restaurant industry regarding exposure to many different types of flavoring. (Fall of 2019-Spring of 2020).

98. **Retained to evaluate the potential adverse effects of vaping of liquids containing nicotine and various flavorings:** In light of the national concerns about whether E-cigarettes are a public health problem or a benefit to those who are trying to quit smoking (or both), this presents a classic opportunity for conducting a health risk assessment. (Winter of 2019-Winter of 2020).

99. **Assessment of soil samples at a former Naval site:** Reviewed all the available information and assembled a scope of work to conduct a sensitivity analysis and risk assessment. (Summer of 2019-Winter of 2020).

100. **Evaluation of claims that pigeon droppings caused chemical hypersensitivity pneumonitis (HP):** It was alleged that an employee of a railroad developed, a potentially fatal fibrotic lung disease, due to occasional, outdoor, workplace exposure to dried airborne pigeon droppings. An analysis of all published and case specific materials indicated that inhalation of fungi, if it occurred, was not causal. (Fall of 2019).

101. **Assessment of the hazards of asbestos containing duct sealer:** Was retained by a firm to evaluate the possible hazards to workers applying, and later attempting to remove, a polymer-based duct sealer. The 1950s product was remanufactured and used in a simulation study (Fall 2018). The results were published in an internal report. We expect a published paper to follow. Consulted on some cases in 2018 and 2019.

102. **Evaluated the reasonableness of claims regarding the carcinogenicity of glyphosate:** Was asked by an investment group to assess the evidence that Glyphosate was a likely human carcinogen and the probability that the courts would make such a conclusion (as related to the numerous personal injury cases that the been filed. (Spring of 2019).

103. **Asbestos Exposure to Millwright at Oil Platform Construction Facility:** Evaluated claims that there was excessive exposure to asbestos fibers while working with gaskets at a site in Louisiana. (Spring of 2019).

104. **Evaluated the alleged health risk of the presence of trace quantities of nitrosamines in a pharmaceutical:** Was asked to evaluate the magnitude of the health risk to patients who took normal quantities of a drug that was in the news, as well as others. (Summer of 2019).

105. **Attempted to Determine for Categorization of Cobalt as a Systemic Toxicant in EU:** Investigated the basis for the categorization of cobalt under the new labeling scheme and submitted comments about the scientific merit of that pending decision. (Spring of 2019).

106. **Asbestos Fiber Transport from Facilities into the Community:** We conducted at least one field simulation study where we evaluated the aerial transport and distribution of chrysotile, crocidolite, and cement particles from a point source. The purpose of the effort was to determine if fibers were "more likely than not" (on an aerodynamic mean diameter basis) to travel a shorter distance than spherical particles. It was anticipated that this would give insight on how to adjust the various air dispersion models. The work was presented at several conferences and is under peer review. (2018-2020).

107. **State of Art Expert in an Asbestos case:** Was retained by a firm who had had employees allegedly involved in installing insulation in homes in the 1950s and 1960s. Claims were made that this union worker was never informed of an asbestos hazard.

108. **Health hazards posed by human exposure to cosmetic talc:** Was retained by a law firm to evaluate a person injury claim that exposure to cosmetic talc was responsible for causing a mesothelioma in a relatively young woman. Exposures were alleged to be due to using it as an anti-perspirant and while powdering her infants while changing diapers. (Mid-2018).

109. **Addressed question regarding vitamin A concentrations in food supplements:** A small supplier of specialized foods accidentally added more than prescribed amounts of Vit A to one of their foods. After conducting a risk assessment, we recommended that the batches of this product that were in transit be returned to the manufacturer. (Late 2018).

110. **Claims regarding the adequacy of MSDS overtime:** A large multi-national firm had been sued for allegedly not providing adequate toxicology and industrial hygiene information in their MSDS on Perfluoro chemicals from 1980-2010. The case involved allegations regarding more than 1,000 chemicals sold by the firm. We conducted a "case by case" analysis of the state-of-the-art regarding whether the MSDS reflected what was known at the time the MSDS was issued. (2018- 2019).

111. **Claims regarding exposure to industrial talc:** Was retained by a tire maker who had experienced allegations regarding exposure to industrial talc and mesothelioma. (Late 2018).

112. **Claims of hair loss due to use of shampoo:** A large private sector client was alleged to have sold a shampoo which caused air loss. A risk analysis of the various components was conducted, and an opinion letter was issued (mid to late 2018). Several published papers came out of this work.

113. **Alleged vapor intrusion by PCE and TCE in basement:** An engineering firm who performed remediation of local groundwaters was alleged to have failed to prevent intrusion of the vapors into the basement of a garage. An analysis of the state of the art regarding expectations in 1990 was conducted and an opinion letter was presented to the court. (Mid to late 2018).

114. **Claims of asbestos related disease in tire workers:** It was claimed that a contractor who worked at a Goodyear facility had developed meso as a result of working part time at that site. An exposure analysis and causation analysis were conducted and presented in an opinion letter. (Mid-2018).

115. **Brake dust from a trolley car:** A claim was made against many firms, including the City of San Francisco, that mechanics of the trolleys were placed at increased risk of asbestos related disease due to their occupation. (Mid-2018).

116. **By-stander and take-home exposure of a refinery spouse:** It was alleged that a spouse of a worker at a Chevron refinery developed mesothelioma due to her alleged exposure associated with visiting her husband at lunch and due to take home exposure. An exposure assessment and causation analysis were conducted in mid-2018.

117. **Alleged disease due to cutting crocidolite containing pipe:** An oil development firm was alleged to have allowed a contractor to cut transite pipe without respiratory equipment which was said to be the cause of his disease. An "employer responsibility" analysis was conducted, and an opinion letter was offered. (Early 2018).

118. **Numerous cases associated with gaskets:** Throughout 2018, various claims were made against pump manufacturers that the asbestos that might have been released during the change of gaskets was the cause of disease. Many opinion letters were offered, and the courts often concluded that the scientific basis of the claims was unfounded. (All of 2018).

119. **TCE in groundwater:** A client who was a government contractor was alleged to have released TCD into a swamp between 1960-1972. The State believed that these actions were a serious infraction of the laws of the time. A state of art analyses of common and widely held "work" practices was conducted (early 2018). An opinion letter was provided to the courts and a deposition followed.

120. **Two deaths due to carbon monoxide:** Was retained to figure out why two persons who lived in an apartment died of carbon monoxide poisoning. Theories included a poorly designed parking area, holes in the floor of the apartment, a defective heating system, a defective auto which was alleged to start on its own, and others were offered (early through mid-2018).

121. **Work practices at an organic foods factory:** Was retained to do a site walk and examine all the quality control practices at an organic food factory. It has been common for these facilities to be the source of salmonella and other bacterial outbreaks. There are common characteristics which we have come to identify which are often the cause (2017-2018).

122. **Contaminated "organic" foods:** A small processer of kale had distributed some contaminated foods and needed an assessment regarding the need for a recall. After analyzing all of analytical data, it was concluded that the recall was necessary, and it was executed (2017).

123. **Occupational exposure to fly ash:** A client who was responsible for cleaning up the fly ash following the great landslide in Tennessee (power plant) was sued regarding allegations of an inadequate industrial hygiene program. We evaluated thousands of pieces of IH data and wrote up an analysis (2017).

124. **The need to shower after daily work at a Foundry:** A very complicated case was filed in the mid -2000s which alleged that workers in a foundry should have clean clothes issued each day and that the employees should shower before going home. This case involved understanding the scientific basis for asking workers to shower and whether it was appropriate to pay them to do so (vs them choosing to do it on their own time for reasons of personal hygiene or comfort) (2017-2019).

125. **Performed national study of exposures to hydrogen peroxide as a sanitize:** We were awarded a contract by manufacturers to assess the magnitude of exposure during typical use of hydrogen peroxice as a sanitizer in the poultry processing, greenhouse, food processing and hospital facilities in the United States. This was part of the requirements of the "data call-in" responsibilities of the new TSCA (2017-2020).

126. **Evaluated the possible hazard of gamma radiation from cleaning oil field pipes of scale:** This is a classic risk assessment involving naturally occurring radiological materials (NORM). We are in the process of the evaluation, but it seems clear that the doses to these employees are not much greater than background (2017)

127. **Assessed the hazards posed by trace contamination of vegan foods by dairy products:** Currently, FDA has no definition for vegan foods. Although rare, some persons are allergic to dairy products even at low concentrations. We attempted to identify the concentration of concern so that quality control measures could be implemented by the food supplier (2017)

128. **Mold release from auto air conditioners:** Beginning in about 2007, a number of lawsuits were filed against many manufacturers that the drivers of cars were being adversely affected by microbes, odors, bacteria, or some agent that blew out of the vents when the car was started (often in the summer in humid climates). This litigation was hugely complicated and required that millions of dollars of air sampling be performed. The work continues to be pursued by 6 or more different consulting firms. We got involved in 2017.

129. **Evaluated potential for residential exposure to asbestos from site emissions:** There is a rapidly evolving mass tort associated with claims that citizens living withing 2-3 miles of a facility which may have emitted or handled asbestos could at risk of asbestos related disease. We have been aggressively evaluating this hazard with consideration given to the aerodynamic mean diameter of fibers vs. regular particulate emissions. We have been retained by several firms who are involved in this litigation (2016-2018).

130. **Assessed the hazards posed by trace contamination of soy in "soy free" products:** Currently, FDA has no definition for what defines soy free. A client found that they had accidentally contaminated a batch of foods and wanted to know if the foods were safe to deliver. Using FDA documents on the subject, and other published papers, we identified the concentration of concern and found that foods at this concentration could be safely ingested (2017).

131. **Assessed the hazard to local citizens of dioxin contaminated sediments in San Jacinto Bay:** We were retained to evaluate a fairly controversial site in the Houston ship channel to determine whether fish and other aquatic life contained a sufficient concentration of dioxins, furans and PCBs to pose a significant health hazard to local residents who ate these foods. (2017-2020).

132. **Performed mechanistic research on normal and unusually large debris from hip implants to evaluate its role in a hip revision:** We were engaged to attempt to figure out the biological cause, if any, for the apparent upswing in the number of revisions of metal- on-metal hip implants over the past 7 years. We collected a large portion of particles from hip simulators run at typical angles and particles from those at articulated angles (far outside the specifications). We concluded that the large wear debris was much more biologically active than normal debris in cell culture tests and we hypothesized that similar results would likely be observed in mammals in tox studies and in humans who had had implants which were at extreme angles. (Kovochich, M., E.S. Fung, E. Donovan, K.M. Unice, D.J. Paustenbach, and B.L. Finley. 2017. Characterization of wear debris from metal- on-metal hip implants during normal wear versus edge-loading conditions. J Biomed Mat Res B. Advanced online publication, May 8, 2017. doi. 10.1002/jbm.b.33902). Other papers followed in 2018-2019.

133. **Libby amphiboles released from a lumber processing facility:** We were asked to evaluate the magnitude of release of asbestos fibers (so-called Libby amphiboles) from a wood processing facility which operated in Libby for several decades. A number of claims had been made that the emissions caused disease in the community. (2017 and 2018).

134. **Retained to evaluate the cause of odors in autos during air conditioning start up:** There have been a number of claims leveled at many auto manufacturers regarding odors that are allegedly released when an air conditioner or heater “starts up”. To understand the validity of the claims, a comprehensive national program was developed which involved sampling the air in a wide range of autos. (Summer of 2017).

135. **Assisting firms who need to meet expectations of TSCA reform:** In 2016, Congress approved of a major reform of TSCA. As a result, many firms have contacted us and asked for advice about what is necessary to meet the new expectations. (Throughout 2017).

136. **Evaluated de minimus contributors to Passaic and other bodies of water:** In 2015-2017, numerous firms were named as contributors to the pollution of the Passaic River. We have been retained by a couple firms who have been drawn into litigation due to claims that they emitted metals and persistent organic pollutants (POPs) to this body of water.

137. **Attempted to determine the cause of a fire in an enclosed vessel at a refinery:** A fire occurred in a refinery, and it was alleged that as many as 20 persons were adversely affected by the products of combustion. We were part of a team of professionals tasked to determine the cause of the fire and why it propagated so rapidly. We also assessed whether various persons who were involved experienced temporary or permanent adverse health effects. (Circa 2016-2017).

138. **Evaluate the hazard posed by release of methyl bromide gas after fumigation of fruit:** There are only a few remaining acceptable uses of MB gas for killing certain pesky insects. We were asked to assess the risks to the community associated with the quantity of MB released to the ambient air. Site was near a river in New Jersey. (Summer of 2016 through Summer of 2018).

139. **Assessment of contaminated groundwater and associated soil gases in a community in San Diego:** We were contacted by one of the PRPs for a rocket parts manufacturing firm about a plume which had been known to exist for the past 40 years. Recently, claims were made by citizens that some homes and a school had much higher than background concentrations of volatile organics. The fingerprint pointed to the plume. A health risk assessment was conducted which mirrored one conducted by the State of California (known as the Ametek site). (2016 – 2019).

140. **Set an occupational exposure limit for diacetyl:** For several decades, we have been involved in setting “internal” occupational exposure limits for corporations. Recently, several firms have asked us to identify an OEL for diacetyl in light of claims about exposure of manufacturing workers, those in the coffee manufacture and distribution industry, and those in the flavorings industry (most of 2016 and 2017).

141. **Characterized the hazard posed by PFOA to water bodies:** Since the late 1990s and early 2000s, we have been studying the fluorinated chemicals with respect to the toxicological hazard and the fate/transport in the environment. In 2015, we were asked to investigate the possible hazard associated with the contribution of textile factories to the contamination of sludges in wastewater treatment plants. This is a highly complex issue which captured the attention of the media and trial lawyers in about 2014 and this continues through 2018.

142. **Potential dermal hazard posed by a popular hair shampoo:** We were retained by a firm who was being investigated by FDA regarding claims by clients that hair loss was occurring with routine use (Spring of 2016 through Fall of 2019). We have been conducting research to evaluate this hazard for about 18 months.

143. **Exposure of persons who cut cement pipe containing asbestos:** We evaluated the possible inhalation hazard associated with cutting pre-1970 cement pipe that contained about 15% chrysotile and 10% crocidolite. We also evaluated the take-home hazard of contaminated clothing (summer of 2016 through spring of 2017). A manuscript describing the exposures was published in late 2017.

144. **Evaluating the take home exposure of persons who cut cement pipe containing crocidolite and chrysotile:** At the request of a client, we conducted a simulation study to evaluate the possible exposure to family members (e.g., take home study) associated with laundering the clothing of person who occasionally cut this type of pipe. A manuscript describing the exposures was published in 2017.

145. **Assessing the dermal irritation of MCHM in water at concentrations relevant to the Charlestown, WV incident:** We were asked to evaluate this hazard and, as a result, conducted a study involving volunteers through a well-respected contract lab. Work was conducted in 2016. The results were published in a peer reviewed paper in early 2017. (Monnot, A.D., R.M. Novick, and D.J. Paustenbach. 2017. Crude 4-methylcyclohexanemethanol (MCHM) did not cause skin irritation in humans in 48-h patch test. Cutan Ocul Tox. Advance online publication, March 13, 2017. doi: 10.1080/15569527.2017.1296854.).

146. **Conducted research to identify the cause of failures (usually loosening) of patients who had received metal-on-metal (MoM) implants:** We were asked to attempt to identify the most likely mechanism by which MoM (or MoP) implants might fail between 1 to 10 years following the surgery. Between 2012 and 2017, we conducted several studies in an attempt to answer that question. At least two or three papers have been published or are in progress which address this issue. I have testified in one court case associated with hip implants (2014-2017).

147. **Assessing the developmental toxicology hazard posed by short term exposure to MCHM in water at concentrations relevant to the Charlestown, WV incident:** We were asked to evaluate this hazard and, as a result, conducted of the birth outcomes of all relevant hospitals before and after the incident. Work was conducted in 2016. The results were published in a peer reviewed paper in 2017. (Benson, S.M., P. Ruestow, K.A. Keeton, R.M. Novick, G.M. Marsh, and D.J. Paustenbach. 2017. The 2014 crude 4- methylcyclohexanemethanol chemical release and birth outcomes in West Virginia. Arch Env Occup Health. Advance online publication, July 10, 2017. doi: 10.1080/19338244.2017.1350132)

148. **Evaluated what was learned in the Shanghai Health Study (SHS) of occupational exposure to benzene conducted in the 1990s era:** Without any outside funding, we decided to review all the published and unpublished work related to the SHS study and conduct a comprehensive assessment of what was learned. The paper was published in Critical Reviews in Toxicology in late 2017.

149. **Evaluated the alleged risks associated with exposure to talc or talcum powders:** In about 2014, a new generation of claims about the hazards posed by talc with respect to causing ovarian cancer and mesothelioma were made. We began to evaluate the scientific legitimacy of these claims at that time. Several papers are in progress which describe our current views (Spring of 2016).

150. **Assessed a former site of a manufactured gas site in Sydney, Australia:** We were contacted to determine the necessary level of clean-up at this site which was in downtown Sydney; as well as give advice on remediation techniques. Dennis also served on an advisory board to the mayor as to how to move forward (called the Barangaroo site) (Summer of 2016).

151. **Evaluated the claims that the number of head injuries in the NFL were increasing and that the incidence of concussions was causing pre-mature death:** Around 2016, we were retained to evaluate the claim that head injuries seen with NFL and other football players was causing pre-mature dementia and/or death. A preliminary epidemiology study was performed.

152. **Evaluated the historical asbestos hazard to radio and TV repairmen of the 1950s and 1960s:** We were asked to evaluate the magnitude of health risk, if any, associated with repairing radios and TVs during the era when asbestos may have been used in the Bakelite base holding the vacuum tubes, the heat shield and dust guards.

153. **Benzene in paints:** A new series of cases were filed which alleged that there was substantial exposure and risk associated with certain paints and stains manufactured in the post-1976 era. A complete review of the literature and mathematical modeling were performed, and several cases went to deposition on this matter.

154. **Assessed a concern that endotoxins were present in a workplace that manufactured shrink wrap:** We conducted a process review and site walk of the facility. We concluded that there was an excessive concentration of airborne endotoxins, and we identified the source. Immediate and long-term corrective actions were recommended and implemented. These were successful (Winter of 2015).

155. **Assessed which party was responsible for salt dome failure:** We were retained to evaluate why several major salt domes failed in Louisiana. This required the use of chemical fingerprint. (Mid-2015).

156. **Assessed hazards associated with organic cosmetics:** We were retained by a firm who had concerns about the possible level of bacterial growth that might be associated with selling purely organic face creams and other cosmetics since no preservatives were present. We conducted an evaluation of several different products and several different carriers of the main ingredients. Indeed, one needs to be careful when manufacturing these products since reuse by the consumer can produce contamination and subsequent dermal effects. (Spring and Summer of 2016).

157. **Lead and arsenic in vitamin supplements:** We were asked to evaluate whether relatively trace concentrations of both arsenic and lead, which were naturally occurring in some zeolites, in a vitamin supplement, posed an unacceptable risk to those taking these nutraceuticals. FDA and California Government were uncertain whether to request withdrawing them from the market (Summer of 2015).

158. **Take-home hazard due to 1950s exposure to crocidolite in paper:** One client asked us to evaluate whether a person who made paper, in the 1950s, might have the potential of causing a take home hazard to his children (Summer of 2015).

159. **Risks of boiler fly ash to local streams:** Was asked by a major utility company to assess their liabilities under the new (2015) EPA coal ash disposal regulations. They had concerns about the amount of ash that had entered streams due to historical piling on or near their site (Summer of 2015).

160. **Evaluated the possible hazards to athletes posed by the use of recycled rubber products in artificial running track:** In about 2016, we were retained to evaluate the loss of volatile organics from turf that was made from shredded tires. The claim was that athletes were getting ill because the tires had not been adequately dried.

161. **Evaluated the possible health hazards to athletes posed by the use of recycled rubber products in artificial grass:** In about 2015, we were retained to evaluate the possible hazards associated with lead in artificial turf. The claim was that the lead was not sufficiently bound to the polymers in the grass and that dermal absorption of lead was a hazard not sufficiently evaluated by the manufacturers.

162. **Evaluated the possibility of skin irritation and damage due to use of an anti-inflammatory cream used by athletes to relieve muscle aches:** There were allegations in around 2015 that use of a new cream was causing irritation, and some skin burns in athletes. We were retained to evaluate the validity of the claims and to study the dermal toxicity of some components of the cream.

163. **Erionite hazard associated with Western U.S. soils:** A number of clients were seeking advice regarding the possible mesothelioma hazard associated with the presence of erionite (of which there are nearly 20 forms) in several hundred locations in the Western States (Spring of 2015).

164. **Asbestos in various laboratory products:** A client approached us to determine the magnitude of health risks (if any) associated with working with historical chrysotile containing laboratory products. These included Bunsen burner heat distributors, asbestos gloves, asbestos blankets and the covering of thongs used to handle very hot test tubes (Summer of 2015).

165. **Arsenic in various wines:** It was reported in a number of national news stories that excessive amounts of arsenic were present in virtually all wines. Several major lawsuits were filed. We were asked to look into this matter. At our expense, we conducted the most comprehensive research ever conducted on this topic which included speciation of the type of arsenic and the presence in a range of wine and red wines. The results were published in two papers in 2016 (work conducted in 2015). (Monnot, A., B.E. Tvermoes, R. Gerads, H. Gurleyuk, and D.J. Paustenbach. 2016. Risks associated with arsenic exposure resulting from consumption of California wines sold in the United States. Food Chem. 211.107-113.; Paustenbach, D.J., A.L. Insley, J.R. Maskrey, J.L. Bare, K.M. Unice, V.B. Conrad, L. Iordanidis, D.W. Reynolds, K.D. DiNatale, and A.D. Monnot. 2016. Analysis of total arsenic content in California wines and comparison to various health risk criteria. Am J Enol Viticult. 67(2).179-187.) (2015 and 2016).

166. **Evaluated the health risks of plasticizers on auto coverings:** At the request of at least one client, we measured the amount of plasticizer on the surface of a variety of autos and then estimate the potential dermal intake. A health risk assessment was also performed. The results were published in a journal in 2017 (work conducted in 2015). (Perez, A., M. Liong, K. Plotkin, K.P. Rickabaugh, and D.J. Paustenbach. 2017. Health risk assessment of exposures to a high molecular weight plasticizer present in automobile interiors. Chemosphere. 167(1).541-550.)

167. **Diacetyl and other gases from processing coffee:** Quite a number of possible clients contacted us to determine if all the discussion in the press of a possible diacetyl hazard due to being a barista was valid. Over the course of months, we assessed this matter. Manuscripts on our work were published (summer of 2015) (Pierce, J.S., A. Abelmann, J.T. Lotter, C. Comerford, K. Keeton, and B.L. Finley. 2015. Characterization of naturally occurring airborne diacetyl concentrations associated with the preparation and consumption of unflavored coffee. Tox Reports. 2:1200-1208).

168. **Arsenic in inexpensive California wines:** In about Feb/March 2015, Dennis was contacted by the Wine Institute to consider evaluating the presence of arsenic (and lead) in wine grown in historical crops. The legal claims surrounded the fact that in many wines, the concentration of arsenic exceeds the drinking water guideline. A couple research papers were generated surrounding this risk-based question (Spring of 2015).

169. **Diesel exhaust and railroad workers:** We were retained by a large railroad to investigate claims that diesel exhaust from historical engines had increased the risk of lung cancer. This has been a hotly debated topic for dozens of years and the recent "re-analysis" of epidemiology literature is the central theme with respect to understanding current views about this possible hazard (Spring of 2015).

170. **Tremolite in vermiculite:** Was requested by a client to evaluate the plausible health risks of asbestosform and non-asbestoform tremolite in various products that contained vermiculites in the 1980s (Spring of 2015).

171. **Phthalates in vinyl auto seat covering:** Due to an unusual set of circumstances on the East Coast, a manufacturer of a very high-end auto was alleged to have upholstery which released a white powder or liquid when the seat warmer was initiated. A risk assessment of the phathlate in the vinyl was performed. (Winter of 2014) (Perez, A.L., Liong, M., Plotkin, K., Kickabaugh, K.P., Paustenbach, D.J. Health risk assessment of exposures to a high molecular weight plasticizer present in automobile interiors. 2017. Chemosphere. 167.541- 550.)

172. **Crocidolite asbestos in pipe coverings:** A number of cases were filed where the claim involved exposure to crocidolite which was present in the tar of some pipe coverings in the 1960s to 1980s. The evidence that this was true was lacking but we performed a health risk assessment nonetheless (Winter of 2014).

173. **Accidental exposure to pharmaceuticals due to shipping hazard:** A single person apparently dropped a vial of some research drug and claimed that they received a dose which was problematic (Winter of 2014). We performed a quantitative evaluation of the airborne and dermal exposures.

174. **Investigated whether diacetyl was present in appreciable amounts during coffee roasting:** We conducted a study to determine the airborne concentrations of diacetyl associated with coffee roasting. We were surprised that the concentrations were much greater than what NIOSH considered likely to be hazardous (Winter of 2014). (Gaffney, S.H., A. Abelmann, J.S. Pierce, M.E. Glynn, J.L. Henshaw, L.A. McCarthy, J.T. Lotter, M. Liong, and B. Finley. 2015. Naturally occurring diacetyl and 2,3-pentanedione concentrations associated with roasting and grinding unflavored coffee beans in a commercial setting. Tox Reports. 2.1171- 1181.; Pierce, J.S., A. Abelmann, J.T. Lotter, C. Comerford, K. Keeton, and B.L. Finley. 2015. Characterization of naturally occurring airborne diacetyl concentrations associated with the preparation and consumption of unflavored coffee. Tox Reports. 2:1200-1208).

175. **Risk of disease posed by take home exposure of either chrysotile or amosite:** A number of cases involving this issue came to our attention in 2012-2015. To address this matter, we conducted a series of "simulation studies" which were published in the peer reviewed literature in 2013-2015. This was the first quantitative assessment of this matter. (Sahmel, J., C.A. Barlow, S. Gaffney, H.J. Avens, A.K. Madl, J. Henshaw, K. Unice, D. Galbraith, G. DeRose, R.J. Lee, D. Van Orden, M. Sanchez, M. Zock, and D.J. Paustenbach. 2016. Airborne asbestos take- home exposures during handling of chrysotile-contaminated clothing following simulated full shift workplace exposures. J Exp Sci Environ Epidemiol 26.48-62.; Sahmel, J., C.A. Barlow, B. Simmons, S.H. Gaffney, H.J. Avens, A.K. Madl, J. Henshaw, R.J. Lee, D. Van Orden, M. Sanchez, M. Zock, and D.J. Paustenbach. 2014. Evaluation of take-home exposure and risk associated with the handling of clothing contaminated with chrysotile asbestos. Risk Anal. 34(8).1448-68.).

176. **Assessment of the airborne hazards from a fire involving a refinery:** We were asked to share our views about the potential short term and chronic hazards posed by the direct and indirect exposure to emissions from a refinery fire in Northern California. We have participated in ten or more similar assessments over the years (Summer of 2014).

177. **Assessing the occupational risk of benzene and butadiene in a non-refinery setting:** We evaluated 10,000 industrial hygiene samples collected in a chemical plant over the course of 30 years and then conducted a health risk assessment of the workers (Spring of 2015).

178. **The sensitization hazard posed by cobalt, chromium and nickel released by hip implants:** Was retained by a manufacturer to determine the likelihood that these metals, when released through normal wear, were likely to induce or elicit a sensitization response in patients. (spring of 2015). (Kovochich, M., E.S. Fung, E. Donovan, K.M. Unice, D.J. Paustenbach, and B.L. Finley. 2017. Characterization of wear debris from metal-on-metal hip implants during normal wear versus edge- loading conditions. J Biomed Mat Res B. Advanced online publication, May 8, 2017. doi. 10.1002/jbm.b.33902; Unice, K.M., A.D. Monnot, S.H. Gaffney, B.E. Tvermoes, K.A. Thuett, D.J. Paustenbach, and B.L. Finley. (2012). Inorganic cobalt supplementation. Prediction of cobalt levels in whole blood and urine using a biokinetic model. Food Chem. Toxicol. 50.2456-2461.; Tvermoes, B.E., B.L. Finley, K.M. Unice, J.M. Otani, D.J. Paustenbach, and D.A. Galbraith. 2013. Cobalt whole blood concentrations in healthy adult male volunteers following two-weeks of ingesting a cobalt supplement. Food Chem Tox. 53.432-439.).

179. **MCHM release into the Elk River:** Was retained by a client to assess the possible health risks associated with ingestion of a coal dust chemical by citizens in West Virginia. This incident occurred in January of 2014 and retention was shortly thereafter. (Paustenbach, D.J., B. Winans, R.M. Novick, and S.M. Green. 2015. Toxicity of crude 4- methylcyclohexanemethanon (MCHM). Review of experimental data and results of predictive models for its constituents and a putative metabolite. Crit Rev Tox. 45(S2).1- 55.

180. **Assessing the hazards posed by talc with respect to ovarian cancer:** Was retained by a pharmaceutical firm to assess the plausible hazard associated with the use of baby powder containing talc and the risk of ovarian cancer (Fall 2014 - Spring 2018).

181. **Advisors to Australian Dept of Agriculture:** Were retained to conduct and/or peer-review a series of health risk assessments associated with the use of various pesticides (Summer 2014).

182. **Conducted follow-up epidemiology study of diacetyl workers:** We conducted a follow- up study of the health status of workers at an Indiana food flavorings facility who had been employed for as many as 30 years in this industry (begun in early 2013, analysis should be published in late 2015). This study was the largest one ever conducted in that 10 or more years of PFT data were evaluated.

183. **Asbestos exposure associated with repairing motorcycles:** In the summer of 2014, we evaluated the plausible historical exposure to asbestos associated with repairing the brakes on motorcycles sold and repaired from 1950 to 1990.

184. **Asbestos exposure while cutting metal sheeting (1950-1980):** We conducted a simulation study involving traditional and non-traditional approaches to cutting certain forms of metal wall board which contained chrysotile asbestos (Summer 2014).

185. **Asbestos hazard in those who worked in power plants:** We conducted an exposure assessment of maintenance workers and craftsmen who worked in the electric power generating industry from 1950-1990 (Summer 2014).

186. **Mini-epi study of large office building:** For a confidential client, we were asked to assess whether there was any validity to claims of a "cancer cluster" associated with working in a particular building (Summer 2014).

187. **Asbestos in electrical components (1950-1980):** We were asked by a former manufacturer of electrical components made of both plastic and Bakelite, which contained chrysotile, whether it was plausible to be exposed to biologically relevant airborne concentrations of asbestos while manipulating these products (Summer 2014).

188. **Evaluated the take-home hazard associated with silica:** In the summer of 2014, we were asked to qualitatively and quantitatively evaluate the plausible workplace take home hazard associated with crystalline silica. This turned out to be a five- year-long project.

189. **Nanoparticles and hip implants:** During 2012-2014, we conducted an evaluation of alleged health risks associated with the release of nanoparticles (and other wear debris) from cobalt-chromium containing metal on metal hip implants. Several papers were published describing our work. (Madl, A.K., M. Liong, M. Kovochich, B.L. Finley, D.J. Paustenbach, and G. Oberdorster. 2015. Toxicology of wear particles of cobalt-chromium alloy metal-on-metal hip implants Part I: Physiochemical properties in patient and simulator studies. Nanomed: Nanotech Biol Med. 11(5):1201-1215; Madl, A.K., M. Kovochich, M. Liong, B.L. Finley, D.J. Paustenbach, and G. Oberdorster. 2015. Toxicology of wear particles of cobalt- chromium alloy metal-on-metal hip implants Part II: Importance of physiochemical properties and dose in animal and in vitro studies as a basis for risk assessment. Nanomed: Nanotech Biol Med. 11(5):1285-1298.

190. **State-of-the-art employee responsibilities:** During 2013 and 2014, we evaluated a couple dozen cases which involved an understanding of the state-of-the-art duties of employers to warn employees about the hazards of asbestos (1955- 1985). (Barlow, C.A., J. Sahmel, D.J. Paustenbach, and J.L. Henshaw. 2017. History of knowledge and evolution of occupational and health and regulatory aspects of asbestos exposure science. 1900-1975. Crit Rev Tox. 47(4).286-316.

191. **Conducted a review of the literature regarding the evidence that diacetyl was the cause of lung disease in food flavorings workers:** Between 2007-2015, ChemRisk evaluated various studies and the evidence that suggested that exposure to diacetyl, or in combination with other chemicals, was the likely cause of one or more different occupational lung diseases. We worked for several clients and developed a comprehensive evaluation of these claims which we considered, in the main, to be lacking sufficient scientific evidence that diacetyl was the causative agent. Our analyses were presented in several published papers or letters-to-the-editor. (Pierce, J.S., A. Abelmann, and B.L. Finley. 2014. Response to Muge Akpinar-Elci Letter to the Editor re. Pierce et al., Diacetyl and 2,3- pentanedione exposures associated with cigarette smoking: implications for risk assessment of food and flavoring workers, Crit Rev Toxicol. 44(5):420-435. Crit Rev Tox. 44(7):640-641.; Finley B. and Scott P. 2014. Diacetyl and "popcorn lung" litigation. Assessing the evidence for general causation. ABA Environ Litig Toxic Torts Newsletter. 15(2).7-10.

192. **Assess the hazard posed by Agent Orange:** We evaluated the hazard to maritime workers who handled drums of Agent Orange during the Vietnam War (exposure was alleged to be associated with cleaning up leaking drums) (Spring of 2014).

193. **Assessed claims that exposure to dioxin, at nearly any concentration, increased the risk of diabetes:** We were retained to examine a group of patients who claimed exposure to dioxin and an increased incidence of diabetes. This claim had been evaluated by the NAS and found to be without merit, but some did not find this convincing. We conducted a different analysis and found that reverse causation explained the results. (Kerger, B.D., P.K. Scott, M. Pavuk, M. Gough, and D.J. Paustenbach. 2012. Re-analysis of Ranch Hand study supports reverse causation hypothesis between dioxin and diabetes. Crit Rev Tox. 42(8):669-87.

194. **Assessed exposure to diacetyl associated with roasting coffee beans:** We evaluated the possible exposure to diacetyl due to roasting and blending coffee beans (diacetyl was naturally occurring). We found exposures above the current TLV (Spring 2014).

195. **BPA in food containers:** A large manufacturer of containers used in food storage asked us to conduct a quantitative risk assessment, as well as a regulatory analysis, regarding the presence of BPA in foods due to be stored in the containers (Spring of 2014).

196. **Conducted an assessment of the health hazards of trace microbial contamination in a Kale snack product:** In the spring of 2014, we evaluated whether Listeria innocua at low concentrations posed a health hazard to consumers, and we recommended approaches for eliminating future contamination. We also insisted that foods in process and transit be recalled.

197. **Evaluated diesel exhaust emissions from power generators near fracking fields:** We evaluated the quantity of various emissions, as well as the airborne concentrations with distance from these generators, which are often relatively close to neighborhoods in various parts of the nation (Spring of 2014).

198. **Evaluation of brominated flame retardants in paints:** In the spring of 2014, a major producer requested a quantitative risk assessment of this class of flame retardants which had been added to paints for a number of years.

199. **Food additive in lollipops:** A candy manufacturer had concluded that an additive which was not approved in the EU, but approved by US FDA, had been used in some products sold in Europe. We evaluated whether this chemical posed a hazard to consumers and whether the relevant agencies agreed with our assessment (Spring of 2014).

200. **Lead paint in Baltimore homes:** We were asked to evaluate recent claims that the remediation of lead from homes in Baltimore had actually increased the blood lead concentrations in children living in those homes (Spring of 2014).

201. **Retained to evaluate the "state-of-art" expectations regarding toxicity testing of the chemicals released as a result of Freedom Chemical incident in West Virginia:** A possible defendant in this case asked us to evaluate the toxicology and warning label history associated with the chemicals that leaked from tanks at this facility. (Spring 2014). (Paustenbach, D.J., B. Winans, R.M. Novick, and S.M. Green. 2015. Toxicity of crude 4- methylcyclohexanemethanon (MCHM): Review of experimental data and results of predictive models for its constituents and a putative metabolite. Crit Rev Tox. 45(S2):1- 55.

202. **Conducted epidemiology study of diacetyl workers:** We evaluated the health status of workers at an Indiana food flavorings facility who had been employed for as many as 20 years in this industry (Begun in early 2013; analysis was published in early 2014). (Ronk, C.J., D.M. Hollins, M.J. Jacobsen, D.A. Galbraith, and D.J. Paustenbach. 2013. Evaluation of pulmonary function within a cohort of flavorings workers. Inhal Tox. 25(2).107-117.

203. **Epidemiology study of persons having hip implant:** In late 2013, we were asked to design and possibly implement an epidemiology study involving persons who had received total hip replacements over the past 15 years. Study was conducted in conjunction with a major university. Completed in 2016. The data sets that we examined were not sufficiently robust to reach a conclusion.

204. **Conducted an evaluation of diacetyl emissions from cigarettes:** In the fall of 2013, we assessed the amount of diacetyl released from cigarettes and compared it to what food workers were receiving. Results were published in a peer-reviewed journal in early 2014. (Pierce, J.S., A. Abelmann, L.J. Spicer, R.E. Adams, and B.L. Finley. 2014. Diacetyl and 2,3-pentanedione exposures associated with cigarette smoking. Implications for risk assessment of food and flavoring workers. Crit Rev Tox. 44(5).420-435.

205. **Quantitatively assess the possible financial liabilities of a firm who contributed to a global asbestos settlement in 1988:** We were asked to assess the state of knowledge in the late 1980s regarding what the plausible financial exposure of a company might be given information on court cases at the time (and incidence of disease). Claims were that insufficient funds had been reserved (Fall of 2013).

206. **Funded to continue evaluation of tire related environmental hazards:** In the Fall of 2013, our contract with the European Tire and Rubber Association (ETRMA) was renewed, so that we could continue to assess whether there are any human and ecological hazards associated with tire wear particles. This project is considered by many to be the best example of a product sustainability effort by nearly any major industry. More than ten papers have been published that discuss this work.

207. **Retained to evaluate the hazard of PCBs in caulking in schools It was determined around 2005 that PCBs had been used in silicone and other caulking materials for many years in the United States:** This issue was addressed in the EU in the 1990s and early 2000s. New claims about the hazard surfaced in the fall of 2013, and we got involved in conducting a state-of-the-art risk assessment.

208. **Involvement in Garlock case involving reasonableness of claims:** We were retained to evaluate whether a major manufacturer of gaskets had properly assessed the health hazards associated with their products which contained chrysotile. A fairly comprehensive exposure assessment of many crafts was conducted. This was rated by Law360 as one of the most important cases of 2013 (Summer/Fall of 2013).

209. **Contaminants in apple juice:** During the summer of 2013, we were asked to evaluate the plausible health hazards associated with various chemical contaminants in apple juice sold primarily to the children's market. Our work was published in a peer-reviewed journal in mid-2014. (Tvermoes, B.E., A.M. Banducci, K.D. Devlin, B.D. Kerger, M.M. Abramson, I.G. Bebenek, and A.D. Monnot. 2014. Screening level health risk assessment of selected metals in apple juice sold in the United States. Food Chem Tox. 71(9).42-50.

210. **Evaluated hazards associated with disposing photovoltaic cells (PVC):** We evaluated the possible health and environmental hazards of disposing of damaged or exhausted PVC cells into municipal landfills (Summer of 2013).

211. **Evaluation of contaminated clothing manufactured in China:** We evaluated the hazards associated with the presence of various "unapproved" dyes in clothing manufactured in China In other related cases, we evaluated the presence of lead in handbags and other leather items (Summer of 2013).

212. **Preservative and fragrance added to soap:** For a confidential client, we evaluated whether a health hazard was posed by the addition of an unapproved fragrance to a large number of bars of soap (Summer of 2013).

213. **Evaluated airborne hazard associated with fracking:** On a pro-bono basis, in the Spring of 2013, we evaluated the airborne concentrations of various volatile chemicals in a school near to a fracking operation in Western Pennsylvania. No significant exposures were found.

214. **Evaluated the hazards associated with the presence of unapproved preservative [1,2 benzisothiazolin-3-one (BIT)] in marking pens:** In the spring of 2013, we were retained by a firm who, in their China facility, had accidently used an unapproved dye in their marking pens. A toxicology and exposure assessment were conducted. Results were published in a peer-reviewed journal. (Novick, R.M., M.L. Nelson, K.M. Unice, J.J. Keenan, and D.J. Paustenbach. 2013. Estimation of the safe use concentrations of the preservative 1,2-benzisothiazolin-3-one (BIT) in consumer cleaning products and sunscreens. Food Chem Tox. 56.60-66.

215. **Evaluated claims of inadequate funding of future product defect claims:** In the spring of 2013, we were retained to do a state-of-the-art analysis of what was known in 1986 (and at various times after that) about the anticipated number of asbestos related health claims that might plague an industry over the next 30 years (through 2016). The claims involved suggestions that not enough money had been held in reserve prior to sale of the firm, considered a major "bellwether case."

216. **Reviewed corporate sustainability plan:** We were asked to peer-review and suggest improvement in a large tech firm's sustainability program (from supply chain to disposition in a landfill) (Spring of 2013).

217. **Evaluated asbestos hazard to railroad workers:** In the spring of 2013, we performed an assessment of workers who had been long term employees of the railroads. We evaluated alleged exposure to asbestos while performing a number of tasks throughout their career.

218. **Assessed the health risks associated with cell phone battery off-gases associated with catastrophic failure:** In 2013, we were retained to study the airborne chemicals released during the destruction of cell phone batteries and the associated short-term risks to health.

219. **Hazards associated with surface spills associated with hydraulic fracking:** On a pro bono basis, we evaluated the possible risks to the environment associated with unanticipated spills around operating platforms associated with fracking units. Our work focused on Colorado. This work was published in J Air and Waste Management (Gross, S.A., H.J. Avens, A.M. Banducci, J. Sahmel, J.M. Panko, and B.E. Tvermoes. 2013. Analysis of BTEX groundwater concentrations from surface spills associated with hydraulic fracturing operations. J Air Waste Manage Assoc. 63(4).424-432.

220. **Evaluation of the hazards associated with systemic exposure to cobalt:** During 2012 and 2013, we conducted a series of volunteer studies where workers were exposed via ingestion to cobalt supplement. The objective was to evaluate claims of toxicity at certain blood concentrations associated with cobalt leaching from hip implants and any claims regarding adverse effects due to taking it as a vitamin supplement. The studies were published in a series of articles in 2013 and 2014.

221. **Dose reconstruction in the petrochemical and refining industry:** In the Fall of 2012, a major oil company retained us to conduct dose reconstructions for as many as five of their refineries (some of which also had chemical plants).

222. **Evaluating the occupational exposure of pressmen to benzene (1938-2006):** In 2012, we were retained by a solvent manufacturer who sold benzene to companies who made ink solubulizer which was used in the printing industry. There were claims that pressmen had been overexposed to benzene as a result of this (and similar) products which contained 2- 50% benzene).

223. **Conducted an evaluation of the risks associated with having slightly lesser quantities of supplemental nutrients/vitamins in flour:** During the spring of 2012, we were retained by a major food company and evaluated whether a 10% lesser (or greater) amount, than specified by FDA of certain additives, posed any health risks to consumers.

224. **Evaluated the comparative hazards of removing and not-removing asbestos containing floor tiles in nearly 500,000 apartments in New York City and Northern New Jersey:** We were asked whether the risks were greater with removing these tiles compared to the hazard associated with the cleaning/repair of these tiles going forward (e.g., leaving them in place) (Winter of 2012).

225. **Formaldehyde exposure to hair stylists. It had been alleged in 2011 (and previously) that the concentrations of formaldehyde in some hair straighteners was causing eye, nose and throat irritation in some hair stylists:** We concluded that many claims were apparently accurate and confirmed this through a simulation study (Pierce, J.S., A. Abelmann, L.J. Spicer, R.E. Adams, M.E. Glynn, K. Neier, B.L. Finley, and S.H. Gaffney. 2011. Characterization of formaldehyde exposure resulting from the use of four professional hair straightening products. J Occup Env Hyg. 8(11).686-699.

226. **Provided assistance in obtaining a pre-manufacture notice (PMN) for a new adhesive used to make modern-era passports:** An American firm asked us to work with both Chinese and Japanese firms to facilitate receiving approval to produce a new glue which had previously only been available in East Asia. This was completed by the end of 2011.

227. **Assessed the risk associated with PFOA or PFOS in MSWTP sludges:** We evaluated the threat to groundwater of using sludges from water treatment plants as soil amendment in farm fields. This practice was apparently common in Alabama and Mississippi where there were textile mills treating carpets with stain repellents (Fall of 2011).

228. **Evaluated the alleged hazard of lead in Lake Merced (and the sources):** Retained by Zurich Insurance to assess the overall risks to the environment and humans, as well as identify the source of the lead (Fall of 2011).

229. **Hazard of housewives to asbestos in the 1950s-1980s:** Over the past five years, there has been a significant increase in the number of mesothelioma cases that are alleged to be associated with exposure to any form of asbestos due to “take home” exposures. From about 2007 to 2011, ChemRisk has been studying this issue with respect to auto mechanics, pipefitters, insulators, phenolic molding compound manufacturers, dry wallers, and numerous other occupations, which have been claimed to pose a “take home” risk to family members. The definitive review on the topic was published in a peer reviewed journal in 2012.

230. **Assessed possible human health hazard of inadvertent use of non-food grade additives in flour:** During the summer of 2011, ChemRisk was asked to evaluate whether the use of non-food grade azo (a trace additive to commercial flour), rather than the food grade version, might pose a health hazard. The additive was made in Korea. Working in conjunction with a well-known law firm, we conducted a comparative analysis and submitted our work to the FDA.

231. **Evaluated the degradation of potassium sorbate (a food preservative) in salty foods:** We were asked in the Summer of 2011 to assess the importance of the degradation of this food preservative in snack foods. Interestingly, this additive degraded by as much as 80 percent in foods before their shelf life had expired.

232. **Evaluated the presence of dioxins, furans, PCBs and flame retardants in shrimp from developing countries:** In conjunction with a famous analytical laboratory, ChemRisk conducted a study of nearly 30 different sources of shrimp to the U.S. food market which were both farm raised and natural. This was the first comparative assessment of its type involving shrimp from 12 countries. The results were reported at 3 national scientific meetings and in two journal articles (2008- 2011). This work was performed as a public service by ChemRisk.

233. **Evaluation of the toxicity of cobalt:** Beginning in July (2011) we began evaluating the potential toxicity of cobalt for a major pharmaceutical and medical device firm.

234. **Retained to characterize the mechanism by which metal-on-metal hip implants were needing revision:** In the summer of 2011, we began evaluating the various biological mechanisms which might cause an implant to need to be replaced. Our research on the mechanism continued through 2014-2018.

235. **Evaluated the possible "take home" exposure (and risks) of asbestos on clothing:** Beginning in mid-2011, we undertook research directed at evaluating the likelihood that biologically important quantities of asbestos might have routinely been taken home by workers who handled asbestos during the 1940-1960s era. The evaluation attempted to assess the validity of various research efforts of the 1960s-1980s which indicated that only a limited quantity of respirable fibers which were present in contaminated clothing were likely to be released in the in-home environment. We funded the initial work, and it was discussed at various scientific conferences in 2012 and 2015. (Donovan, E., B.L. Donovan, M.A. McKinley, D.M. Cowan, and D.J. Paustenbach. 2012. Evaluation of take home (para- occupational) exposure to asbestos and disease. A review of the literature. Crit Rev Tox. 42(9).703-731.

236. **Assessed the possible inhalation hazard associated with the manufacture of a select family of food flavorings (including diacetyl):** In mid-2011, we evaluated the historical pulmonary function tests of workers in a food flavorings plant in an attempt to determine if their work had produced an increased incidence of lung disease (much like claims that diacetyl had caused mild to serious lung illnesses).

237. **Assessment of the human health hazard posed by asbestos containing floor tiles:** In mid-2011, we were retained to evaluate the historical and future health risks associated with living in apartments which had asbestos containing floor tiles manufactured from about 1935-1975. The assessment was used to resolve financial claims associated with renovating apartments which, in total, contained more than 10,000,000 square feet of these tiles. Publication is in progress in 2017.

238. **Conducted an overall chemical hazards analysis of a new chemical process to be used to manufacture the next generation of passports:** During the first half of 2011, for a combined private sector and government entity, we evaluated the chemicals used (and related emissions) to manufacture a new type of passport that is believed to be above counterfeiting.

239. **Conducted product sustainability assessment of various household products:** In 2011, ChemRisk was asked to evaluate a number of consumer products produced in China and Vietnam to ensure that no chemicals were present at concentrations that might be considered a health hazard by any reasonable regulatory body (including California Prop 65) or non-government organization. This was one of a number of related evaluations of products produced in developing countries.

240. **Evaluated the inhalation hazard associated with using solvents containing less than 0.1% benzene sold between 1955-1975:** During these years, a number of paints and solvents were sold into commerce that contained relatively small concentrations of benzene (usually as a trace contaminant). Allegations have been made that although OSHA concluded that these cleaners/solvents did not pose a significant human health hazard, literally thousands of lawsuits were filed between 2007 and the present asserting that this was not the case. We were retained in 2011 by numerous clients to evaluate their own data or data from simulation studies to estimate exposure of workers and any related health hazards.

241. **Evaluated the human health hazard posed by contaminated cheese:** In May 2011, we evaluated the safety and health hazard associated with the presence of plastic chips and strands from a process filter in large quantities of bulk cheese. The various potential possible adverse effects, as well as risks to the business were assessed.

242. **Conducted an assessment of the cause of death for two citizens:** We were asked to try to identify how two adults died of carbon monoxide poisoning associated with falling asleep in a mobile recreational vehicle. We conducted an exposure assessment and concluded that an unfortunate series of events had to occur for this event to happen (Spring of 2011).

243. **Conducted a scientific evaluation regarding the basis for a class certification:** As ChemRisk has been asked numerous times in the past, there are essential elements which support the appropriateness of certifying a class for processing by the courts. We evaluated an incident where thousands of pounds of an irritant were volatilized in a relatively short period of time and dozens of residents reported transient irritant symptoms. This incident was evaluated and recreated; and testimony was provided in New Orleans in the spring of 2011; client was Dow Chemical.

244. **Assessment of the presence of isothiazolinone in paints and paper markers:** In early 2011, some firms were using this stabilizer/preservative in marking pens at very low concentrations. In recent years, there has been increasing concerns about whether this known skin sensitizer might pose a hazard if it remained in commerce. As part of a product sustainability assessment, we attempted to identify the threshold dose at which this additive might produce some response in already sensitized persons, as well as compare that dose against guidelines being proposed or implemented in various countries. This work was published in a peer- reviewed journal.

245. **The hazards of having used crocidolite in some phenolic molding materials:** In early 2011, we were retained to evaluate the possible health hazard of using crocidolite as a filler in some materials made from phenolic molding compounds produced from 1940-1970.

246. **Assessed the possible hazard to airplane mechanics of brake wear debris from airplanes:** Like most brakes manufactured from 1910-1980s, asbestos was a major constituent of those used in airplanes. In early 2011, we evaluated this hazard and attempted to quantitate the relative magnitude of historical exposure of these mechanics.

247. **Assessing the risks of chips of asphalt covered asbestos fabric in soils:** During early 2011, ChemRisk began quantifying the possible human health hazards associated with having various size scraps (usually less than 1 x 1”) of asbestos cloth (covered on one or two sides with mastic) present in soils predominately located in farm fields. These were present due to the removal of pipes which were part of the national oil pipeline. At the time, various early guidance was being developed to help direct such assessments. Many thousands of acres were potentially contaminated.

248. **Assessed claims regarding the historical hazard posed by chrysotile fabric in radios and televisions produced between 1950-1975:** For some of these appliances, it was deemed appropriate to have one or more asbestos containing parts so as to minimize the likelihood of failure or a fire. Some claims had been made that repairmen might have been at risk of developing asbestos related disease. Evaluation was conducted in 2010 and 2011.

249. **Assessed the hazards posed by dioxin in poultry contaminated with dioxins in Germany:** In January 2011, provided pro bono advice to a regulatory agency who was trying to assess the human health hazard associated with having fed dioxin- tainted oils (with feed) to chickens which then had a "higher than normal" concentration of dioxins and furans.

250. **Assessed the health risks posed by contamination of cookies (via contaminated air) by the local use of pesticides to control insect infestation:** In late 2010 and into 2011, we were asked to determine whether the peanut butter cookies sold by this organization might have absorbed amounts of pesticides that would have compromised their edibility (all due to pesticide use in the warehouse where final product cookies were stored). We recommended destroying this product.

251. **Evaluated claims of contamination of sediments involving the Presidio:** In November 2010, ChemRisk performed a critical evaluation of the scientific bases for remediating sediments in an important lake on the former military site. The client was Zurich Insurance Company. Testimony was provided. The courts agreed with our analysis and reduced Zurich liability by almost $300,000,000.

252. **Evaluated the life cycle and sustainability issues related to photovoltaic cells (PVC):** This industry is attempting to modify the way it produces cells and wishes to minimize the adverse impact on the environment of their ultimate disposal. ChemRisk began work with several firms in the industry in late 2010 and this evolved in some applied research which ChemRisk conducted and discussed at scientific meetings.

253. **Conducted a preliminary epidemiology study of the number of increased ankle injuries due to running on artificial turf:** In about 2010, we were retained to respond to claims that cleats used on genuine grass turf, when being used on artificial turf, caused an increased number of ankle injuries. We conducted a preliminary study of injuries of high school and college students in several different leagues.

254. **Evaluating the health risks to the remediation teams, on the boats, at the British Petroleum (BP) oil spill in Louisiana:** In the Spring and Summer of 2010, the BP oil rig off the Louisiana coast was the cause of the largest oil spill in history. ChemRisk was contacted by both PRPs about the possible benzene, xylene and toluene hazards to the boatman. Nearly 35,000 samples were evaluated and, interestingly, we found that the concentrations of benzene, toluene, xylene and ethylbenzene were no greater during the remediation than during regular use of the boats for fishing. These results were presented in the fall of 2010 at two major scientific meetings. The results were published in a major journal. No compensation was received.

255. **Provided assistance to patent lawyers regarding protection of a patent on a process for removing PBDE flame retardants from pharmaceutical drugs:** One of the new blockbuster drugs now on the market is based on concentrating oils from fish caught in Scandinavian countries that are known to contain considerable concentrations of dioxins, furans, PCBs and PBDEs. In the summer of 2010, ChemRisk assisted in helping them understand the possible hazards and evaluated their patent for removing these chemicals.

256. **Evaluated an episodic exposure to hydrogen sulfide at a refinery:** In mid-2010, we were asked to evaluate a case involving a young man who had allegedly been exposed for about 10 minutes to concentrations of 4-50 ppm of hydrogen sulfide. About 19 months after the incident, he went into a comatose state or vegetative state and some suggested that it was due to his exposure during this incident. Weeks of work were invested in recreating the incident and testimony was offered as to the event and the likely cause of his disease.

257. **Attempted to determine the cause of contact dermatitis in babies wearing specific types of clothing:** In mid-2010, ChemRisk was retained by a major supplier of children's clothing to try to understand the cause of dermatitis that was present on the backs of some children who wore certain underclothing which had a stenciled label. This case involved claims of a sensitization response to a dye in the label. It was a project not unlike others ChemRisk has tackled involved chemicals that cause allergic contact dermatitis at very low concentrations. About 4,000 children may have been temporarily affected.

258. **Evaluated the plausible historical exposure of persons who were "bystanders" to those workers actively involved in handling asbestos products during the 1930-1975 era:** Over the course of several years, which culminated in the presentation of our work at a couple scientific conferences in 2009 and 2010, ChemRisk developed a predictive model based on empirical data for characterizing the exposure of persons working within 5 to 50 feet of asbestos workers. The work was published in a peer-reviewed article in 2010.

259. **Evaluated claims (late 2009 and early 2010) that exposure to petroleum catalysts used in the production of crude oils might have caused acute and chronic respiratory disease:** ChemRisk was asked to investigate the individual exposure records, including medical records for those persons who claimed injury. It was their view of the plaintiffs that handling dry catalysts, including spent catalyst, posed a special constellation of diseases, primarily due to the metal content. The medical and Industrial hygiene records failed to support that claim and the client, HOVIC, was not found liable for any alleged diseases (U.S. Virgin Islands). Our evaluation of the catalysts was published in a peer- review journal.

260. **Assessed the developmental hazard of inks used in various printers:** Allegations of developmental effects in children were made against select manufacturers of printers who used certain inks. We conducted an assessment of the relevant historical toxicology data and conducted an exposure assessment.

261. **Evaluated the hazards posed by methylene bisphenyl isocyanate (MDI) in foams used in coal mines:** A ten-year long litigation involved claims that MDI vapor (and perhaps contact with the skin) emanating from a foam used to fill cracks in the walls and ceilings of mines had sensitized hundreds of miners. ChemRisk evaluated all the available data and various reports of experts. Ultimately, these claims were found to be without merit and the case was resolved. Work first performed in 2003 then again in 2010.

262. **Evaluated the historical exposure of tire workers to benzene:** From about 2005-2010, ChemRisk collected industrial hygiene data from a tire manufacturing plant and estimated the typical airborne concentrations in the facility over a period of about 30 years. This study may result in a journal article on this topic.

263. **Evaluated the legitimacy of claims that Coca-Cola was contaminated with elemental mercury:** In November 2009, ChemRisk was contacted by the client that there were claims that persons had been poisoned in Beijing as a result of drinking Sprite from their local manufacturing facility. Previously, ChemRisk had experience in evaluating similar “canning lines” and moved a small team to Beijing to conduct a hazard evaluation of the manufacturing process. ChemRisk focused on the possible presence of elemental mercury and was able to definitively show that no mercury was ever present in that facility. Regulatory authorities in China read our analysis and agreed.

264. **Assessed the adequacy of a new water treatment facility:** In mid-2009, ChemRisk was asked by the City of Sante Fe to evaluate the possible health risks of the treated and untreated water from the local river. The city planned to install a state-of-the art water treatment system and wanted to be sure that certain toxicants, including radionuclides, would be removed to concentrations that were safe for consumption. This was a long and difficult exercise which lasted 2-3 years.

265. **Conducted an evaluation of the hazards posed by bisphenol A (BPA) in various consumer products:** About 2009, as a result of increased social, regulatory and legal interest in BPA (a plasticizer), our staff conducted an evaluation of several consumer products to evaluate the possible perceived or actual human health risks.

266. **Contacted by counsel to assess some of the clinical data surrounding the death of Michael Jackson:** In mid-2009, ChemRisk was contacted by one of the key parties involved in this incident. The question involved whether the combination of chemicals thought to be taken by Mr. Jackson was likely to be fatal (considering dose and duration and pharmacokinetics) and whether the treating doctor may have been negligent. Advice was offered under the privilege, and no compensation was received, and no report was issued.

267. **Pharmaceuticals and personal care products in drinking water (PPCPs):** In mid- 2009, ChemRisk was asked to conduct a state-of-the-art evaluation of the regulatory initiatives, worldwide, which were focused on the possible health hazard posed by PPCPs in drinking water. We did this work first for the City of Melbourne (Australian) and then later for a major bottling company.

268. **Evaluation of complex, batch style, specialty chemicals facility:** In early 2009, on behalf of a large chemical company, ChemRisk evaluated 30 years of industrial hygiene data in an attempt to characterize the likely exposure of workers to about 30 different chemicals. Novel approaches to assessing the data were applied. ChemRisk published those analyses in late 2011.

269. **Hazards of asbestos in airplanes:** In early 2009, ChemRisk assessed for a major client the potential range of exposures to asbestos which might occur during the manufacture and repair of airplanes for the time period 1940-1985. The analyses needed to consider all aspects of exposure, with an emphasis on government knowledge (and Navy knowledge) of asbestos over time. The analysis of state-of- the-art issues was published in a peer- reviewed journal.

270. **Performed comprehensive toxicologic and medical assessment of a wide variety of health and property damage claims related to perchloroethylene (PCE), trichloroethylene (TCE), and other volatile byproducts (TCA, DCA):** In January 2009, ChemRisk performed a comprehensive toxicologic and medical assessment of a wide variety of health and property damage claims in a related case. Exposures were alleged to have arisen from contamination of groundwater, associated with historical releases of various solvents. Summary judgment for the defense was returned on all claims involving our client, Cargill, in consolidated litigation brought by approximately 300 plaintiffs (recruited by Erin Brockovich) in Grand Island, Nebraska.

271. **Evaluation of the vehicle emissions from the M5 tunnel in Sydney, Australia:** The evaluation focuses on how to best use exhaust fans and other control devices to minimize emissions into the surrounding neighborhoods. The work was conducted throughout 2007 and into 2008. The client was the Sydney Regional Transportation Authority. A peer-reviewed manuscript was published describing our evaluation.

272. **Leaking drums of PCBs in the basement of an office building in Japan:** It was believed that a few drums of PCBs had been accidentally stored in the basement of an office building in Tokyo and that they leaked as much as 5 gallons of liquid (which seeped into the concrete). ChemRisk investigated a claim that air had channeled through the walls to a particular floor where persons were alleged to have adverse effects due to the volatilized PCBs (Fall 2008).

273. **Evaluated the risk to children posed by lead in various consumer products produced in China:** During 2007-2008, ChemRisk evaluated the content and possible release of lead from plastics and other media which are used to make a number of consumer products in the United States. Based on these data, a health risk assessment was conducted, and the results were compared to various regulatory and non-regulatory criteria.

274. **Evaluating the hazards of various occupations exposed to silica:** Over a 3-year period (2005-2008), ChemRisk evaluated the history of occupational exposure of sandblasters to silica and the evolution of the associate personal protective equipment. This resulted in two peer-reviewed publications.

275. **Assessment of the hazard posed by asbestos in laboratory equipment:** In mid- 2009, a major supplier of laboratory glassware and related items asked about the possible hazards associated with using metal meshwork pads (which contained asbestos and were used to be used with Bunsen burners) and then handling hot glassware with asbestos gloves. A simulation study may ultimately be performed.

276. **Assessed the minimal lifetime cumulative dose of chrysotile needed to increase the risk of mesothelioma:** Assembled the published literature and estimated the exposure of various cohorts to chrysotile. Determined that doses at least as high as those required to cause asbestosis were required to possibly increase the risk of mesothelioma; if chrysotile even had the capacity to do so. Results were published in a peer-reviewed journal in 2008.

277. **Evaluated the health risks posed by a contaminant in candy:** In 2008, we evaluated the presence of a contaminant in a food that was sold in a number of countries. Assisted the firm in evaluating the possible health risks and interacted with regulatory agencies to answer questions about our assessment. Ultimately, the firm chose to recall some of the product; no litigation occurred.

278. **Analysis of the historical exposure of semiconductor workers to methanol:** Since the 1990s, workers in the quality control department of many companies have cleaned wafers with methanol before placing them in a "flatness/imperfection" machine. In 2007, conducted both modeling and simulation studies to quantitatively measure the acute and chronic airborne concentration of methanol in the workplace. The results were published in a journal in 2008.

279. **A complex analysis (with colleagues) of the historical lifetime cumulative intake of asbestos for auto mechanics in the U.S. over the past 40 years compared to mechanics in Europe:** The data can be used to understand the risk following low level exposure, since at least 7-10 epidemiology studies are available for these cohorts. The work was conducted in 2005, and the manuscript was published in a peer-reviewed journal in 2007.

280. **A study of the exposure of citizens to historical aerial emissions of dioxins and furans from a wire cable recycler/producer:** During 2006 and 2007, conducted a major study of the blood concentrations of dioxins/furans in workers and some community members and compared the results against NHANES data. The data may be submitted to a journal for publication.

281. **Study of exposure of steel workers to asbestos:** In about 2006, we completed a dose-reconstruction analysis of exposure to asbestos in various jobs in steel mills for the period 1950-1980.

282. **Community Mercury Exposure Near a Gold Mine in Peru:** Around 2006, we were retained to evaluate an incident that occurred in Peru involving neighborhood exposure to elemental mercury which was associated with a gold mine. Cumulative and acute exposures to elemental mercury were assessed for over 40 individuals based on written summaries of their exposure history. Microenvironments included home (near and away from spilt mercury), outside playing with mercury, outside walking near mercury and ambient air exposure. This was one of the first major "toxics" cases against a firm in the US who was performing work in a foreign country. The difficult part of the analysis was later published in a peer reviewed journal.

283. **A proposed approach to characterizing the risks posed by airborne irritants in the work environment:** In 2005 and 2006, conducted a pro bono analysis of current methods for assessing occupational irritants, and proposed a new approach to setting occupational exposure limits. ChemRisk was invited to present our results at an international conference in Cologne, and the paper describing the work was published in 2006.

284. **Conducted evaluation of the hazards posed by DEHP in computer and phone cables:** Due to a challenge under California's Proposition 65, in about 2005, ChemRisk conducted a health risk assessment for a telecommunications firm to assess whether enough DEHP might be released from the handling of the cable to exceed the Prop 65 "safe harbor."

285. **An epidemiology study of contaminated indoor air (California):** In 2005, evaluated a concern by numerous employees in a large office building in San Francisco of a potential increased incidence of breast cancer in the workforce.

286. **Analysis of metals from degraded concrete in household dust (Pleasanton, CA):** In 2001, conducted an analysis of metals in dust that had flaked off the surface of concrete in a home. A risk assessment was conducted on behalf of the plaintiffs (e.g., homeowners).

287. **Analysis of maximum daily load (TMDL) of mercury in Savannah River (Georgia):** In 2000, worked with Savannah River Nuclear Site and regulatory agencies to develop a program to assess the mercury burdens to the river and the need to comply with new TMDL guidelines.

288. **Analysis of meat contamination from methyl bromide (Arizona/California):** In 2000, for the plaintiffs, evaluated the human health risks posed by treating frozen bacon and processed meats with methyl bromide. Expert testimony was provided in a deposition.

289. **A complex pharmacokinetic study of furan elimination from children exposed to excessive dioxin concentrations due to contaminated soil:** Over the years, a number of models have been proposed to predict the past and future blood levels of dioxin/furans in children due to ingestion of breast milk and other foods. Based on data collected from various studies conducted around the globe, a new model was built and validated. The results were published in a journal in 2008.

290. **A pharmacokinetic analysis of dioxin and furan elimination in children:** Between 2003 and 2006, conducted an extensive analysis of published and unpublished data on the biologic half-life of these persistent chemicals in young children and adolescents. In collaboration with researchers involved in the Seveso incident in Italy, a pharmacokinetic model was developed. Our resulting analyses could impact views about the hazard to children from eating dioxin contaminated soil. The analysis appeared in the press in 2008.

291. **A study of the integrity of a mobile home with respect to its ability to keep carbon monoxide from the living quarters, as well as an evaluation of various safeguards:** The investigation was conducted as a result of two deaths due to carbon monoxide. Many simulation studies were conducted under a number of conditions in New Mexico (Nov. 2007). A manuscript describing the findings from this study was published.

292. **State of art research regarding the bioavailability of dioxin/furans in soil:** Building upon 20 years of research on bioavailability, designed and executed a study to understand the various parameters that influence the release of these chemicals. Work was conducted in 2005-2007 and was submitted for publication.

293. **Assessment of the historical exposure of workers who use mineral spirits assumed to contain "trace concentrations of benzene" (between 0.001 and 1%):** In 2007, conducted a simulation study that characterized the near-field and far-field airborne concentration of benzene exposure to workers who use reasonable quantities of mineral spirits. Results were published in 2012. The work was funded by ChemRisk.

294. **Assessment of the possible human exposure to asbestos resulting from handling and opening boxes that contained clutches:** Because of concerns about asbestos in the workplace, conducted a simulation study of workers who repeatedly handled boxes containing clutches by measuring both the acute and chronic airborne concentrations in the breathing zone. The work was conducted in 2007, and the results were published in early 2008.

295. **Evaluation of the possible human health hazard posed by benzene in soft drinks:** In 2006 and 2007, there were a number of reports regarding the detection of benzene in certain fruit flavored soft drinks. As a public service, conducted a pro bono sampling program and risk analysis of a particular manufacturer's soft drinks. The results have been presented in a peer-reviewed journal.

296. **Assessment of the historical exposure to benzene associated with using the rust dissolver known as Liquid Wrench:** In 2005, conducted a simulation study to assess the airborne concentration of benzene associated with using about 10 ml of liquid wrench, assuming it contained between 1-30% benzene. The results were published in a peer- reviewed journal in 2007. In addition, a companion paper describing our urinary monitoring study was also published.

297. **Assessment of ethylene oxide emissions from a hospital sterilizer (Illinois):** In 2005-2006, evaluated historical exposures to EO that may have occurred as a result of sterilizer use. There were some allegations that acute and chronic health effects were observed in former and current workers.

298. **Assessment of N-nitrosodimethylamine (NDMA) and perchlorate in drinking water (California):** During 1998-2006, evaluated the health risk to residents who might have ingested water containing various concentrations of dimethylnitrosamine and perchlorate. The assessment was conducted at a former rocket test site.

299. **Assessment of possible human exposure to asbestos resulting from handling and opening boxes that contained historical brake linings:** Because of concerns about asbestos in the workplace, conducted a simulation study of workers who repeatedly handled boxes containing brake linings by measuring both the acute and chronic airborne concentrations in the breathing zone. The work was conducted in 2006, and the results have been submitted for publication.

300. **Assessment of the possible dioxin contribution to diet from ingesting wild and farm raised catfish:** Questions have been raised about regional differences in the blood concentrations of dioxins in humans. In order to assess whether ingesting catfish might produce a typical fingerprint pattern and a typical contribution to diet, in 2006, farm and wild catfish were captured and assayed. The results will be submitted to a journal for publication. This work was funded by ChemRisk.

301. **Evaluation of all available studies of asbestos air samples from on-board shipping vessels:** More than 100 vessels were studied in an attempt to determine if the presence of asbestos in intact insulation released measurable concentrations of fibers. The work was conducted in 2006 and was published in a peer-reviewed article in 2008.

302. **Evaluation of potential sources of lead and data usability for source characterization in support of cost allocation litigation for railroad sites in Missouri:** Chemical concentration data at these sites were evaluated to determine whether potential sources of lead could be identified, and remediation costs could be allocated to several defendants (including a mining company) who transported lead using the railroad (Summer 2006).

303. **Evaluation of lifetime cumulative asbestos dose by mechanics due to brake wear debris:** Between 2002 and 2006, characterized the historical exposure of mechanics to asbestos and brake wear debris. This particular study calculated the plausible range of lifetime doses for auto mechanics in the United States and Europe for the period 1950-2007 and compared those doses to other occupations. The study was published in a peer- reviewed journal in 2007.

304. **Evaluation of recent data on worker sensitization to beryllium:** In 2005-2006, conducted an evaluation of the past five years of industrial hygiene and biomonitoring data (e.g., BLPT data) at a large manufacturing complex. The results were published in a peer- reviewed manuscript.

305. **Evaluated a pharmaceutical manufacturing facility causing reported allergic skin responses:** In the hope of identifying what was causing an outbreak of apparent allergic contact dermatitis (ACD) and eye irritation incidents in a large fraction of the workers, we were retained to evaluate the manufacturing facility and procedures used to handle a rather common agent used to minimize nasal decongestion (Fall 2005).

306. **Assessment of historical exposure of craftsman in refineries to asbestos (various locations):** In 2004-2005, conducted a thorough review of the literature regarding historical exposure of various tradesmen to asbestos in the petrochemical industry (and some others). The results were presented at two conferences and then published in a peer- reviewed manuscript in 2007.

307. **Assessment of perfluorooctanoic Acid (PFOA) in groundwater (West Virginia):** In 2003, ChemRisk was retained by DuPont to characterize the human health hazards posed by various concentrations of PFOA in groundwater, ambient air, soil, and other media near their Parkersburg manufacturing site. The work was completed in 2005. It appears to have been the first study to document the possible importance of airborne emissions with respect to groundwater.

    The results were so surprising that the EPA funded a team of professors at Harvard and Emory to duplicate our study. 18 months later they issued a report and published paper confirming the validity of our work. Our analysis was published in a peer-reviewed scientific journal, and it was presented at the First International Symposium on Fluorinated Alkyl Organics in the Environment (Toronto, Canada) in August 2005.

308. **Assessment of asbestos in brakes used in very heavy earthmoving equipment (various locations):** In 2004-2005, evaluated published and unpublished literature regarding the characteristics of brake wear debris, as well as the likelihood of exposure to mechanics, involved in repairing heavy equipment**.**

309. **Assessment of benzene exposure by ship workers (various locations):** In 2004- 2005, conducted an exposure assessment of persons who loaded and unloaded fuel oils from ships. The analysis was published in a peer-reviewed journal.

310. **Assessment of PAHs in soils at a former Manufactured Gas Plant (MGP) site (Chicago**): In 2005, ChemRisk was asked to help assess the current hazard of PAHs in soils to citizens living near a remediated MGP site. A risk assessment involving contaminated soil and vapor loss was conducted.

311. **Assessment of the possible human exposure to asbestos resulting from replacing gaskets in chemical piping and fittings:** Due to concerns about asbestos in the workplace, evaluated the published and unpublished literature in order to characterize the range of exposures. The work was conducted in 2005, and the results were published in 2007.

312. **A study of the human and ecological hazards of Tire Wear Particles (TWP):** Between 2005 and 2012, our firm had responsibility for designing and managing the research efforts to understand the domestic and international questions regarding health impacts of TWP. About 2,000,000 tons per year of TWP are released globally each year, and questions have been raised about TWP presence in ambient air and sediment. Funded by the 11 major tire manufacturers, studies of various types were conducted internationally over a period of five years. Nearly $8M in research and as many as 10 peer-reviewed papers were published as a result of this classic project in "product sustainability".

313. **Evaluation of asbestos release from gaskets (Nationwide):** During 2003-2005, conducted a thorough review of the published literature regarding the possible release of asbestos during the handling and removal of gaskets from flanges, pumps, and autos. This work was published in a peer-reviewed paper.

314. **Evaluation of diesel particulate emissions from mining operations (California):** In an effort to understand the potential human health impact associated with diesel particulate emissions from sand and gravel mining and processing operations, our staff performed air dispersion modeling to predict diesel particulate emissions during these operations. This work was performed in order to fill permit requirements under the California Environmental Quality Act (Spring 2004).

315. **Asbestos, dioxin, and other materials in dust from the World Trade Center (New York):** In 2004-2005, conducted an analysis of how to determine whether WTC dust had entered a building and assessed the possible associated health risks (at the former 90 Church Street site). The fingerprinting analysis was published in a peer-reviewed journal.

316. **Assessment of asbestos in phenolic thermosetting resins (National):** In 2004, ChemRisk was retained to assist a manufacturer in characterizing the exposure and risks posed by asbestos in thermosetting resins used to make Bakelite-like products. A simulation study was conducted, and the results were published in a peer-reviewed journal.

317. **Assessment of PAHs in creosote treated wood (Louisiana):** In 2004, conducted an exposure and risk assessment of persons who handled treated wood, as well as an assessment of the dioxins released from this wood when burned in fireplaces.

318. **Evaluated the historical hazard of the use of asbestos in truck brake linings:** During the 1920-1985 era, most truck brakes contained 20-50% chrysotile asbestos. Because some of the techniques used to conduct maintenance work on these trucks is different than autos, beginning in 2004, we evaluated the complete repair operation using historical data and information from simulation studies to characterize the plausible health risks. This work was conducted in several large truck repair facilities.

319. **Assessment of efficacy of an air cleaning device for the home (National):** In 2000-2003, conducted a series of field studies and some laboratory studies to assess the capability of an air cleaning device to remove and/or destroy various microbes, fungi, and other airborne chemicals.

320. **Assessment of risks posed by asbestos in automotive friction materials (National):** During 2001-2003, conducted a state-of-the-art evaluation of the toxicology, epidemiology, regulatory, and exposure components of the use of chrysotile asbestos in friction materials (brakes and clutches) in automobiles and trucks. This work has been published in a peer-reviewed journal.

321. **MTBE in California groundwater (California):** In 1999-2002, involved in work to evaluate the magnitude of the impact of MTBE on California groundwater. During the course of the evaluation, a number of papers were presented at conferences and published in literature. About 7 different papers were published in peer- reviewed journals.

322. **Study of brain cancers in a large office building (California):** In 2000-2002, established a strategy for conducting a dose reconstruction and epidemiology study of the inhabitants of a formerly occupied office building in Southern California. The assessment was recently completed.

323. **Assessment of asbestos in glues, adhesives, coatings, and mastics (National):** In 2002, ChemRisk was retained to assist Amchem, Inc. in characterizing the exposure and risks posed by asbestos in glues, coatings, mastics, and sealants. A simulation study was conducted, and the results were published in a peer- reviewed journal.

324. **Assessment of health hazards posed by CCA-treated wood (National):** During 2001-2002, directed a team that evaluated the possible human health hazards posed by contact with the arsenic, copper, and chromium present on the surface of treated wood. The concern of regulatory agencies and society was the risk to children who played on decks and treated wood in playgrounds. Work was presented to USEPA Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) sampling and analysis plan (SAP).

325. **Human and ecological assessment of multi-chemical contaminated site (California):** Conducted an assessment of nearly 100 chemicals present in soil under a former drum- storage area. The assessment was the first multi-component soil assessment (pre- and post- remediation) accepted by the South Coast Board and the California USEPA. Cleanup of the site was conducted by a steam-heated auger under an USEPA research grant program (Summer 2002).

326. **Assessment of thiocarbamate in wines (California):** In 2002, ChemRisk was retained by the Wine Institute to evaluate the results of the recently completed NTP bioassay on thiocarbamate. Conducted a screening cancer risk assessment to assess the possible impact on the wine industry.

327. **Dose reconstruction of benzene in the Pliofilm (Rubber) workers (National):** In 2001-2002, conducted an update of Dr. Paustenbach's prior (1992) exposure analysis of this cohort, using Monte Carlo techniques and some recently discovered data. The analysis used virtually all of the available methodologies for characterizing the plausible range of exposures to this cohort. This analysis will be published in Toxicology and Environmental Health.

328. **Dose reconstruction of occupational exposure to benzene (Texas):** From 1999- 2002, began to conduct a retrospective exposure assessment of office workers at an acetic acid plant to about 400 chemicals Exposure began in the 1960s and ended in the 1980s; data may be used in an epidemiology study. Some of the analysis was used in a personal injury litigation case. The results were published in a peer-reviewed journal.

329. **Epidemiology study of chromium (VI) (Ohio):** During 1998-2002, served as a science advisor on work involving the assembly of all available personnel, medical, and industrial hygiene data for the Painesville, Ohio, workers (so-called Diamond Shamrock Cohort). The results of the research were used by OSHA during their reevaluation of the dose- response curve and cancer potency factors for airborne hexavalent chromium. About four papers describing this work were published in peer-reviewed journals.

330. **Evaluation of aerosol generating medical device:** In 2001-2002, conducted a risk assessment that addressed the possibility that small metal particles (less than 1 µm) might be released from an aerosolizer during use.

331. **Evaluation of the eye irritation potential of a mining dust [trona] (National).** In 2000-2002, worked with a number of physicians to develop a sensitive methodology for evaluating eye irritation associated with various levels of exposure to airborne mining dusts. The methodology involved photographing the surface of the eye and using a computer to objectively scale the irritation level.

332. **Assessment of lead and cadmium in talc and other baby products (California):** In 2000-2002, conducted a risk assessment that evaluated the likelihood that trace amounts of lead and cadmium in talc and zinc oxide ointment in baby products might penetrate the skin and produce an absorbed dose in excess of the California Proposition 65 "safe harbor" concentration.

333. **Assessment of lead in consumer products (California):** In 2000-2001, conducted scientific evaluations and supplied expert reports for claims involving ingestion and dermal absorption of lead (at least 6 cases). As a result of claims under California's Proposition 65, a series of lawsuits were filed from 1996-2001 involving human exposure to lead. The suits generally focused on ingestion and dermal absorption. ChemRisk designed a number of studies to understand some of the various consumer products and the likelihood that enough lead could be absorbed to exceed the "safe harbor" standards. The media of interest included telephone cables, vacuum cleaner hoses, computer battery charger cables, and related materials.

334. **Assessed the loss of silicon from drug injection devices due to historical use of talc:** In 2001, ChemRisk examined silica exposures in drug injection devices. For a number of years, up to the present, some manufacturers of automatic drug injection devices used talc as a lubricant or coating on the inside of the device. Concerns were raised about the possible hazards posed by the silica in the talc. The amount of talc released by the device during various injections was measured and the health risks were evaluated.

335. **Assessment of risks posed by bioterrorism (Virginia):** As a result of post- September 11, 2001, concerns, retained by a major credit card firm to evaluate the possible threat to their enterprise from chemically or biologically contaminated mail. The evaluation identified the primary areas of concern, and suggestions were offered as to how to improve engineering controls and implement a more comprehensive protection program.

336. **Retrospective risk assessment at Oak Ridge National Laboratory (Tennessee):** In 1995-2001, served as a science advisor on this largest-ever risk assessment, which involved reconstructing the possible historical offsite exposures to the public as a result of 50 years of operations at the Oak Ridge Reservation. Project is described in Chapter 14 (pp. 735- 870) of Human and Ecological Risk Assessment, (John Wiley and Sons), Dennis Paustenbach (ed.).

337. **Conducted exposure assessment of phthalates released from the tubing of certain fluid delivery pumps:** In 2001, a medical device firm had developed a "finger pump" which could introduce very small or large quantities of blood, or a drug used for cancer treatment into the human bloodstream. However, the continued flexing of the plastic tubing was alleged to increase the leach rate of DEHP and other phthalates from the tubing. ChemRisk conducted a study using human blood and various pharmaceutical agents and measured the percentage loss of phthalates from the tubing over time and then assessed the possible health hazard.

338. **Assessment of chromium (VI) in groundwater (California):** During 2000–2001, interacted with regulatory agencies to help characterize risk from chromium. A number of studies showed that chromium (III) and chromium (VI) were in groundwater throughout the state as a result of naturally occurring and industrial sources. The concentrations of chromium (VI) were very low, but a public health guidance (PHG) value had been proposed.

339. **Assessment of dioxin emissions from oil refineries (California):** From 1995- 2001, evaluated data regarding the contribution of refinery dioxin emissions in air and water to the environment. Provided testimony to several government agencies on this issue.

340. **Assessment of dioxin in soils near a former hazardous waste incinerator (Midland, MI):** In 2001, retained by Dow Chemical Company to characterize the possible human health hazards posed by the historical deposition of dioxins and furans into soils downwind of their incinerator. The soil concentrations were in the range of 50-400 ppt toxic equivalents (TEQ).

341. **Assessment of hazards posed by coal tars in shampoo (California):** In 2000- 2001, retained by a pharmaceutical company to evaluate claims that the content of polycyclic aromatic hydrocarbons (PAHs) in its shampoo products exceeded the amount allowable under California's Proposition 65. An exposure assessment was conducted, and a deposition was taken.

342. **Assessment of hazards to children from arsenic in playground soils (National):** In 2001, retained by two national trade associations to evaluate the possible hazards posed by the leaching of arsenic into soils from timber treated with chromated copper arsenic (CCA). This analysis was the first major health risk assessment related to the national initiative to specifically focus on the possible hazards of certain contaminants and media to children. At least one paper describing our work was published.

343. **Assessment of health risks of water contaminant (New Jersey):** In 1996, designed a number of toxicology studies and conducted a health risk assessment to evaluate risk of adverse health effects from ingestion of low concentrations of a "trimer" that had leaked from soil into groundwater at the so-called Toms River site. The work was completed in 2001.

344. **Established OEL for glycol ethers for Union Carbide Corporation (National):** In 1996-2000, participated on a blue-ribbon panel. Because no OEL had been established to prevent the developmental or reproductive hazard to workers, this firm asked that a panel be formed to recommend a guideline. This panel was convened in 1996 and recommended that research be conducted to support a physiologically based pharmacokinetic model. Four years later, after the research work was completed, a new panel was established, and the data was used to establish a new OEL (work was published in 2001).

345. **Assessment of community health hazards and diesel exhaust from truck terminal (Phoenix, AZ):** In 2000, conducted a semi-quantitative assessment as a result of recent USEPA positions that diesel exhaust from trucks may pose a human health hazard. A petition was filed to prevent construction of a truck terminal that was to hold 500 vehicles in response to USEPA's position. Attended a public hearing in September 2000.

346. **Mercury in gas line pressure regulators (Michigan and Illinois):** In the summer of 2000, developed a field program to measure airborne mercury in the 0.01 to 5.0 µg/m3 range and to evaluate the possible public health significance (assuming exposure had occurred for many years). It had been reported that, in homes built between 1935-1975, gas pressure regulators often contained elemental mercury (1-3 teaspoons each). Apparently, during installation and removal (which occurred to a large extent over the past 25 years) some small spills occurred. Managed the sampling of over 1000 homes and analyzed the data.

347. **Risk assessment of fish contaminated with PCB/1,1,1-trichloro-2,2-bis (p-chlorophenyl)-ethane (DDT) from the Palos Verdes Shelf (California):** From 1995-2000, developed and implemented a unique Monte Carlo-based assessment of the health risks from ingested fish containing PCB and DDT from contaminated sediments in Los Angeles harbor. This was, at the time, the costliest cleanup litigated by the Department of Justice (~ $1B). The assessment was peer- reviewed by an outside panel of professors, and later published in a peer-reviewed journal and book chapter. The case was resolved in federal court in late 2000 (commonly known as "the Montrose case at Palos Verdes").

348. **Assessment of benzene in diesel exhaust from locomotives (South Dakota):** During 1999-2000, conducted a series of dose reconstruction studies in a turn-of- the-century roundhouse involving diesel exhaust, nitrous oxides, carbon monoxide, and benzene. These studies were performed because of the application of a different legal standard regarding causation criteria with respect to occupational exposure of railroad workers. A question was raised regarding the possibility that benzene present in diesel exhausts could contribute to an increased incidence of leukemia and other diseases in these workers.

349. **Assessment of dioxin and furans in soils and sediments (Midland, Michigan):** In 2001-2005, conducted a comprehensive risk assessment of dioxins surrounding a former incinerator at the Dow facility in Midland. In addition, provided advice on how to assess the human and ecological hazards associated with the presence of these chemicals in flood plains downstream of the facility.

350. **Evaluation of medical device safety (California):** In 2000, evaluated the hazard from micron-sized metal airborne particles unintentionally released by a medical device that delivered drugs via inhalation.

351. **Assessment of occupational health hazards posed by airborne beryllium (Ohio and Arizona):** Beginning in early 1999, designed a comprehensive program for characterizing the hazards posed by airborne and dermal contact with beryllium in the workplace. Ultimately, was involved in numerous research efforts to understand the a) usefulness of beryllium blood lymphocyte proliferation test (BLPT) monitoring; b) importance of particle size and chemical form of the beryllium in determining the hazard; c) relationship between disease and the other routes of exposure to beryllium; and d) importance of establishing different dose- metrics. Worked with the client and ACGIH to put together an international symposium on beryllium, held September 1999 in Washington, DC.

352. **NAFTA litigation involving MTBE (Toronto, Canada):** In 1999, retained by Methanex Corp. to address the environmental and human health hazards associated with the use of MTBE by the United States as an oxygenate. Their claim was that California banned MTBE in an arbitrary manner. The case was settled in the World Court in 2004.

353. **Assessment of health hazards of formaldehyde in mobile homes (Missouri):** In 1999, evaluated historical health hazards, including asthma, cancer, and immune and respiratory system diseases associated with presence of various concentrations of formaldehyde in mobile homes. Some of this work was used in personal injury litigation.

354. **Assessment of the benzene hazard posed by mineral spirits (Arizona):** In 1999, conducted a series of dose reconstruction experiments to evaluate benzene hazards in railroad workers. It was alleged that, from 1960-1990, mineral spirits might have contained enough benzene to be measurable in air and, therefore, increase the probability of developing acute myelogenous leukemia in railroad workers. A manuscript describing the work was published.

355. **Assessment of the hazards posed by occupational exposure to sensory irritants (National):** In 1999, served as chairman of the meeting that presented various papers regarding considerations for setting OELs for sensory irritants. The symposium was convened by the American Chemistry Council (ACC) as a result of international concern about the relatively small margin of safety inherent in many OELs for irritants. These papers were published in a special issue of the AIHA journal.

356. **Assessment of the occupational hazards of various types of beryllium in different settings (California):** In 1999, conducted dose reconstruction to evaluate the average and peak lifetime exposure to various forms of beryllium for a worker in the aerospace industry. Assessed the quality of employer efforts to minimize exposure to this agent over the last 50 years.

357. **Evaluation of 2-ethylcyanoacrylate (ECA) as a respiratory sensitizer (National and Europe):** In 1998-1999, conducted a literature evaluation of all published studies on ECA to evaluate increased concern about chemicals in the workplace and in consumer products that might induce or elicit an asthmatic response. Conducted an exposure assessment on location in Puerto Rico. Also conducted an epidemiology study of this worker cohort and published the results in a journal. A qualitative assessment of a facility in Dublin, Ireland was also conducted; this information was then presented to and evaluated by the ACGIH Threshold Limit Value (TLV) Committee, who revised the TLV, in part, based on this study. Two manuscripts were published describing the work.

358. **Risk assessment of emissions from a waste incinerator (Louisiana):** In 1998- 1999, conducted a risk assessment of airborne emissions released from the largest existing waste incinerator. Determined that improved emission controls were necessary to limit potential dioxin exposures to farmers living near the incinerator and to prevent intake by cattle. The design of the air pollution control devices was modified, and a permit was then issued so that it could operate. For other reasons, the new facility never went into production.

359. **Assessment of risks posed by chromium (III) in Hackensack River (New Jersey):** From about 1994-1999, evaluated the risks to aquatic species and humans posed by the presence of chromium in the sediments of the Hackensack River.

360. **Retrospective assessment of trichloroethylene (TCE) in drinking water (Phoenix, AZ):** In 1997-1998, conducted research and a health risk assessment to assess exposure to TCE vapors from contaminated tap water. This work was later published in the journal Risk Analysis.

361. **State-of-the-art assessment involving disposal of wastes in landfills (Ohio and Michigan):** In 1998, conducted an evaluation of the appropriateness of behavior by major rubber and steel casting firms who chose to place industrial wastes in municipal landfills in the 1950s and 1960s. Testimony regarding the evaluation was presented in state court in Akron, Ohio.

362. **Assessment of di(2-ethylhexyl) phthalate (DEHP) in packaged cheeses under Proposition 65 (Washington, DC):** In 1997-1998, evaluated the validity of claims that DEHP was present at unacceptable levels (under Proposition 65) in processed cheeses using a stratified, random focused sampling program. A risk assessment evaluating 60 cheeses was conducted.

363. **Assessment of dioxin emissions from a combustor (Columbus, OH):** In 1997- 1998, was retained by plaintiffs to evaluate the legitimacy of a claim that airborne emissions of dioxins from a combustor may have raised dioxin levels in blood in citizens. The data indicated that the uptake was negligible, which was not surprising, since the amount of farmland near the combustor was negligible.

364. **Assessment of hazards posed by combustion products from commercial fires (California):** In 1997-1998, evaluated the possible public health hazards associated with the emission and transport of combustion products produced during a fire involving a chemical plant. Assessed hazards due to carbon dioxide, dioxins, PAHs, soot, and fine particles.

365. **Assessment of the emissions from a combustor/vitrifier (Louisiana):** In 1998, conducted a state-of-the-art screening risk assessment of all aerial emissions from what was to be the world's largest combustor. The primary focus of this work was to assess the long-term impact on aquatic species from runoff and entry into streams/bayous.

366. **Assessment of toxics in sediment, groundwater, and soil (Louisiana):** From 1992-1998, oversaw a major human and ecological toxicology assessment involving the evaluation of potential hazards resulting from emissions from one of America's largest chemical manufacturing plants adjoining a wetlands and bayou. The study occurred over six years and produced nearly 3,000 pages of text. The study's chemical of primary interest was Hexachlorobenzene.

367. **Evaluation of the safety of silicone breast implants (SBI) (Columbus, OH):** In 1997-1998, conducted an evaluation of the toxicology information, medical literature, epidemiology, case reports, biomaterials information, and related materials to assess the safety of humans of using SBI. SBI were used by plastic surgeons throughout the world for physical enhancement from about 1964 to 1984.

368. **Environmental assessment of proposed golf course (Ohio):** In 1996-1997, conducted a comprehensive risk assessment to characterize issues associated with a proposed golf course. Pesticide runoff and water consumption have concerned communities across America since about 1985; as a result, most new golf courses cannot be built without a comprehensive assessment. This assessment was one of the first times risk assessment was used to characterize the issues.

369. **Assessment of PCBs in Hudson River (New York):** In 1987-1997, designed studies of the concentrations and location of PCBs in sediments. A creel study was conducted of local and Native American fisherman in an effort to characterize the health risks resulting from PCBs. A risk assessment was later conducted.

370. **Assessment of property near a former landfill subsequently used as a residential site (Northern California):** From 1990-1997, served as principal-in- charge of health risk assessment for a residential development built in the vicinity of an excavated former landfill. A comprehensive risk assessment was conducted, and the analysis was presented in state court.

371. **Ecotoxicology assessment of contaminated sediments in the Newark Bay watershed (Passaic River, NJ):** In 1991-1997, directed a project involving a complete assessment of the human, aquatic, and avian hazards posed by dioxin, PCBs, and other long-lived chemicals in sediments.

372. **Assessment of dioxin in soil (California):** In 1995-1996, served as project manager of risk assessment of polychlorinated dibenzo-p-dioxin/furans (PCDD/Fs) from a combustor source in soil, sediment, and air. Approach evaluated tetra- through hepta-CDD/F separately from octa-CDD/F to allow use of congener- specific disposition and toxicity data.

373. **Assessment of risks of chromium (VI) in drinking water (California):** In 1995- 1996, developed a multi-pathway exposure and risk assessment to estimate the retrospective exposure history of approximately ten test plaintiffs with alleged exposure to hexavalent chromium in groundwater. Designed and conducted a series of bench scale testing and human pharmacokinetic studies to examine the fate of hexavalent chromium in humans after tap water ingestion. Nearly twenty peer-reviewed papers and/or abstracts were developed and presented at various scientific meetings.

374. **Dose reconstruction of chromium workers at the Painesville site (Ohio):** Beginning in 1996, conducted an evaluation of historical occupational exposure information for one of the largest chromium processing sites in the United States, which operated from 1940- 1970. Project involved reviewing documents including air sampling data, medical data, and manufacturing information. The analysis resulted in the publication of four different manuscripts that are, in a large measure, the basis for OSHA regulations regarding airborne chromium (VI).

375. **Sampling of chromium (VI) in air:** For two summers, served as the principal-in-charge of a major research program devoted to collecting more than 300 twelve- hour samples of airborne dust near sites containing COPR in soils. At least four papers were published about this work (mid 1990s).

376. **Risk assessment of CERCLA cost recovery (California).** In 1994-1995, conducted a comprehensive critique of remedial investigation, risk assessment, and feasibility study documents prepared for a heavily investigated CERCLA site in California. Examined the strengths and weaknesses of the reported findings. Data showed that client was a de minimus contributor.

377. **Exposure assessment of herbicide risks (California):** During 1994-1995, developed plausible estimates of dioxin uptake resulting from specific work activities. Designed and conducted experiments to quantitate exposure to aerosols for a roadside weed abatement operator resulting from blowback. Developed dermal uptake models that were integrated with PBPK models to estimate total dioxin uptake and blood concentrations of dioxin.

378. **Risk assessment and research of Pentachlorophenol/dioxin (Georgia):** In mid- 1990s, managed a project that delineated the state of scientific knowledge from 1950 to present regarding the known environmental fate and transport characteristics of pentachlorophenol products. Provided a summary and analysis of site-characterization data, and a screening level risk assessment for two facilities seeking cost recovery.

379. **Research of metals bioavailability (Louisiana):** In the mid-1990s, conducted research of several issues pertaining to the production of a vitrified aggregate material from soils originally contaminated with lead. Provided research materials relevant to the critical issues of metals accumulation and the transport and fate and risk assessment considerations to identify safe uses of the aggregate. The aggregate has been used in both concrete and asphalt. Served as an expert witness in federal court to address the regulatory compliance issues with respect to toxicology and risk assessment.

380. **Assessment of aquatic and human health hazards posed by sediment containing very high levels of TCDD (Sydney, Australia):** In 1994-1995, conducted a preliminary risk assessment of the hazards posed by harbor sediments containing 2,3,7,8-TCDD at concentrations as great as 20 mg/kg. This site is near Homebush Bay in Sydney Harbor, and portions of the area were eventually remediated and used as part of the Olympic Village for the 2000 Olympics.

381. **Assessment of benzene in indoor air as a result of contaminated soil (California):** In mid-1990s, for the plaintiffs (homeowners), managed risk assessment that evaluated cancer risk due to potential indoor exposure to benzene emitted from contaminated soil underlying residences.

382. **Assessment of dioxin emissions from oil refineries (Northern California):** In 1995, assembled available air and water emissions from refineries and assessed the hazard to humans and wildlife. Conducted the first comprehensive study of dioxin runoff to the San Francisco Bay resulting from airborne deposition from all possible sources. The work was presented at the International Dioxin Conference and was published in a peer-reviewed journal.

383. **Assessment of dioxin emissions from world's largest incinerator (Michigan):** In the mid- 1990s, served as an expert in determining the possible health risks associated with the aerial emissions from the City of Detroit's municipal waste incinerators, at the request of former mayor Coleman Young. The issue was of considerable interest to Canada, which believed that the majority of the emissions were being deposited onto Canadian soils.

384. **Assessment of dioxin-contaminated sediment in Passaic River (New Jersey):** From 1991-1995, served as the principal-in-charge and key interface to USEPA on a project involving dioxin-contaminated sediment in New Jersey. Developed scoping documents and work plans for sediment sampling on at least six different occasions. Helped write the work plan for the remedial investigation, which required four years to implement.

385. **Assessment of lead from a rotary kiln (Louisiana):** In 1991, prepared human and ecological risk assessments for a former hazardous waste treatment facility. The assessment included the evaluation of lead in site soils using the USEPA integrated exposure uptake/biokinetic (IEUBK) model. This assessment was a landmark risk assessment because it addressed the thorny issue of recycling so-called "low risk" combustion by- products. The case was argued in federal court in Louisiana against the U.S. Department of Justice in about 1995.

386. **Ecotoxicology assessment of chromium in sediment in the Hackensack River (New Jersey):** Between 1994 and 1995, performed technical oversight for an ecological assessment involving site-collected sediments, crabs, and other wildlife.

387. **Health assessment of contaminated groundwater at a major Superfund site (California):** In 1994-1995, conducted an assessment of three pesticides in soil to determine whether one of the suppliers of these chemicals could have produced soil contamination sufficient to warrant cleanup.

388. **Risk assessment of refinery emissions (California):** In the mid-1990s, conducted an analysis of chemical emissions and the associated risks from a refinery in California. Past chemical emissions were found in refinery records, and a retrospective assessment of exposures of 4,000 nearby residents was conducted.

389. **Assessment of cadmium-contaminated sediment on the Hudson River (Marathon Battery, New York):** In the 1990s, conducted a preliminary ecological assessment of the potential hazards to aquatic and avian species due to cadmium in both surface and buried sediments. Ultimately, the sediments at this site were remediated under Superfund.

390. **Assessment of chloroform from a paper mill (Georgia):** In the mid-1990s, served as principal-in-charge of assessment of chronic chloroform inhalation exposure. Health risk was characterized using alternative cancer slope factor and presented a strong defense for the use of a physiologically based pharmacokinetics (PBPK) model approach that considered species-specific metabolic pathways.

391. **Assessment of ecotoxicology hazards associated with sediments contaminated with organochlorines (California):** In 1994, conducted risk assessment of human health and aquatic hazards associated with concentrations of DDT, chlordane, heptachlor, and dieldrin in surface sediments.

392. **Chemical fingerprinting of sediments (New Jersey):** From 1990-1994, used polytropic vector analysis and multivariate analysis (fingerprinting) to understand dioxin contamination of river sediments. Six papers on our work were published in the peer-reviewed literature.

393. **Development of method for detecting airborne chromium (VI) in particulates:** In the early 1990s, the primary concern associated with the presence of chromium (VI) in soil was the potential inhalation hazard of chromium (VI), a likely human carcinogen when present in acid mist. Since no ambient method for detecting ppb concentrations was available at the time, ChemRisk developed a new method based on the work of the California Air Resources Board and NIOSH. It was later accepted by ASTM as the standard method for sampling ambient concentrations of chromium (VI) in airborne particulates.

394. **Dose reconstruction at Rocky Flats (Colorado):** From 1991-1994, served as a science advisor for a $6M toxicologic review and dose reconstruction project for the Colorado Department of Health. This retrospective assessment quantitatively evaluated the possible offsite health impacts of radionuclide and chemical emissions from the Rocky Flats Nuclear Weapons Facility since operations began in 1953. Several peer-reviewed papers were published describing the work.

395. **Evaluation of occupational standards for formaldehyde (Washington, DC):** In 1993-1994, on behalf of the Formaldehyde Institute, convened a panel of experts to identify the airborne concentration of formaldehyde that does not produce sensory irritation. A 50- page peer-reviewed paper was eventually published.

396. **Evaluation of reasonableness of USEPA model for predicting soil concentrations in airborne particulates (New Jersey):** In 1992-1994, conducted an air sampling program near a chromium-contaminated site with the intent of validating Cowherd's soil suspension model. The results were published in a peer- reviewed journal.

397. **Assessment of 1,4-dioxane in wine under Proposition 65 (California):** In 1992, evaluated the accuracy of claims that 1,4-dioxane was present at unacceptable levels in wine. Developed a PBPK model for the chemical and published results in a journal in 1994.

398. **Assessment of cadmium in sediments (New York):** In 1994, evaluated the possible ecological and human health hazards posed by cadmium contamination of sediments in a bay. Ultimately, the so-called Marathon Battery site was among the first to undergo sediment remediation.

399. **Risk assessment of chloroform, hydrogen sulfide (H2S), dioxin (Washington):** In 1993, conducted risk assessment of possible airborne chemical emissions during the dredging and remediation of a wastewater treatment facility. More than thirty expert reports and datasets provided by the plaintiffs' experts were analyzed. The chemicals of interest were chlorinated dioxins and furans, chloroform, methylene chloride, and other chlorinated compounds, H2S and mercaptans, mustard gas, oxides of sulfur and nitrogen, nitrosamines, and sulfuric acid mist or fog. Testified in state court in 1994.

400. **Retrospective exposure assessment of benzene (Pliofilm) workers:** In 1991- 1993, under a contract with American Petroleum Institute (API), reviewed industrial hygiene and process engineering records for three pliofilm manufacturing facilities in Ohio and reconstructed the likely uptake of benzene by almost 400 different people who worked during the period 1939-1976. These exposure estimates were then combined with mortality data, and the cancer potency factor for benzene based on the human epidemiology experience was calculated. Our work was considered during the 1997 reassessment of the USEPA/OSHA cancer potency factor for benzene. The current TLV [0.3 ppm (1994)] was based on our analysis. Work was published in a peer-reviewed journal, which has been frequently used in international regulations involving benzene.

401. **Assessment of aquatic hazard from contaminated sediments (California):** In 1991-1993, evaluated the possible impact of contaminated sediments on a reach of the Port of Oakland (Heckathorn site). Following a field study, ChemRisk showed that the most significant adverse effect on aquatic life was from high levels of suspended solids in waters due to boats moving in and out of the shipping channel.

402. **Re-Opening Records of Decision (ROD) on Dioxin Contaminated Soils:** In the winter of 2012, we were retained by one of our historical clients to evaluate claims by EPA that a ROD should be revisited due to the change in dioxin in soil clean-up guidelines. We conducted the original assessment in 1990. The assessment was still ongoing in late 2014.

403. **Assessment of dioxin emissions from an industrial oil-fired boiler (Portland, ME):** In the early 1990s, served as a principal reviewer for an assessment that predicted the dioxin concentration in local crops and grazing stock near an oil-fired boiler.

404. **Assessment of hazards posed by contaminated soil at a former battery recycling facility (New York and Pennsylvania):** In 1992-1993, conducted an ecological and human health assessment of lead-contaminated soil at two former battery recycling centers. This site ultimately underwent partial (selective) remediation and was considered a model for remediation because of how the risk assessment directed the cleanup approach.

405. **Assessment of mercury in fish (Puerto Rico):** In 1993, a quantitative health risk assessment of inorganic and methyl mercury exposures was developed in a fishing village in Puerto Rico (Superfund site, USEPA Region II). Analyses included pharmacokinetic modeling of chemical uptake into beef and dairy cows and edible marine organisms, and subsequent evaluation of maternal body burden and breast-milk levels in exposed human populations. Monte Carlo analysis of fish tissue data was used to evaluate human exposure.

406. **Comprehensive risk assessment of chromium in soil:** Over a period of two years (1991- 1993), ChemRisk conducted a number of risk assessments of the cancer and dermatitis hazards posed by the presence of COPR in soil. Our analyses were published in three different peer-reviewed journals.

407. **Proposed reference concentration for chromium (Consortium Clients):** From 1991-1993, prepared and submitted comments to USEPA's proposed "reference concentration" for airborne chromium. Was invited to serve as advisor to an USEPA work group to evaluate better methods for setting these criteria. The work was published in a peer-reviewed journal.

408. **Risk assessment of PCBs in recycled paper products (California):** In 1992-1993, was asked to determine if the possible human health risks due to using napkins and paper towels made from PCB-containing recycled paper exceeded limits established under California's Proposition 65. Risks were determined to be well below the Proposition 65 risk criteria.

409. **Human health and ecological risk assessment of a southern California port (California):** In the early 1990s, served as lead scientist and project manager for the risk assessment of a remedial action plan for metal recycling facility located in a port district area. Assessed potential exposures to on-site workers, nearby residents, and marine life related to heavy metals, PAHs, and PCBs. Developed a risk-based sampling approach to collect data to support using site-specific leaching profile and conducted the risk assessment.

410. **Assessment of wood treatment chemicals in soils and sediments (North Carolina**): In the early 1990s, examined contaminant migration and potential human health risks to community members from a former wood treatment facility contaminated with creosote, PAHs, pentachlorophenol, and dioxin compounds. Evaluated whether alleged chemical releases could have adversely affected residents.

411. **Assessment of cyclodiene pesticides in soil (Illinois):** From 1990-1992, prepared a multipath way exposure and risk assessment for a state Superfund site in Illinois. It was alleged that aerial emissions from a pesticide-formulation facility increased the concentration of dieldrin, chlordane, aldrin, and heptachlor in the city soils. The city was scheduled to be partially evacuated and was to undergo extensive remediation. USEPA ultimately issued a no-action alternative for the site, and no remediation was required.

412. **Assessment of dioxin-contaminated site (Arkansas):** In 1991 and 1992, identified acceptable cleanup levels for soil at an industrial site known as the Vertac site, which had housed a former manufacturer of 2,4,5- Trichlorophenoxyacetic acid (Silvex).

413. **Assessment of mercury in soil (Puerto Rico):** In 1992, evaluated the acceptability of post- remedial levels of mercury in soil at a site in Puerto Rico.

414. **Assessment of New Jersey Department of Environmental Protection's (NJDEP) soil clean-up levels (New Jersey):** In 1991-1992, prepared an extensive critique of NJDEP's proposed 1991 Regulation for Soil Cleanup Standards. The critique included detailed comments regarding appropriate methods for assessing contaminant uptake and developing health-protective levels for chemicals and metals in soils, groundwater, and interior surfaces of buildings. Ultimately, the proposed standard adopted most of our recommendations.

415. **Evaluation of a residential community built on a landfill (Southern California):** In the early 1990s, evaluated the possible future health risks associated with a 40- acre municipal landfill, above which a large residential community was to be located.

416. **Critique of proposed regulation of airborne hydrogen sulfide (Washington):** In the early 1990s, reviewed a hazard communication document released by a state health department regarding hydrogen sulfide toxicity. Supervised research and documentation to support comments to the state regarding the hazard communication revision.

417. **Assessment of questions about an allergic contact dermatitis hazard:** This work involved developing an original research program to identify the minimum- elicitation threshold for chromium (VI) in sensitized people. This $1-million program involved 54 volunteers who were patch-tested in order to determine the threshold of elicitation. The work was published in a peer-reviewed journal in 1995. Work was conducted in the early- 1990s.

418. **Assessment of chromium (VI) leaching from soil as a result of contact with sweat:** In the early 1990s, conducted research that evaluated the dermal bioavailability of chromium (VI) and chromium (III) in soil when present in sweat. The work was published in a peer- reviewed journal.

419. **Bioaccessibility research was conducted on dioxin contaminated soils and clean-up levels were derived:** Original lab work was conducted to derive the oral bioaccessibility. A Monte Carlo-based risk assessment was conducted. We participated in community and regulatory meetings to explain the significance of our studies. A similar project may be conducted in New Zealand for Dow Chemical (also involving dioxins in soil).

420. **Assessment of Hudson County (New Jersey):** Over a period of eight years, beginning around 1990, served as the primary toxicologist and risk assessor evaluating the hazards posed by the presence of chromium (VI) and chromium (III) in soils in both residential and industrial areas. During this time, we conducted a significant amount of original research to evaluate the possible health hazards posed by chromite ore processing residue (COPR) in soils. Virtually all of this work was published in a series of peer reviewed papers.

421. **Assessment of incinerator emissions (Martinez, CA):** From 1988-1991, managed and prepared a multi-pathway assessment of the health risks associated with emissions of particulates and vapors from a combustor. The State of California had previously determined that the dioxin risks were significant, and that the client would have to warn the surrounding communities. ChemRisk developed one of the nation's first multi-pathway exposure analyses, accounting for ingestion of mother's milk, vegetables, and crops, as well as for indirect exposure from grazing animals (meat and dairy products).

422. **Risk-based remedial investigation, Rockwell Site (Southern California):** From 1989-1991, used risk assessment principles to design and guide remedial investigation activities at a federal Superfund site involving mixed wastes. USEPA approval of the sampling designs, which greatly minimized the number of environmental samples collected (residential soil, groundwater, garden vegetables, and ambient air), saved a substantial amount over the originally estimated sampling and analytical costs, yet provided more useful information than the previous approach.

423. **Avian risk assessment (Illinois):** From 1989-1990, conducted an avian assessment of the possible hazard from cyclodienes in surface soil at a state Superfund site. Assessment involved collecting and analyzing live and dead birds potentially exposed to high levels of pesticides. Pathology and tissue analyses were conducted.

424. **Assessment of exposure to airborne selexol (Chicago, IL):** In 1990, evaluated the potential health hazards to the community following an episodic event where a chemical used to scrub gaseous vinyl chloride and benzene emissions from landfill gas was accidentally released to the atmosphere.

425. **Environmental Sources of Blood Lead and Trends in Children's Blood Lead Levels in Colorado:** In the late 1980s, we developed a position paper on environmental sources of blood lead and trends in children's blood lead levels which related to proposed remedial actions at various Superfund sites. Additionally, a statistical analysis of paired child blood lead and environmental lead data was developed to identify relationships that could be included in predictive models, such as the USEPA's IEUBK Model.

426. **Assessment of Love Canal (New York):** In 1989, evaluated more than ten years of work by numerous groups, and served as an expert witness in the personal injury litigation involving exposure to dioxin and volatile organic compounds in Love Canal resident homes.

427. **Proposed ambient water quality criteria for 2,3,7,8-TCDD (Texas, Louisiana, and Georgia):** During the late 1980s, assisted in preparing the assessment that served as a scientific basis for setting dioxin emission standards for pulp and paper mills on waterways in the three above-mentioned states. These limits were later accepted by regulatory agencies and courts in ten other states.

428. **Assessment of TCDD from incinerator (Times Beach, Missouri):** In 1986-1988, evaluated hazards from the aerial emissions associated with combusting dioxin-contaminated soils. Work was conducted for USEPA, which had hoped to install a temporary incinerator at Times Beach. In 1990, published a peer-reviewed paper describing the analyses.

429. **Risk assessment of radionuclides in phosphate mine slag used to construct cement blocks (Tennessee):** In 1979-1982, conducted original research at Purdue University to determine radon emanation rate from brick, concrete block, and insulation derived from phosphate slag. Performed a risk assessment that considered the resulting airborne radon concentration in typical homes, as well as background levels. Concluded there was negligible risk to persons living in these homes and later published the assessment in a peer-reviewed journal.

430. **Evaluation of the use of a "non-target-specific" pesticide to eradicate coyotes in a farming area (California and Arizona):** In 1988, served as a pro-bono consultant to Friends of the Earth (Washington, DC), the plaintiffs in the case. Submitted an affidavit indicating that the approach proposed by the United States government was not sufficiently target-species-specific enough to warrant the benefit. Because of this analysis and other reasons, the program was not implemented.

431. **Evaluation of Times Beach (Missouri):** From 1984-1987, directed the scientific research that evaluated the possible health risks associated with exposure to 2,3,7,8-TCDD in soil. Managed a group of three full-time PhD research toxicologists and a number of contract labs conducting work involving exposure assessment and low-dose extrapolation analyses. Directed the original research involving a) dermal bioavailability of dioxin in soil; b) oral bioavailability of dioxin in soil; c) ingestion rates of soil by children (via Dr. Calabrese at the University of Massachusetts); d) vapor pressure measurements of dioxin; e) soil erosion wind models; f) low dose model sensitivity analyses (with Sielken); and g) dermal uptake via human skin in vitro. Resulted in eight peer-reviewed publications. Following three years of effort, in 1987, USEPA decided that our analyses had merit, and chose to recommend that the risk-specific dose (RSD) for the 1 in 1,000,000 cancer risk for dioxin be changed from 6 fg/kg-day to 100 fg/kg-day (the first USEPA reassessment). Our work on soil ingestion and our risk assessment methodology was used in decision- making for dozens of sites contaminated with dioxin in the 1980s and 1990s. At least ten peer- reviewed papers describing the work were published.

UNEXPECTED OUTCOMES

1. **A study of Los Alamos:** While working on a government contract, we determined that those involved in the Manhattan Project were unaware that they had had a small amount of radioactive material in a duct “go critical” and that the explosion and gases were blown into an adjoining housing complex on the Los Alamos site (Gaffney, S.H., E.P. Donovan, J.J. Shonka, M.H. Le, and T.E. Widner. 2013. An independent evaluation of plutonium body burdens in populations near Los Alamos Laboratory using human autopsy data. Int J Hyg Env Health 216(3):263-270. We briefed the community and our contract officer on the event. This work has been described by the DOE and DOD as an exposure assessment which is relative to their mission.

2. **Airborne endotoxins in the workplace:** On two occasions, once in the fiberglass manufacturing industry, and other time in the “shrink wrap” industry, we identified the presence of airborne endotoxins and documented higher than preferred concentrations. We recommended a significant set of changes that, in one case, required the building to be abandoned and a new factory constructed.

3. **Benzene and Pliofilm Workers:** Many years ago (circa 1990-1994), we reported that we could explain why the AML rate was so high in these workers (by job task) and that those data made sense given what was in the literature regarding threshold doses (Paustenbach, D.J., P.E. Price, W. Ollison, C. Blank, J.D. Jernigan, R.D. Bass, and D. Peterson. 1992. Re- evaluation of benzene exposure for the Pliofilm (rubber worker) cohort (1936-1976). J Toxicol Environ Health. 36(3):177- 231. In the view of many, this paper did not reflect well on the manufacturer at the time. NIOSH once believed that our assessment was unnecessarily conservative (Utterback, D. F., and Rinsky, R. A. 1995. Benzene exposure assessment in rubber hydrochloride workers: a critical evaluation of previous estimates. Am. I. Ind. Med. 27:661-676, so we re-conducted the study using a Monte Carlo analysis. The work was described in a published paper (Williams, P.R. and D.J. Paustenbach. 2003. Reconstruction of benzene exposure for the Pliofilm cohort (1936-1976) using Monte Carlo techniques. J Toxicol Environ Health A. 66(8):677-781. It seems to have satisfied the concerns of NIOSH.

4. **Beryllium in workplace air:** We recommended to a client that they needed to brief both NIOSH and OSHA that the different forms of beryllium had genuinely different hazard potential and toxicity. We also recommended that they voluntarily adopt an internal occupational exposure limit that was 10-50-fold lower than the "then current" PEL and that they should set 3-4 different OELs, depending on the form of beryllium. (Kolanz, M.E., A.K. Madl, M.A. Kelsh, M.S. Kent, R.M. Kalmes, and D.J. Paustenbach. 2001. A comparison and critique of historical and current exposure assessment methods for beryllium: Implications for evaluating risk of chronic beryllium disease. Appl Occup Environ Hyg. 16(5):593-614.; Paustenbach, D.J., A.K. Madl, and J. Greene. 2001. Identifying an appropriate occupational exposure limit (OEL) for beryllium: Data gaps and current research initiatives. Appl Occup Environ Hyg. 16(5):527-38.

5. **Brazilian Blow-Out (hair dye/straightener):** We were the first to document that it could be expected that some persons who worked in certain workplaces would experience eye and nose irritation (mostly hairdressers) when they used this particular hair straightener. We published these results in 2015 (Pierce, J.S., A. Abelmann, L. J. Spicer, R. E. Adams, M. E. Glynn, K. Neier, B. L. Finley & S. H. Gaffney. 2011. Characterization of formaldehyde exposure resulting from the use of four professional hair straightening products. J Occup Env Hyg. 8(11):686-699. The manufacturer then sued ChemRisk. The case was dropped when the State of California and Federal CPSC agreed with our assessment.

6. **Contaminants in candy:** After being asked to evaluate the presence of a contaminant that had been found in a candy, we recommended a partial recall of the product (which meant all products in the warehouse and in shipment had to be brought back and destroyed). The manufacturer had not hoped for that outcome but complied and the FDA agreed that our conclusion was appropriate.

7. **Contaminants in produce:** After evaluating a considerable amount of data over a long weekend, we recommended a food recall of kale products by a firm who had retained us regarding possible contamination by salmonella. The manufacturer had not hoped for that outcome but complied.

8. **Dioxins in beef:** Even though it was suspected that grazing animals might be the source of the intake of most dioxins found in the blood of Americans in the 1960s to the 1980s, we were the first to prove the quantitative relationship (Fries, G.F. and D.J. Paustenbach. 1990. Evaluation of potential transmission of 2,3,7,8 tetrachlorodibenzo-p-dioxin- contaminated incinerator emissions to humans via foods. J Toxicol Environ Health. 29:1- 43. Prior to our work, it had been advocated by several other consulting groups that the number of dioxins in foods was trivial and that they did not understand how Americans had developed relatively high concentrations of dioxins/furans in their adipose and blood. They suspected it might be due to inhalation or drinking water or some unknown source. Ultimately it was shown that most of the dioxins in humans was due to particulate emissions from combustors (because the particles landed on vegetation which was ingested by grazing cows and sheep).

9. **Exposure to amphiboles and mesothelioma:** Several years ago, we concluded that, historically, there were some tasks that were part of the "manufacture" of plastics that could involve significant exposure to asbestos. We then concluded that if crocidolite was used in the process, that there was likely to be a fairly high incidence of mesotheliomas. These exposures normally occurred in the 1940s and 1950s (perhaps even into the 1960s) and some epidemiology studies confirmed our suspicions.

10. **Labeling of vitamins**: After review of available information, we recommended to the client, and we reported this to FDA, that they needed to warn that a vitamin supplement containing zeolites contained concentrations of lead and cobalt that were too high to be given to children. We advocated that the warning needed to identify the presence of certain metals. This position was later embraced by the FDA and the product was no longer manufactured.

11. **PFOA in groundwater:** We conducted an analysis that explained why groundwater many miles from a facility could become contaminated via aerial deposition (Paustenbach, D.J., J.M. Panko, P.K. Scott and K.M. Unice. 2007. A methodology for estimating human exposure to perfluorooctanoic acid (PFOA): A retrospective exposure assessment of a community (1951-2003). J Toxicol Environ Health A. 70(1):28-57. This was the first occasion where any aerially distributed chemical was found to be able to reach groundwater. The EPA, NGOs and various professors were surprised that we made the connection and that we reported our data in a journal. Three major universities were hired by EPA to check whether our methodology and conclusions were correct; and they confirmed the results of our analyses (Shin H.M., V.M. Vieira, P.B. Ryan, R. Detwiler, B. Sanders, K. Steenland and S.M. Bartell. 2011. Environmental fate and transport modeling for perfluorooctanoic acid emitted from the Washington Works Facility in West Virginia. Environ Sci Technol. 15:45(4):1435-42.) A published paper by an environmental advocacy group applauded us for our work on the PFCs (Lerner, S. 2015. The Teflon Toxin, from https://theintercept.com/2015/08/11/dupont- chemistry- deception/).

12. **PFOA in groundwater due to use of water treatment sludges as soil amendments (Alabama):** We had been asked to evaluate whether sludges from former carpet and clothing treating facilities, when applied to soil, could contaminate groundwater. We concluded that in some cases, where the water table was shallow, this could occur. The EPA considered this an important work in the environmental movement.

13. **The results of the Trinity Test:** While working on a government contract, we concluded that scientists in the Federal government had underestimated the power of the Trinity Test blast. Due to this underestimate, we had to share with the city that more persons were affected by the blast than had been previously known. It is described in a published paper (Widner, T.E. and S.M. Flack. 2010. Characterization of the world's first nuclear explosion, the Trinity Test, as a source of public radiation exposure. Health Physics. 98(3):480-497. Not surprisingly, our contractor was disappointed that we had reached such a conclusion.

14. **Wear Debris and Hip Implants:** We have documented, although not the first to do so, that large wear debris from an articulating implant offers the opportunity for a myriad of adverse effects. Part of this work was published in 2017 by (Kovochich, M., E.S. Fung, E. Donovan, K.M. Unice, D.J. Paustenbach, and B.L. Finley. 2017. Characterization of wear debris from metal-on-metal hip implants during normal wear versus edge-loading conditions. J Biomed Mat Res B. Advanced online publication, May 8, 2017. doi: 10.1002/jbm.b.33902. It will likely be the basis for future in-vivo studies which will likely show that for many patients this will be the primary cause of the need for a hip replacement. We found that normal wear from a normal implant almost never produced particles which could cause the cascade of events that might cause bone erosion and the ultimate need to replace the implant. Spring of 2015.

15. **Occupational hazards at Hanford:** In 2015, members of our firm were asked to serve on an audit committee of work practices used at the Hanford site in the State of Washington (part of the original Manhattan project). The team wrote a rather jarring report that indicated that there were numerous significant shortcomings in the historical and current "health and safety" programs at this important site (report available upon request)

16. **Occupational exposure to crocidolite by cement pipe manufacturers:** We were retained around 2015 to evaluate a number of tasks involving the cutting of cement asbestos containing pipe. We reported that the airborne concentrations, although perhaps considered acceptable at the time, would be considered quite excessive by current standards.

EXPERT TESTIMONY

Available upon request

LEGISLATIVE TESTIMONY

Available upon request

SCIENCE ADVISORY PANELS

2024: Served on an advisory panel to the Sand and Gravel Manufacturing Association with 45 other asbestos scholars. This was a 3-day meeting in Virginia which was termed the Third Monticello Conference

2023: Served on the EPA Science Advisory Panel which evaluated the proposed Legacy Asbestos regulation or guideline. This document focused on the systematic review process for setting exposure limits for chrysotile, amosite, tremolite and crocidolite, as well as, the results of their evaluation. Comments were submitted and processed on Nov. 23, 2023.

2022: Served on an advisory panel to the Sand and Gravel Manufacturing Association with 45 other asbestos scholars. This was a 3-day meeting in Virginia which was termed the Second Monticello Conference. It was convened to attempt to reach consensus on the hazards of different forms of asbestos and particles that had "fiber like" characteristics.

2020: As a member of the EPA SAB, I submitted comments on the April draft of proposed EPA Updated Cancer Guidelines or what EPA calls the Human Toxicity Assessment Guidelines. Then wrote comments on May's draft of New Approach Methods and Reducing the Use of Laboratory Animals for Chronic and Carcinogenicity Testing (NAM). Then participated in a 2- day SAB meeting in June.

2020: Invited reviewer of EPA's new proposed Risk Assessment Guidelines for Chrysotile. About 139 pages of comments were submitted on May 26, 2020, to the SAB and EPA. Gave an oral presentation to SAB at the beginning of the meeting which was held in early June. Was not a panel member.

2019: Appointed to the US EPA Science Advisory Board (SAB). All members of SAB were released in February 2021. The first assignment involved reviewing the proposed EPA Updated Cancer Guidelines of 2019.

2019: Organized a conference on the scientific aspects of the talc litigation in conjunction with the DRI and the Inter Soc of Regulatory Tox and Pharm (ISRTP). Nearly 200 persons participated. September. Washington, DC.

2017: Served on an advisory panel to the Sand and Gravel Manufacturing Association with 45 other asbestos scholars. This was a 3-day meeting in Virginia which was termed the Monticello Conference. It was convened to attempt to reach consensus on the hazards of different forms of asbestos and particles that had "fiber like" characteristics.

2017: Served on an advisory panel to the City Council of Sydney, Australia regarding the recommended approach for remediating contaminated soils in downtown, which was to be the site of a new park and skyscraper.

2012: Served on the Danish SAB to evaluate the hazards of asbestos. [Represented by Dr. Christy Barlow]

2008-2014: Served on the United States Environmental Protection Agency (USEPA) Board of Scientific Counselors (BOSC) Executive committee, which has oversight for the research programs at the USEPA (ORD).

2007: Served as an outside peer-reviewer for the European Union (EU) and Health and Safety Executive (HSE) position document on how to assess the human health hazard posed by chemical mixtures in the occupational environment.

2007: Served on National Academy of Science (NAS) panel, which evaluated research involving "Soldier Protective Clothing and Equipment Feasibility of Chemical Testing Using a Fully Articulated Robotic Mannequin" (Mid-April; Washington, DC).

2007: Served as a member of the National Institute for Occupational Safety and Health (NIOSH) scientific advisory panel on Epidemiology, Exposure Assessment and Lab Medicine Methods in Occupational Health (April 10- 11; Washington, DC).

2006: Served as an invited speaker for NAS Institute of Medicine (IOM) panel on Risk Assessment for the pharmaceutical industry (May 30-31; Washington, DC).

2004-2007: Member of USEPA BOSC to review the quality of the USEPA cancer research programs at their National Center for Research (Research Triangle Park, NC).

2003: Member of the Science Advisory Board to the National Soap and Detergent Association which reviewed the International Guidelines for Conducting Risk Assessments of Consumer Products (Washington, DC).

2003: Member of the National Academy of Sciences Panel on Quality of Information (Washington, DC).

2002-2006: Appointed by the Secretary, Tommy Thompson (Human Health Services), to serve as a Member of Advisory Committee to the Director, National Center for Environmental Health, Centers for Disease Control and Prevention (CDC).

2002-2004: Member of the University of North Carolina School of Toxicology and Veterinary Sciences Science Advisory Board (Research Triangle Park, NC).

2002: Member of the Vietnam United States Scientific Delegation on Human Health and Environmental Effects of Agent Orange/Dioxin (March 3-6; Hanoi, Vietnam).

2002: Member of the USEPA-sponsored Science Advisory Panel to address the risks of dust in buildings near the World Trade Center (Oct 21-22; New York, NY).

2001: Member of the USEPA Advisory Board on Exposure Assessment for Guidelines for Complying with the Voluntary Children's Chemical Evaluation Program (VCCEP) for Children's Health (December 8-10; Washington, DC).

2001: Member of the Governor's Science Panel to evaluate human health hazard posed by ingestion of chromium (VI) in drinking water (May- August; Sacramento, CA). Resigned prior to public meeting.

2000-2001: Member of the Society of Toxicology National Panel to develop guidelines for assessing the health hazards posed by mixtures (Washington, DC).

2000-2001: Member of the USEPA Science Advisory Board that evaluated the "EPA Reassessment" of dioxin and dioxin-like compounds (Washington, DC). Chaired by Dr. Mort Lippmann.

2000: Chairman of the Expert Panel to set occupational exposure limits (OEL) for select glycol ethers [2-methoxyethanol (2-ME), 2-ethoxyethanol (2-EE), and 2-ethoxyethanol acetate (2-EEA)] for Union Carbide Corporation.

2000: Member of the Department of Energy Science Panel charged with evaluating the radiological and chemical hazards posed by the storage of the nuclear submarines in the former USSR (Chicago, IL). Chaired by Dr. Paul Ziemer; Former Assistant Secretary of Energy. No meetings were held due to the lack of funding.

2000: Member of the USEPA Science Advisory Board that evaluated benefits of reductions in exposure to hazardous air pollutants (June 22-23; Washington, DC). Chaired by Dr. Roy Albert.

2000: Served on the Harvard Science Advisory Panel charged with assessing the possible human health hazards posed by styrene (February and May; Boston, MA). Chaired by Dan Krewski.

1998-2006: Member of the Science Advisory Board of the Mickey LeLand National Urban Air Toxics Research Center, a center established by Congress to study the health hazard of airborne particles. (Houston, TX). In this role, I was responsible for innovative research grants. One of the grants, which I wrote, was for the Center to fund the development of a 24 hr, low volume, sampling pump which would allow for in-home exposure assessment. This was successfully accomplished in conjunction with SKC. It has been used since 2007 as the standard for assessing students and elderlies in the in-home environment.

1998-2001: Member of the Environmental Board of Directors for the Savannah River Site.

1997-1998: Member of the Advisory Board for the Harvard Center for Risk Analysis.

1997: Ad hoc member of the USEPA BOSC (that was charged to give guidance to the USEPA Office of Research and Development). Reviewed both the Cincinnati Engineering program and National Center for Environmental Assessment program (August and September; Washington, DC).

1997: Member of the Oversight Board for the Environmental Engineering and Sciences Program at the University of California at Los Angeles (UCLA) (March 20 and 21; Los Angeles, CA).

1996-1998: Served on the German Commission for setting national soil clean-up standards and risk assessment guidelines (Represented by Mr. Tom Long at two meetings in Munich, Germany).

1996: Member of the expert panel to set OELs for select glycol ethers (2-ME, 2- EE, and 2-EEA) established by Union Carbide Corporation.

1996: Member of the International Life Sciences Institute (ILSI) expert panel to review the new USEPA cancer risk assessment guidelines (May 8; Washington, DC).

1995-1997: Served on the United Nations' International Atomic Energy Commission to identify an international approach for classifying hazardous wastes (December 10-15, 1995; Vienna, Austria).

1995: Chairman of Risk Assessment program (with Dr. Roger McClellan) of the International Symposium on Butadiene and Isoprene (June 27-29, Blaine; WA).

1994-1995: Served on the USEPA Science Advisory Board that reviewed USEPA's Reassessment of Dioxin – 1994-95 (the second dioxin reassessment) (May 15- 16, 1995; Washington, DC). Chaired by Dr. Morton Lippmann.

1994-1995: Member of the Commission for Incorporating Epidemiology into Health Risk Assessment, sponsored by Federal Focus (November 2; Washington, DC; March 9, New York; and October 7-9; London).

1993-2001: Charter member of the Scientific Committee 87-2 (Risk-Based Waste Classification) of the National Council on Radiation Protection (NCRP). Quarterly meetings.

1991: Chairman of the Industrial Health Foundation's Expert Panel, charged with recommending an OEL for formaldehyde (June-December).

1990: Served on the Scientific Review Board of the Chemical Industry Institute of Toxicology (CIIT) on Ethylene Oxide (November 8; Chapel Hill, NC).

1988-1989: Served on USEPA ad hoc Science Advisory Board Dioxin Subcommittee (first dioxin reassessment) (December 6-8; Washington, DC). Chaired by Dr. Bernard Goldstein.

1987-2002: Consulting Member of the EPA USEPA Science Advisory Board- Environmental Health Committee (Washington, DC).

1985-1987: Appointed to the California Hazard Evaluation System and Information Services Advisory Committee. Chaired by Dr. Bob Spear, University of California at Berkeley.

1985: Appointed to one of California Governor George Deukmejian's Technical Advisory Committees of the task force on toxics, waste, and technology. The group assessed the effectiveness of California-sponsored programs for remediating hazardous waste sites (October-December). The task force was reconvened in November 1987.

1985: Chaired expert panel to assess developmental and reproductive hazards posed by glycol ethers as they were used in the semiconductor industry.

1984-1990: Member of the Council for the Health and Environmental Safety of Soils, a group established to set consensus United States cleanup goals for soils. Sponsored by the University of Massachusetts at Amherst.

1984: Served as a member of the National Nutrition Foundation Expert Panel which evaluated the 1984 proposed Congressional Office of Science and Technology Policy, "Risk Assessment Guidelines for Carcinogens."

PEER-REVIEWER OF PROPOSED GOVERNMENT REGULATORY POSITIONS/DOCUMENTS

2023: Served on the EPA Science Advisory Panel which evaluated the proposed Legacy Asbestos regulation or guideline. This document focused on the systematic review process for setting exposure limits for chrysotile, amosite, tremolite and crocidolite, as well as, the results of their evaluation. Comments were submitted and processed on Nov. 23, 2023.

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2020: Invited reviewer of the EPA's new proposed Risk Assessment Guidelines for Chrysotile. About 139 pages of comments were submitted on May 26, 2020, to the SAB and EPA. Gave an oral presentation to SAB at the beginning of the meeting which was held in early June.

2019: Submitted, by invitation, as a member of the EPA Science Advisory Board (SAB), as a member of the Chemical Assessment Advisory Committee, comments on the Proposed EPA Guidelines for Carcinogen and Non-Cancer Risk Assessment. The full report was submitted to the Honorable Andrew Wheeler (Administrator) on July 15, 2019.

2017: Was the invited outside peer-reviewer by the EPA of New South Wales for the plan to remediate and then modernize the Bangoloro site in downtown Syndey (Australia). This was a controversial remediation because it was near many residences, and it was a former gas manufacturing plant site. The work extended over 3 months.

2015: Invited peer reviewer of the new Australian guidelines for assessing risks posed by ambient air concentrations of industrial and mobile emitters.

2012: Peer-reviewer for the proposed Pharmaceutical and Personal Care Products (PCPP) regulations for water which were to be promulgated in Australia and New Zealand.

2010: Invited peer-reviewer of the British HSE document on asbestos fiber potency called the "Watch" document. (London, U.K).

2007: Invited peer-reviewer of the proposed United Kingdom guidelines on mixtures which was developed by the Royal Commission assigned to this task. (London, U.K.).

2007: Peer-reviewer of the proposed Centers of Disease Control/Agency for Toxic Substances and Disease Registry (CDC/ATSDR) guideline for dioxin/furans in contaminated soils (Atlanta, GA).

2006: Invited peer-reviewer of USEPA's Building Preliminary Remediation Goals (BPRG) calculator for radionuclides and the associated documentation and users' guide.

2005: Invited peer-reviewer of the European Union proposal to Develop a Harmonized Approach to Setting Occupational Exposure Limits (London, England).

2005: Invited peer-reviewer of the USEPA's proposed Exposure Assessment guidelines for Children (Washington, DC).

2004: Invited peer-reviewer of the NIOSH Guidelines on Assessing Risk of Dermal Uptake of Workplace Chemicals (Cincinnati, OH).

2002: Invited peer-reviewer of the NIOSH guidelines document entitled Dermal Exposure Assessment (Cincinnati, OH).

2001: Invited peer-reviewer of the Study of Benzene and Other Chemicals in the Australian Refinery Industry (Health Watch Study) for Exxon-Mobil (Australia).

2001: Invited peer-reviewer of the United Kingdom's National Guidelines for Assessing the Health Hazards Posed by Contaminated Soils (London, England).

1998: Invited peer-reviewer for the USEPA of the draft Methodology for Assessing Health Risks Associated with Multiple Pathway Exposure to Combustor Emissions (Cincinnati, OH).

1997: Invited peer-reviewer of the Draft ATSDR policy on Acceptable Concentrations of Dioxin in Soil (January: Atlanta, GA).

1996: ATSDR peer-reviewer of the draft Toxicological Profile for 2,3,7,8-Tetrachlorodibenzo- p-dioxin (dioxin) (December).

1996: Invited to be on NIOSH advisory panel on Dermal Uptake of Workplace Chemicals (represented by Dr. Brent Kerger) (September 25-26, Washington, DC).

1995: Member of the five-person CDC/ATSDR expert panel that evaluated the Bioavailability of Mercury in Soils (August 28-29; Atlanta, GA).

1994: Member of the CDC/ATSDR review panel for Toxicological Profile for Dioxin (August 10; Atlanta, GA).

1992: Member of USEPA expert panel to review the draft document Exposure Assessment of Dioxin-like Compounds (September 14: Washington, DC).

1991: Served on the Congressional Office of Technology Assessment advisory panel to evaluate Dioxin Treatment Technologies (May 29, Washington, DC). Proceedings were published as OTA-BP-0-93 and ISBN 0-16-036007-2.

1987: Reviewed the first USEPA risk-specific dose for dioxin-like compounds.

PUBLICATIONS

PEER-REVIEWED PUBLICATIONS

1. **Paustenbach, D. J.,** Kissell, K., and Shakya, A. 2025. A Comparative Toxicological and Epidemiological Evaluation of the Dioxins and PFAS Chemicals. Critical Reviews in Toxicology. 1-58.

2. Shakya, A., Andersen, M. E., Kissell, K., Underwood, P., and **Paustenbach, D.** 2025. Can four decades of experience with dioxins inform scientists and regulatory agencies about how to regulate PFAS chemicals. Journal of Toxicology and Regulatory Policy. 1(1): 1-37.

3. McCauley, K., Hua, M., Khachatoorian, C., and **Paustenbach, D.** 2025. An introduction to the Alliance for Risk Assessment Beyond Science and Decisions XIV: PFAS Limits: How Did We Get So Far Apart?. Journal of Toxicology and Regulatory Policy. 1(1): 1-25.

4. Stevens ME, **Paustenbach DJ**, Korchevskiy A. 2025. Exposure-response analysis of recent epidemiological data: Proposed risk based occupational exposure limits for various mineral types of asbestos. Chemico-Biological Interactions. 419: 1-13.

5. Smallets, S., Litvin, S., Abele, G., Kirsh, S., and **Paustenbach, D.** 2025. The acute adverse health effects of kratom: an evaluation of case reports. Frontiers in Pharmacology. 16: 1-12.

6. Turla, T., Abele, G., Hua, M., and **Paustenbach, D.** 2025. Reported associations between kratom and seizures: a systematic review. Frontiers in Pharmacology.

7. Berlinski, S., Calder, B., and **Paustenbach, D.** 2025. An evaluation and risk assessment of children's exposures to water-soluble per- and polyfluoroalkyl substances through winter gloves. Toxicology Reports. 15: 1-7.

8. Cohen, S. M., Rietjens, I. M. C. M., Levy, L., **D. Paustenbach**, and Alliance of Scientists for Evidence-Based Risk Assessment. 2025. Chemical risks and children's health—building on progress based on science, not panic. Toxicological Sciences. 206(1): 1-3.

9. Hua M., McCauley K., Brew D., Heywood J., Siracusa J., Stevens M., and **Paustenbach, D. J**. 2025. United States Environmental Protection Agency's Perfluorooctanoic Acid, Perfluorooctane Sulfonic Acid, and Related Per- and Polyfluoroalkyl Substances 2024 Drinking Water Maximum Contaminant Level: Part 1 – Analysis of Public Comments. Critical Reviews in Toxicology. 55(3): 321-367

10. **Paustenbach, D. J.,** McCauley, K., Siracusa, J., Smallets, S., Stevens, M. E., Deckard, B., Brew, D., and Hua, M. 2025. United States Environmental Protection Agency's Perfluorooctanoic Acid, Perfluorooctane Sulfonic Acid, and Related Per- and Polyfluoroalkyl Substances 2024 Drinking Water Maximum Contaminant Level: Part 2 – Fifteen Misconceptions About the Health Hazards. Critical Reviews of Toxicology. 55(3): 368-415.

11. Brew, D. W., Stevens, M. E., Langer, A. M., and **Paustenbach, D. J.** 2025. A risk assessment of mechanics who changed chrysotile asbestos containing vehicle brakes in the 1950s- early 2000s era: An update on the 2004 evaluation. Critical Reviews in Toxicology. 1-50.

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MAJOR REPORTS

1. NCRP Report No. 139 (2002). Risk-Based Classification of Radioactive and Hazardous Chemical Wastes. Scientific Committee 87-2 Members: A.G. Croff, M.J. Bell, **D.J. Paustenbach**, Y. Cohen, V.C. Rogers, L. C. Keifer, A. Wallo, III. and D.C. Kocher. National Council on Radiation Protection and Measurements, Bethesda, MD.

2. ILSI (1996). Human Health Assessment and Life-cycle Assessment: Analysis by an Expert Panel. Panelists: T.A. Burke, J. Doull, T.E. McKone, **D.J. Paustenbach**, R. Scheuplein, H.A. Udo de Haes and J.L. Young. Health and Environmental Sciences Institute, International Life Sciences Institute, Washington, DC. The meeting was held on June 7-9, 1995.

PRESENTATIONS AT SCIENTIFIC CONFERENCES

1. 2023 (October). Dioxins vs. PFOA/PFOS: Similarities and Differences. Presentation at the PFAS Conference. October 17-19, 2023. Washington, D.C.

2. 2022 (March). Current Thoughts on Risk Assessment Methodologies Used to Assess the Carcinogenic Risk of N-Nitrosodimethylamine (NDMA) and N- Nitrosodiethylamine (NDEA). Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3172. March 27-31, 2022. San Diego, CA.

3. 2022 (March). A Methodology for Estimating 4-ABP Dermal Intake via Contaminated Consumer Products. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3964. March 27-31, 2022. San Diego, CA.

4. 2022 (March). Historical Use of Laboratory Equipment as a Risk Factor for Mesothelioma: A Weight of Evidence Approach. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3908. March 27-31, 2022. San Diego, CA.

5. 2022 (March). An Analysis of Historical Workplace Exposures to Asbestos at US Steel Facilities (1972-2001). Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3961. March 27-31, 2022. San Diego, CA.

6. 2021 (May) Presented a paper at the Annual Meeting at the American Industrial Hygiene Conference & Exposition (AIHce). The title was "Asbestos Exposure During Simulated Use of Synthetic Rubber Solution Duct Sealer." The conference was held virtually on May 10-13, 2021.

7. 2021 (April) Presented a paper at the Second Monticello conference in Virginia. A meeting of 40 internationally recognized experts who were brought together to discuss asbestos and EMP. The title was "Historical Dose-Response Curves for Asbestos used by Regulatory Agencies."

8. 2020 (December) Presented paper at the annual meeting of the Society for Risk Analysis (SRA). The title was "Major changes in the risk assessment profession over the past 30 years." The Conference was virtual and was held from Dec. 14- 17, 2020.

9. 2019 (September). Presented a paper at the Conference on Scientific and Legal aspects of the talc litigation entitled "Areas of scientific disagreement regarding within the talc cases: Do they have merit?" (Washington, DC).

10. 2019 (September). The costs of chasing increasingly lower soil clean-up levels vs. the likely return in benefits to public health. Discussed at the John Evans scientific conference and celebration of his contributions to risk analysis. Harvard University, Boston, MA.

11. 2019 (March). Evaluating the Phototoxic Potential of a Hair Cleansing Conditioner. Accepted Poster Presentation at Society of Toxicology Annual Meeting, March 10-14, 2019, Baltimore, MD.

12. 2019 (March). A Tier-Based Skin Sensitization Testing Strategy for Personal Care Products. Accepted Poster Presentation at Society of Toxicology Annual Meeting, March 10-14, 2019, Baltimore, MD.

13. 2019 (March). An In Vitro Human Assay for Evaluating Immunogenic and Sensitization Potential of Personal Care and Cosmetic Products. Presentation at Society of Toxicology Annual Meeting, March 10-14, 2019, Baltimore, MD.

14. 2019 (March). Examination of the FDA Adverse Event Reporting System to Assess the Halo Effect and Potential Reporting Bias. Presentation at Society of Toxicology Annual Meeting, March 10-14, 2019, Baltimore, MD.

15. 2018 (May). Asbestos Fiber Transport from Facilities into the Community: Results of Field Studies. Presented at American Industrial Hygiene Conference & Exposition (AIHce), May 20-23, 2018, Philadelphia, PA.

16. 2018 (May). Take-home exposures following cutting cement pipe. Presented at American Industrial Hygiene Conference & Exposition (AIHce), May 20-23, 2018, Philadelphia, PA.

17. 2018 (May). Airborne Chrysotile and Crocidolite Exposure to Workers and Bystanders during Use of a Powered Abrasive Saw to Cut Asbestos-Containing Cement Pipe. Presented at American Industrial Hygiene Conference & Exposition (AIHce), May 20-23, 2018, Philadelphia, PA.

18. 2018 (March). Understanding Divergent Outcomes in MoM-THA Patient Populations with Well-Fixed Components: A Critical Appraisal of Patient Management Protocols and Revision Trends (2000-2017). Presented Society of Toxicology Annual Meeting, March 11-15, 2018, San Antonio, TX.

19. 2018 (March). Characterizing cytotoxic and inflammatory responses to metal- on-metal wear debris from normal versus edge-loading conditions. Presented at Society of Toxicology Annual Meeting, March 11-15, 2018, San Antonio, TX.

20. Hoang, M., E. Fung, D. Drechsel, K. Towle, C. Poteete, D. Paustenbach, and A. Monnot. Screening-level safety assessment of personal care product constituent safety using publicly available data. Society of Toxicology, March 11-15, 2018. San Antonio, TX.

21. 2017 (June). The 2014 Crude 4-Methylcyclohexanemethanol Chemical Release and Birth Outcomes in West Virginia. Poster presentation at Society of Epidemiologic Research, June 20-23, 2017, Seattle, WA.

22. 2017 (March). Estimating Asbestos Emissions from Former Industrial Sites and Estimating Resulting Airborne Concentrations in the Surrounding Community: A Review of Methodologies. Abstract # 3248. Poster Presentation at Society of Toxicology Annual Meeting, March 12-16, 2017, Baltimore, MD.

23. 2017 (March). Perineal Use of Cosmetic Talc as a Risk Factor for Ovarian Cancer: A Weight-of-Evidence Evaluation. Abstract # 1288. Poster Presentation at Society of Toxicology Annual Meeting, March 12-16, 2017, Baltimore, MD.

24. 2017 (March). Characteristics of Cobalt Related Cardiomyopathy in Metal Hip Implant Patients. Abstract # 2142. Poster Presentation at Society of Toxicology Annual Meeting, March 12-16, 2017, Baltimore, MD.

25. 2017 (March). Characterization of Wear Debris from Metal-on-Metal Hip Implants during Normal Wear versus Edge Loading Conditions. Abstract # 2143. Poster Presentation at Society of Toxicology Annual Meeting, March 12-16, 2017, Baltimore, MD.

26. 2017 (March). An Evaluation of Dermal Irritation Potential of Crude 4-methylcyclohexane methanol (MCHM) in Humans in 48-Hour Patch Tests. Abstract # 2950. Poster Presentation at Society of Toxicology Annual Meeting, March 12-16, 2017, Baltimore, MD.

27. 2016 (November). Health risk assessment of exposures to a high molecular weight plasticizer present in automobile interiors. Poster presentation at Society of Environmental Toxicology and Chemistry (SETAC), 37th Annual Meeting, November 6-10, 2016, Orlando, FL.

28. 2016 (May). Anthophyllite Asbestos: State of the Art Understanding of its Toxicological Properties. Podium Presentation at American Industrial Hygiene Conference & Exposition (AIHce) May 21-26, 2016, Baltimore, MD.

29. 2016 (March). Historical Ambient Airborne Asbestos Concentrations in the United States - An Analysis of Published and Unpublished Literature. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #1690. March 13-17, 2016. New Orleans, LA.

30. 2016 (March). Flame-Retardants in Upholstered Furnishings: An Assessment of Health Risk and Fire-Related Deaths in the Era of California Technical Bulletin (TB- 117). Poster Presentation at Society of Toxicology Annual Meeting. Abstract #2669. March 13-17, 2016. New Orleans, LA.

31. 2016 (March). Risks Associated with Arsenic Exposure Resulting from the Consumption of California Wines Sold in the United States. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #1264. March 13-17, 2016. New Orleans, LA.

32. 2016 (March). Estimate of 4-Methylcyclohexanemethanol (MCHM) Exposure with Normal Use of Contaminated Water during the Elk River Spill. Poster Presentation at Society of Toxicology Annual Meeting. Abstract 1693. March 13- 17, 2016. New Orleans, LA.

33. 2016 (March). QSAR Modeling Toxicity Predictions of the Constituents of Crude 4-Methylcyclohexane methanol (MCHM) and Structurally Related Chemicals. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #1445. March 13- 17, 2016. New Orleans, LA.

34. 2015 (May). Characterization of Chrysotile Asbestos Fiber Removal Rates from Air. Presentation Number: SR-119-03. Podium presentation at the American Industrial Hygiene Conference & Expo (AIHce), May 30-June 4, 2015, Salt Lake City, UT.

35. 2015 (March). Toxicology Based Cancer Causation Analysis of CoCr-Containing Hip Implants: A Quantitative Assessment of Genotoxicity and Tumorigenicity Studies. Abstract Number: 2006.Poster presentation at the 54th Annual Meeting and Society of Toxicology (SOT) Meeting at the San Diego Convention Center, March 22-26, 2015, San Diego, CA.

36. 2014 (June). Impact of the Evolving Regulatory Environment on Industrial Hygiene. Presentation Number: RT205-04. Roundtable Session at The American Industrial Hygiene Conference & Exposition (AIHce) May 31 – June 5, 2014, at the Henry B. Gonzalez Convention Center (HBGCC), San Antonio, TX

37. 2014 (March). Age-Related Trends in US Pleural Mesothelioma and Soft Tissue Sarcoma Rates: Evidence for a Longevity Effect. Abstract Number: 225. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo, March 23-27, 2014, Phoenix, AZ.

38. 2014 (March). Nanoparticles from the Wear of Cobalt-Chromium Alloy Metal- on-Metal Hip Implants: Physicochemical and Dose Analysis of Patient and Toxicology Studies. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo, Phoenix, AZ; March 23-27, 2014. Abstract Number: 603j.

39. 2014 (March). Correlation of Blood Cr (III) and Adverse Health Effects: Application of PBPK Modeling to Determine Non-toxic Blood Concentrations. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo, Phoenix, AZ; March 23- 27, 2014. Abstract Number: 2233.

40. 2014 (March). Evaluation of Four Alpha-Diketones for Toll-Like Receptor-4 (TLR- 4) Activation in Human Embryonic Kidney Cells. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo, Phoenix, AZ; March 23-27, 2014. Abstract Number: 1610.

41. 2014 (March). A Refined Co Biokinetic Model Based on Human Data. Presented at Society of Toxicology 53rd Annual Meeting and ToxExpo, Phoenix, AZ; March 23- 27, 2014. Abstract Number: 77.

42. 2013 (August). Pulmonary Obstruction among Workers at a Food Flavorings Manufacturing Facility. Poster presentation at The International Society of Exposure Science (ISES), Basel, Switzerland, August 19 - 23, 2013. Abstract ID: 5315.

43. 2013 (August). Occupational Exposures Measured in a Food Flavoring Manufacturing Facility. Poster presentation at The International Society of Exposure Science (ISES), Basel, Switzerland, August 19 - 23, 2013. Abstract ID: 5397.

44. 2013 (May). Evolution of Warnings and Labels on Encapsulated Asbestos- Containing Products (1930–1990). Session: Legal, Regulatory, Guidelines, Standards. Poster presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 21, 2013; 10:00 AM – 12:00 PM. Presentation Number: SR-403-08.

45. 2013 (May). Historical Outdoor Airborne Asbestos Concentrations Associated with Emission Sources in the United States: A Review of Published and Unpublished Data. Session: Aerosols. Poster presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 20, 2013; 10:00 AM – 12:00 PM. Presentation Number: SR-401-03.

46. 2013 (May). Measurement of Airborne Asbestos Fiber Settling Rates in a Simulation Study of Clothes Handling. Session: Exposure Assessment Strategies. Podium presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 20, 2013; 3:00 PM – 3:30 PM. Presentation Number: SR-108-04.

47. 2013 (May). Evaluation of Potential Para-Occupational Exposure to Chrysotile Asbestos during Laundering Activities through a Simulation Study. Session: Exposure Assessment Strategies. Podium presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 20, 2013; 3:30 PM – 4:00 PM. Presentation Number: SR-108-05

48. 2013 (May). Exposure to Food Flavorings at a Manufacturing Facility. Session: IH General Practice. Podium presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 22, 2013; 2:00 PM – 2:30 PM. Presentation Number: SR-131-03.

49. 2013 (May). Pulmonary Restriction among Workers at a Food Flavoring Manufacturing Facility; A Follow Up Study. Session: Occupational and Environmental Epidemiology. Podium presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 23, 2013; 1:30 PM – 2:00 PM. Presentation Number: SR-146-03

50. 2013 (March). Cobalt Whole Blood Concentrations in Healthy Adult Volunteers Following Two-Weeks of Ingesting a Cobalt Supplement. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas, March 12, 2013. Safety Assessment: Non pharmaceuticals; 1:00 P.M. to 4:30 P.M.; Exhibit Halls C&D. Abstract number 1555/Poster Board 616.

51. 2013 (March). Airborne Diacetyl from Cooking and Consumption of Microwave Popcorn: Estimation of Consumer Exposure with a Two-zone Near-field/Far- field Model. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 12, 2013. Exposure Assessment: New Characterizations, Methods, and Models; 1:00 P.M. to 4:30 P.M.; Exhibit Halls C&D. Abstract number 1268/Poster Board 230.

52. 2013 (March). Method: Measuring Protein-Bound and Free Cobalt (II) in Human Serum - Size Exclusion Liquid Chromatography with ICP-MS. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 12, 2013. Metals I; 9:00 A.M. to 12:30 P.M.; Exhibit Halls C&D. Abstract number 1168/Poster Board 620.

53. 2013 (March). Dose-response relationships for blood cobalt concentrations and associated health effects. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 12, 2013. Metals II; 1:00 P.M. to 4:30 P.M.; Exhibit Halls C&D. Abstract number 1514/Poster Board 573.

54. 2013 (March). Cobalt blood concentrations and health effects in adult volunteers during a 90-day cobalt supplement ingestion study. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 14, 2013. Late-Breaking Abstract Session; 8:30 A.M. to 12:00 P.M.; Exhibit Hall A. Abstract number 2556/Poster Board 144.

55. 2013 (March). Lack of an Association between Cumulative Exposure to Diacetyl and Changes in Pulmonary Health among Workers at a Food Flavorings Manufacturer. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 11, 2013. Epidemiology: Exposures and Associations; 1:00 P.M. to 4:30 P.M.; Exhibit Halls C&D. Abstract number 478/Poster Board 253.

56. 2013 (March). Speciation of Chromium Released from Metal-on-Metal Hip Implants. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 12, 2013. Metals I; 9:00 A.M. to 12:30 P.M.; Exhibit Halls C&D. Abstract number 1166/Poster Board 618.

57. 2013 (March). Effects of Cobalt Dietary Supplementation on Cobalt Body Burden, Steady-State Levels and Selected Biochemical Parameters. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 14, 2013. Late-Breaking Abstract Session; 8:30 A.M. to 12:00 P.M.: Exhibit Hall A. Abstract number 2555/Poster Board 143.

58. 2012 (December). Evaluation of Chrysotile Fiber Adherence to Clothing Exposed to Known Airborne Asbestos Concentrations Before and After Handling and Shaking Out of the Clothing. Presented at the 2012 Society for Risk Analysis (SRA) Annual Meeting; December 9-12, 2012; San Francisco, CA.

59. 2012 (December). Derivation of a Chronic Oral Reference Dose for Cobalt. Presented at the 2012 Society for Risk Analysis (SRA) Annual Meeting; December 9-12, 2012; San Francisco, CA.

60. 2012 (December). Evaluation of Potential Take Home Exposure during Laundering Activities: A Simulation Study of Airborne Chrysotile Concentrations Associated with Handling Clothing Exposed to Known Levels of Airborne Asbestos. Presented at the 2012 Society for Risk Analysis (SRA) Annual Meeting; December 9-12, 2012; San Francisco, CA.

61. 2012 (December). Investigation of Cobalt Steady-State Levels in Five Healthy Adult Volunteers Taking 14-Daysof a Cobalt Supplement. Presented at the 2012 Society for Risk Analysis (SRA) Annual Meeting; December 9-12, 2012; San Francisco, CA.

62. 2012 (October). Evaluation of Potential Take Home Exposure during Laundering Activities: A Simulation Study of Airborne Chrysotile Concentrations Associated with Handling Clothing Exposed to Known Levels of Airborne Asbestos. Presenting at the 2012 International Society of Exposure Science conference, Seattle WA: October 28-Nov 1, 2012.

63. 2012 (October). Evaluation of Chrysotile Fiber Adherence to Clothing Exposed to Known Airborne Asbestos Concentrations Before and After Handling and Shaking Out of the Clothing. Presenting at the 2012 International Society of Exposure Science conference, Seattle WA: October 28-Nov 1, 2012.

64. 2012 (June). Comparing Pulmonary Function Data in Flavorings Manufacturing Workers. Presented at the 45th Society for Epidemiological Research (SER) Annual Meeting. June 27 - 30, 2012, Minneapolis, MN.

65. 2012 (June). Analysis and Modeling of Airborne BTEX Concentrations from the Deepwater Horizon Oil Spill. To be presented at the "Assessing Exposure During Disaster Response: The Gulf Oil Spill Experience" roundtable. Presented at 2012 American Industrial Hygiene Conference & Expo (AIHce), June 16-21, 2012, Indianapolis, IN.

66. 2012 (March). Is dermal sensitization for 1,2-benzisothiazolin-3-one [BIT] in consumer products a cause for concern? Presented at Society of Toxicology's (SOT) 51st Annual Meeting, March 11–15, 2012, at the Moscone Convention Center in San Francisco, California. March 12, 2012. Safety Evaluation: Nonpharmaceuticals; 9:30 AM - 12:30 PM; Exhibit Hall; 273 Poster Board -360.

67. 2012 (March). An Analysis of Historical Exposures of Pressmen to Airborne Benzene (1930s to 2006). Presented at Society of Toxicology's (SOT) 51st Annual Meeting, March 11–15, 2012, at the Moscone Convention Center in San Francisco, California. March 14, 2012. Exposure Assessment: Case-Specific Characterizations; 9:00 AM - 12:30 PM; Exhibit Hall; 2106 Poster Board -530.

68. 2012 (March). Derivation of LOAEL and NOAEL for Tremolite Asbestos. Presented at Society of Toxicology's (SOT) 51st Annual Meeting, March 11–15, 2012, at the Moscone Convention Center in San Francisco, California. March 14, 2012. Exposure Assessment: Case-Specific Characterizations; 9:00 AM -12:30 PM; Exhibit Hall; 2114 Poster Board -538.

69. 2012 (March). Tremolite Asbestos Exposures Associated with the Use of Commercial Products. Presented at Society of Toxicology's (SOT) 51st Annual Meeting, March 11–15, 2012, at the Moscone Convention Center in San Francisco, California. March 14, 2012. Exposure Assessment: Case-Specific Characterizations; 9:00 AM - 12:30 PM; Exhibit Hall; 2115 Poster Board -539.

70. 2012 (March). Human PBPK Modeling of Benzene Inhalation Based Chinese Worker Urinary Metabolite Data: Comparison of Human and Mouse Metabolism. Presented at Society of Toxicology's (SOT) 51st Annual Meeting, March 11–15, 2012, at the Moscone Convention Center in San Francisco, California. March 14, 2012. Disposition and Pharmacokinetics; 9:00 AM - 12:30 PM; Exhibit Hall; 1893 Poster Board -226.

71. 2011 (October). Evaluation of Para-Occupational Exposures to Asbestos: A Review of the Literature (1900-Present). Presented at the 21st Annual ISES Conference. Advancing Exposure Science for Environmental Health. October 23 - 27, 2011. Baltimore, Maryland.

72. 2011 (October). Government and Navy knowledge regarding health hazards of asbestos: A state of the science evaluation (1900 to 1970). Presented at the 21st Annual ISES Conference. Advancing Exposure Science for Environmental Health. October 23 - 27, 2011. Baltimore, Maryland.

73. 2011 (October). Airborne Concentrations of Metals and Total Dust during Solid Catalyst Loading and Unloading Operations at a Petroleum Refinery. Presented at the 21st Annual ISES Conference. Advancing Exposure Science for Environmental Health. October 23 - 27, 2011. Baltimore, Maryland.

74. 2011 (May). Analysis of Historical Industrial Hygiene Data: A Case Study Involving Benzene Exposures at a Petrochemical Manufacturing Facility (1974- 1999). Presentation at American Industrial Hygiene Conference and Expo (AIHce), May 14 – May 19, 2011, Portland, Oregon.

75. 2011 (May). Exposure Reconstruction in Occupational Human Health Risk Assessment: Current Methods and a Recommended Framework. Presented at American Industrial Hygiene Conference and Expo (AIHce), May 14 – May 19, 2011, Portland, Oregon.

76. 2011 (May). The Role of Exposure Reconstruction in Occupational Human Health Risk Assessment: Current Methods and a Recommended Framework. Presented at American Industrial Hygiene Conference and Expo (AIHce), May 14 – May 19,2011, Portland, Oregon.

77. 2011 (May). Determinants of Deathly Carbon Monoxide Exposure inside a Recreational Vehicle. Presented at American Industrial Hygiene Conference and Expo (AIHce), May 14 – May 19, 2011, Portland, Oregon.

78. 2011 (April). Putting on the brakes or stepping on the gas? The future of automotive asbestos litigation. Presented at the 2011 American Bar Association meeting on Emerging Issues in Motor Vehicle Product Liability Litigation meeting. March 30- April 1, 2011, Phoenix, AZ.

79. 2011 (March). Product sustainability: The role of chemical watch lists in chemical deselection from products manufacturing processes. Presented at the Society of Toxicology (SOT) Annual Meeting, Thursday, March 10, 2011. 8:30 AM to 12:00 Noon. Washington, D.C.

80. 2011 (March). A Case Control Study of Chronic Myelomonocytic Leukemia in Shanghai, China. Presented at the Society of Toxicology (SOT) Annual Meeting, Tuesday, March 8, 2011. 9:00 AM to 12:30 PM. Abstract #1210, Poster Board #713. Washington, D.C. (Epidemiology and Exposure Evaluations)

81. 2011 (March). Evaluation of Airborne Toxicant Concentrations from the Deepwater Horizon Oil Spill. Presented at the Society of Toxicology (SOT) Annual Meeting, Tuesday, March 8, 2011. 9:00 AM to 12:30 PM. Abstract #1262, Poster Board #834. Washington, D.C. (Exposure Assessments and Biomonitoring Applications)

82. 2011 (March). Reverse Causation of Dioxin Dose-Response Trends for Risk of Diabetes Mellitus Type 2 Among Operation Ranch Hand Vietnam Veterans. Presented at the Society of Toxicology (SOT) Annual Meeting, Wednesday, March 9, 2011. 1:00 PM to 4:30 PM. Abstract #2255, Poster Board #359. Washington, D.C. [Persistent Organic Compounds (POPs)]

83. 2011 (March). An assessment of the bioavailability of cadmium in thin-film PV modules. Presented at the Society of Toxicology (SOT) Annual Meeting, Thursday March 10, 2011. 8:30 AM – 12:00 PM. Abstract #2909, Poster Board #155. Exhibit Hall, Convention Center. Washington, D.C. (Grace Period Abstracts - Session IV)

84. 2010 (December). Final Results of CDC's Los Alamos Historical Document Retrieval and Assessment (LAHDRA) project. Presented at the Society for Risk Analysis (SRA) Annual Meeting, Wednesday December 8, 2010. Abstract #M3- E.5. Salt Lake City, UT.

85. 2010 (December). Estimating the airborne concentrations of benzene and other relevant volatiles in boats operating in or near oil spills: A comparison between the Exxon-Valdez and Deepwater Horizon incidents. Presented at the Society for Risk Analysis (SRA) Annual Meeting, Wednesday December 8, 2010. Abstract #W3-E.1. Salt Lake City, UT.

86. 2010 (December). Reflections on the role of risk assessment at OSHA over the 40-year journey: has it been a big disappointment? Presented at the Society for Risk Analysis (SRA) Annual Meeting, Wednesday December 8, 2010. Abstract #M3-A.4.

87. 2010 (November). Possible risks associated with coalbed methane-produced water. Presented at the Society of Environmental Toxicology and Chemistry (SETAC) North America 31st Annual Meeting; Monday November 8th, 2010. Abstract #MP235. Portland, OR.

88. 2010 (May). Task-based analysis of benzene air concentrations associated with refinery operations. American Industrial Hygiene Conference & Exposition, May 22 to May 27, 2010. Podium presentation in Hazardous Occupational Exposures – Case Studies session, May 24, 2010. Denver, CO.

89. 2010 (March). Job-based analysis of benzene air concentrations associated with refinery operations. Poster presentation at The Society of Toxicology's Annual Meeting. # 1851 PB 406. March 7 – 11, 2010. Salt Lake City, UT.

90. 2010 (March). Gasoline: Unappreciated villain or unwarranted suspect? Poster presentation at The Society of Toxicology's Annual Meeting. # 124 PB 206. March 7-11, 2010. Salt Lake City, UT.

91. 2010 (April). Reflections on thirty years in chemical risk assessment… and thoughts about the next ten years. Presented at the Conference on Environmental Decisions: Risks and Uncertainties – Switzerland. April 25-29, 2010, Centro Stefano Franscini, Monte Verità, Switzerland.

92. 2009 (September). Gasoline: A complex chemical mixture or a dangerous vehicle for benzene exposure. Presented at Benzene 2009: Health Effects and Mechanisms of Bone Marrow Toxicity Implications for t-AML and the Mode of Action Framework, September 7-11, 2009, Munich, Germany.

93. 2009 (September). Job and task-based analysis of benzene air concentrations associated with refinery operations. Presented at Benzene 2009: Health Effects and Mechanisms of Bone Marrow Toxicity Implications for t-AML and the Mode of Action Framework, September 7-11, 2009, Munich, Germany.

94. 2009 (September). Comparison of modeled and measured concentrations of airborne benzene from the use of petroleum-based solvents spiked with low levels of benzene. Presented at Benzene 2009: Health Effects and Mechanisms of Bone Marrow Toxicity Implications for t-AML and the Mode of Action Framework, September 7-11, 2009, Munich, Germany.

95. 2009 (August). Distribution of polychlorinated biphenyl concentrations in wild- caught and farm-raised shrimp by geographic region. Presented at the 29th International Symposium on Halogenated Persistent Organic Pollutants, August 23-28, 2009, Beijing, China.

96. 2009 (August). Concentrations of polychlorinated biphenyls in seafood products from the U.S. retail market. Presented at the 29th International Symposium on Halogenated Persistent Organic Pollutants, August 23-28, 2009, Beijing, China.

97. 2009 (August). An investigation of wild-caught and farm-raised shrimp samples with high concentrations of polychlorinated biphenyls. Presented at the 29th International Symposium on Halogenated Persistent Organic Pollutants, August 23-28, 2009, Beijing, China.

98. 2009 (March). Toxicogenetics and toxicogenomics – Science fiction or the future of toxic torts? Presented at the Defense Research Institute's Toxic Torts and Environmental Law Seminar, March 19-20, 2009, Phoenix, AZ.

99. 2009 (March). Meddling with science – Is scientific research manipulated for purposes of litigation or regulation? Presented at the Defense Research Institute's Toxic Torts and Environmental Law Seminar, March 19-20, 2009, Phoenix, AZ.

100. 2009 (March). Potential for occupational exposures of hairdressers to vinyl chloride in hairspray (1967-1974). Presented at the 48th Annual Society of Toxicology's Meeting and ToxExpo, March 15-19, 2009. Baltimore, MD.

101. 2009 (March). Mechanic and bystander chrysotile exposures during heavy equipment brake removal. Presented at the 48th Annual Society of Toxicology's Meeting and ToxExpo, March 15-19, 2009. Baltimore, MD.

102. 2009 (March). Lead testing wipes contain measurable background levels of lead. Presented at the 48th Annual Society of Toxicology's Meeting and ToxExpo, March 15-19, 2009. Baltimore, MD.

103. 2009 (March). PBPK Modeling of benzene metabolites in bone marrow of humans with varied workplace exposure patterns and CYP2E1 activity. Presented at the 48th Annual Society of Toxicology's Meeting and ToxExpo, March 15-19, 2009. Baltimore, MD.

104. 2009 (January). Reflections on the future practice of occupational hygiene. Presented at the YUMA Industrial Hygiene Meeting, January 21-23, 2009, San Diego, CA.

105. 2008 (October). A review of historical ambient airborne asbestos concentrations in cities and buildings: 1950s to the present day. Abstract 1274. Presented at the Annual International Society of Exposure Analysis Conference, October 12-16, 2008. Pasadena, CA.

106. 2008 (October). Evaluation of bystander exposure to asbestos: Review of the literature (1950s – present). Abstract 1011. Presented at the Annual International Society of Exposure Analysis Conference, October 12-16, 2008. Pasadena, CA.

107. 2008 (October). Potential occupational exposures of hairdressers to vinyl chloride in hairspray (1962-1973). Abstract 1150. Presented at the Annual International Society of Exposure Analysis Conference, October 12-16, 2008. Pasadena, CA.

108. 2008 (October). Conducting risk assessments of chemicals in consumer products. Abstract 922. Presented at the Annual International Society of Exposure Analysis Conference, October 12-16, 2008. Pasadena, CA.

109. 2008 (October). Benzene exposure in refinery workers (1976-2006). Abstract 864. Presented at the Annual International Society of Exposure Analysis Conference, October 12-16, 2008. Pasadena, CA.

110. 2008 (August). Principal components analysis (PCA) of PBDE concentrations in fish tissue from southern Mississippi. Presented at the 28th International Symposium on Halogenated Persistent Organic Pollutants (POPs) – Dioxin Conference, August 17-22, 2008. Birmingham, England, UK.

111. 2008 (August). Analysis of fish tissue concentrations of dioxins and furans using principal components analysis (PCA). Presented at the 28th International Symposium on Halogenated Persistent Organic Pollutants (POPs) – Dioxin Conference, August 17-22, 2008. Birmingham, England, UK.

112. 2008 (June). Comparing equal delivered doses of airborne benzene for 8 hr/day steady exposure vs. peak exposure regiments using a PBPK model. Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Minneapolis, MN.

113. 2008 (June). Comparison of modeled and measured concentrations of airborne benzene from the use of petroleum-based solvents spiked with low levels of benzene. Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Minneapolis, MN.

114. 2008 (June). Validation of two different exposure models using the results of a simulation study involving exposures to methanol vapors during the cleaning of semiconductor wafers. Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Minneapolis, MN.

115. 2008 (June). Benzene exposure in refinery workers: Baytown, TX (1978-2006). Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Minneapolis, MN.

116. 2008 (June). Residential and occupational indoor surface dust criteria for PAHs and PCDD/PCDF. Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Minneapolis, MN.

117. 2008 (June). Analysis of historical air monitoring data for copper beryllium at a manufacturing plant (1964-2000). Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Minneapolis, MN.

118. 2008 (June). Groundwater vapor intrusion into a school – modeled concentrations vs. monitoring results. Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Minneapolis, MN.

119. 2008 (March). Assessment of the health risks posed by benzene in certain soft drinks. Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Seattle, WA.

120. 2008 (March). The importance of asbestos fiber length as a predictor of potency for asbestos-related disease. Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Seattle, WA.

121. 2008 (March). Occupational exposure to benzene at the ExxonMobil refinery in Joliet, Illinois (1977-2006). Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Seattle, WA.

122. 2008 (March). A study of airborne chrysotile concentrations associated with handling, unpacking, and repacking boxes of automobile clutch discs (circa 1950-1980). Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Seattle, WA.

123. 2008 (March). Detailed asbestos fiber size and morphology analyses of automobile clutch discs, brake pads, and brake shoes. Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Seattle, WA.

124. 2008 (March). Cancer mortality in Chinese populations surrounding an alloy plant with chromium smelting operations (1960-1978). Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Seattle, WA.

125. 2008 (March). The possible community health hazards posed by emissions from automobile traffic tunnels: A review of the literature. Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Seattle, WA.

126. 2008 (April). Lessons learned after 10 years of brake dust litigation. Presented paper at American Bar Association. Biltmore Hotel, Phoenix, Arizona.

127. 2007 (December). Keynote speaker at the annual national meeting of the Australian Institute of Occupational Hygiene (AIOH). Held in Melbourne, Australia.

128. 2007 (October). Exposure to chrysotile asbestos associated with handling, unpacking, and repacking boxes of automobile clutch discs. Presented at the 17th Annual Conference of the International Society of Exposure Analysis, October 14-18, 2007. Durham/Research Triangle Park, NC.

129. 2007 (October). Residential area development and potential for public exposures around the Manhattan Project and early AEC sites. Presented at the 17th Annual Conference of the International Society of Exposure Analysis, October 14-18, 2007. Durham/Research Triangle Park, NC.

130. 2007 (October). Factors influencing the development of warnings on regulated and non-regulated consumer products. Presented at the 17th Annual Conference of the International Society of Exposure Analysis, October 14-18, 2007. Durham/Research Triangle Park, NC.

131. 2007 (September). Chair for session regarding the study of soils in Midland, Michigan. 30th International Dioxin Meeting, Sept. 4-8. Tokyo, Japan.

132. 2007 (July). A review of the world's first test of a nuclear device (Trinity Site, July 16, 1945) and potential radiation exposures to residents of New Mexico. Presented at the 52nd Annual Meeting of the Health Physics Society, July 8-12, 2007. Portland, OR.

133. 2007 (July). A review of residential areas developed around Manhattan Project and early AEC sites and potential pathways for public exposures. Presented at the 52nd Annual Meeting of the Health Physics Society, July 8-12, 2007. Portland, OR.

134. 2007 (March). Meta-analysis of asbestos-related disease among skilled craftsmen in various occupational settings. Presented at the Society of Toxicology (SOT), March 25-29, 2007. Charlotte, NC.

135. 2007 (March). Development of PCDD/F and dioxin-like PCB serum concentration reference values for the general U.S. population using the 2005- WHO TEFs and the 2001-2002 NHANES data. Presented at the Society of Toxicology (SOT), March 25- 29, 2007. Charlotte, NC.

136. 2007 (March). Assessment of potential human health risks posed by benzene in a commercial beverage. Presented at the Society of Toxicology (SOT), March 25-29, 2007. Charlotte, NC.

137. 2007 (March). Dietary intake of PBDEs based on consumption of catfish in Southern Mississippi. Presented at the Society of Toxicology (SOT), March 25- 29, 2007. Charlotte, NC.

138. 2007 (March). Occupational exposures associated with petroleum-derived solvents containing trace levels of benzene. Presented at the Society of Toxicology (SOT), March 25-29, 2007. Charlotte, NC.

139. 2007 (February). Role of practice of industrial hygiene. Presented at the NASA Ames Research Center, February 14, 2007. Mountain View, CA.

140. 2006 (September). Evaluation of bricklayer exposures to airborne asbestos in steel mills (1972-1981). Presented at the International Conference on Environmental Epidemiology and Exposure (ISEE/ISEA), September 5-6, 2006. Paris, France.

141. 2006 (September). Background concentrations of airborne asbestos onboard maritime shipping vessels (1978-1992). Presented at the International Conference on Environmental Epidemiology and Exposure (ISEE/ISEA), September 2-6, 2006. Paris, France.

142. 2006 (September). Assessment of the beryllium lymphocyte proliferation test: Lessons learned from a long-term occupational surveillance program. Presented at the International Conference on Environmental Epidemiology and Exposure (ISEE/ISEA), September 5-6, 2006. Paris, France.

143. 2006 (September). Retrospective exposure assessment of asbestos related to skilled craftsmen at a petroleum refinery in Beaumont, Texas (1940-2005). Presented at the International Conference on Environmental Epidemiology and Exposure (ISEE/ISEA), September 4, 2006. Paris, France.

144. 2006 (September). The challenge of setting Occupational Exposure Limits (OELs) for odorants and irritants. Presented at the International Conference on Environmental Epidemiology and Exposure (ISEE/ISEA), September 4, 2006. Paris, France.

145. 2006 (September). Airborne concentrations of benzene associated with the historical use of Liquid Wrench. Presented at the International Conference on Environmental Epidemiology and Exposure (ISEE/ISEA), September 4, 2006. Paris, France.

146. 2006 (September). Factors affecting the bioaccessibility and bioavailability of hexavalent chromium and dioxin contaminants in soil, and their relative risk assessment. Presented at the International Conference on Environmental Epidemiology and Exposure (ISEE/ISEA), September 4, 2006. Paris, France.

147. 2006 (September). Reliability of spot urine samples. Poster Session I. Presented at the International Conference on Environmental Epidemiology and Exposure (ISEE/ISEA), September 3-4, 2006. Paris, France.

148. 2006 (August). Development of PCDD/F TEQ serum reference values for the U.S. Population for use in evaluating biomonitoring results. Presented at the 26th International Symposium on Halogenated Environmental Organic Pollutants and POPs, August 21-25, 2006. Oslo, Norway.

149. 2006 (August). Comparing polychlorinated biphenyls in farm-raised and wild- caught catfish from Southern Mississippi. Presented at the 26th International Symposium on Halogenated Environmental Organic Pollutants and POPs, August 21-25, 2006. Oslo, Norway.

150. 2006 (August). Polybrominated diphenyl ethers in southern Mississippi catfish. Presented at the 26th International Symposium on Halogenated Environmental Organic Pollutants and POPs, August 21-25, 2006. Oslo, Norway.

151. 2006 (August). Are dioxin body burdens surrogates for other risk factors in associations between dioxin and diabetes? Presented at the 26th International Symposium on Halogenated Environmental Organic Pollutants and POPs, August 21- 25, 2006. Oslo, Norway.

152. 2006 (August). Comparing PCDDs, PCDFs, and dioxin-like PCBs in farm-raised and wild-caught catfish from Southern Mississippi. Presented at the 26th International Symposium on Halogenated Environmental Organic Pollutants and POPs, August 23, 2006. Oslo, Norway.

153. 2006 (May). Reconstruction of exposure of skilled craftsmen to asbestos at the Beaumont, Texas Refinery (1946-2004). Presented at the American Industrial Hygiene Conference & Expo (AIHce), May 13-18, 2006. Chicago, IL.

154. 2006 (May). Reconstruction of benzene exposures during the simulated use of a penetrating and de-rusting agent. Presented at the American Industrial Hygiene Conference & Expo (AIHce), May 13-18, 2006. Chicago, IL.

155. 2006 (May). Assessment of exposure-response patterns for beryllium sensitization and chronic beryllium disease. Presented at the American Industrial Hygiene Conference & Expo (AIHce), May 13-18, 2006. Chicago, IL.

156. 2006 (May). Total phenol and t,t-muconic acid as biomarkers for individual exposure to benzene: evidence of high background concentrations in some persons. Invited presentation at the AIHce & Vent Conference, May 13-18, 2006. Chicago, IL.

157. 2006 (March). DNA-protein crosslinks as a potential biomonitor of hexavalent chromium exposure in rainbow trout. Presentation at the Society of Toxicology's 45th Annual Meeting, March 5-9, 2006. San Diego, CA.

158. 2006 (March). An integrated toxicokinetic model for estimating childhood body burdens of dioxins based on various studies. Presentation at the Society of Toxicology's 45th Annual Meeting, March 5-9, 2006. San Diego, CA.

159. 2006 (March). Implications of age-dependent half-lives of dioxins on assessment of breast milk dose and body burden. Presentation at the Society of Toxicology's 45th Annual Meeting, March 5-9, 2006. San Diego, CA.

160. 2006 (March). Dioxin and diabetes: Does the current weight of evidence demonstrate a relationship? Presentation at the Society of Toxicology's 45th Annual Meeting, March 5-9, 2006. San Diego, CA.

161. 2006 (March). Persistence of DNA-protein crosslinks in erythrocytes of channel catfish after acute hexavalent chromium exposure. Presentation at the Society of Toxicology's 45th Annual Meeting, March 5-9, 2006. San Diego, CA.

162. 2006 (March). Beryllium exposure and the prevalence of chronic beryllium disease and beryllium sensitization. Presentation at the Society of Toxicology's 45th Annual Meeting, March 5-9, 2006. San Diego, CA.

163. 2006 (March). High background levels of urinary benzene metabolites found in volunteer study. Presentation at the Society of Toxicology's 45th Annual Meeting, March 5-9, 2006. San Diego, CA.

164. 2006 (March). Evaluation of PCDD/F and dioxin-like PCB serum concentration data from the 2001-2002 National Health and Nutrition Survey in the United States. Presentation at the Society of Toxicology's 45th Annual Meeting, March 5- 9, 2006. San Diego, CA.

165. 2006 (March). Reconstruction of benzene exposures during the simulated use of a penetrating and de-rusting agent. Presentation at the Society of Toxicology's 45th Annual Meeting, March 5-9, 2006. San Diego, CA.

166. 2006 (March). Reconstruction of exposure of skilled craftsmen to asbestos at the Beaumont, Texas Refinery (1946-2004). Presentation at the Society of Toxicology's 45th Annual Meeting, March 5-9, 2006. San Diego, CA.

167. 2006 (March). Hormesis: A challenge to the linear dose-response model, and its implications in risk assessment, regulatory policy, and biomedical research. Presentation at the Society of Toxicology's 45th Annual Meeting, March 5-9, 2006. San Diego, CA.

168. 2005 (November). Exposures associated with tasks performed on marine vessels. Annual International Society of Exposure Analysis Conference (ISEA), October 30-November 3, 2005. Tucson, AZ.

169. 2005 (November). A historical review of asbestos exposures among skilled craftsmen. Annual International Society of Exposure Analysis Conference (ISEA), October 30-November 3, 2005. Tucson, AZ.

170. 2005 (November). Exposure and the prevalence of chronic beryllium disease and beryllium sensitization. Annual International Society of Exposure Analysis Conference (ISEA), October 30-November 3, 2005. Tucson, AZ.

171. 2005 (November). Estimation of inhalation exposures to the OFF! Mosquito Coil III following indoor use. Annual International Society of Exposure Analysis Conference (ISEA), October 30-November 3, 2005. Tucson, AZ.

172. 2005 (November). Airborne benzene exposure of mechanics and gasoline service station attendants. Annual International Society of Exposure Analysis Conference (ISEA), October 30-November 3, 2005. Tucson, AZ.

173. 2005 (September). Incorporating hormesis into the risk assessment paradigm. 9th International Conference on Environmental Mutagens & 36th Annual Meeting of the Environmental Mutagen Society, September 3-8, 2005. San Francisco, CA.

174. 2005 (August). Age and concentration-dependent TCDD elimination half-life in Seveso children. 25th International Symposium on Halogenated Environmental Organic Pollutants and POPs, 20th International Symposium on Polycyclic Aromatic Compounds, August 21-26, 2005. Toronto, Canada.

175. 2005 (August). Age and concentration-dependent elimination half-lives of chlorinated dibenzofurans in Yusho and Yucheng patients. 25th International Symposium on Halogenated Environmental Organic Pollutants and POPs, 20th International Symposium on Polycyclic Aromatic Compounds, August 21-26, 2005. Toronto, Canada.

176. 2005 (August). Identifying a soil clean-up criteria for dioxin in residential soils: How has 20 years of research and risk assessment experience impacted the analysis? 25th International Symposium on Halogenated Environmental Organic Pollutants and POPs, 20th International Symposium on Polycyclic Aromatic Compounds, August 21- 26, 2005. Toronto, Canada.

177. 2005 (August). Retrospective modeling of potential residential exposure to perfluorooctanic acid (PFOA) releases from a manufacturing facility. First International Symposium on Fluorinated Alkyl Organics in the Environment, August 18-20, 2005. Toronto, Canada.

178. 2005 (May). Introductory lecture on "Recent trends in asbestos research: The old is like the new." Annual meeting of the American Industrial Hygiene Association. May 24. Anaheim, CA.

179. 2005 (March). Updating exposure limits: How to preserve the future of the industrial hygiene profession. Presentation at Organization for Resource Councilors Annual Scientific Conference, California Section. Napa, CA.

180. 2005 (March). An age-dependent half-life model for estimating childhood body burdens of dibenzodioxins and dibenzofurans. Presentation at the Society of Toxicology's 44th Annual Meeting, March 6-10, 2005. New Orleans, LA.

181. 2005 (March). Chrysotile asbestos exposure associated with removal of automobile exhaust systems (Circa 1946-1970). Presentation at the Society of Toxicology's 44th Annual Meeting, March 6-10, 2005. New Orleans, LA.

182. 2005 (March). Exposure reconstruction of historical airborne benzene concentrations: Case study of a deck crewman on board crude oil and chemical tankers. Presentation at the Society of Toxicology's 44th Annual Meeting, March 6-10, 2005. New Orleans, LA.

183. 2005 (March). Current regulatory and scientific views regarding chemical hazards to children. Presentation at the Society of Toxicology's 44th Annual Meeting, March 6-10, 2005. New Orleans, LA.

184. 2004 (December). Evaluating asbestos exposures associated with vehicle brake cleaning and machining activities using short-term and TWA measurements. Annual Meeting of Society for Risk Analysis (SRA), Conference, December 5-8, 2004. Palm springs, CA.

185. 2004 (December). Cumulative occupational asbestos exposures of U.S. brake repair mechanics. Society for Risk Analysis 2004 Conference, December 5-8, 2004. Palm springs, CA.

186. 2004 (December). Session rappatour for one day meeting on European Exposure Assessment Toolbox. Society for Risk Analysis (SRA) 2—4 Conference, December 5- 8, 2004. Palm Springs, CA.

187. 2004 (December). An evaluation of the Toxic Equivalency Factor (TEF) for 2,3,4,7,8-PCDF (4-PCDF) using data from the recent NTP dioxin bioassays. Society for Risk Analysis 2004 Conference, December 5-8, 2004. Palm springs, CA.

188. 2004 (October). A review of recent surface dust sampling methodologies and their usefulness for exposure assessment of residences. International Society of Exposure Analysis 2004 Conference, October 17-21, 2004. Philadelphia, PA.

189. 2004 (October). Analysis of endogenous and exogenous sources of acetone exposure in children. International Society of Exposure Analysis 2004 Conference, October 17-21, 2004. Philadelphia, PA.

190. 2004 (October). Chrysotile asbestos exposure associated with removal of automobile exhaust systems (Circa 1950-1974): Preliminary Findings of a Simulation Study. International Society of Exposure Analysis 2004 Conference, October 17-21, 2004. Philadelphia, PA.

191. 2004 (October). Occupational exposure to airborne asbestos during installation and removal of asbestos-containing gaskets and packings: A Review and interpretation of published and unpublished studies. International Society of Exposure Analysis 2004 Conference, October 17-21, 2004. Philadelphia, PA.

192. 2004 (October). An evaluation of historical exposure to airborne asbestos by bystanders and workers during the installation and removal of gaskets and packing (1982-1991). International Society of Exposure Analysis 2004 Conference, October 17-21, 2004. Philadelphia, PA.

193. 2004 (September). An approach to calculating childhood body burdens of dioxin using age-dependent half-lives. International Dioxin Meeting. September 6-10, 2004. Berlin, Germany.

194. 2004 (September). Surface dust criteria for dioxin and dioxin-like compounds for re-entry to buildings. International Dioxin Meeting. September 6-10, 2004. Berlin, Germany.

195. 2004 (July). 32nd Annual IADC Trial Academy. Featured expert witness. July 30- August 7, 2004. Stanford Law School. Palo Alto, California.

196. 2004 (June). Reflections on twenty years in risk assessment: Lessons learned and how they relate to incorporating hormesis or non-linearity into the risk assessment paradigm. Presentation at the University of Massachusetts Conference on Non-Linearity, June 8, 2004. Amherst, MA.

197. 2004 (June). Our future is tied to updating exposure limits. Presentation at American Industrial Hygiene Association, Northern California Section meeting, June 2, 2004. Oakland, CA.

198. 2004 (May). Converting exposure to absorbed dose: An evolving field. Lecture in graduate course at Stanford University, May 26, 2004. Palo Alto, CA.

199. 2004 (March). Occupational exposure to airborne chrysotile asbestos during use and removal of mastics, coating, and adhesives (circa 1940's-present day) (with F. Mowat, and M. Bono). Presentation at the Society of Toxicology's 43rd Annual Meeting, March 22-25, 2004. Baltimore, MD.

200. 2004 (March). Reentry criteria for dioxin and dioxin-like compounds for building surfaces (with J. Greene and G. Brorby). Presentation at the Society of Toxicology's 43rd Annual Meeting, March 22-25, 2004. Baltimore, MD.

201. 2004 (March). Consideration relevant to constructing a human PBPK model for Perfluorooactanoic Acid (PFOA) (with G. Jepson). Presentation at the Society of Toxicology's 43rd Annual Meeting, March 22-25, 2004. Baltimore, MD.

202. 2004 (March). Dose reconstruction and exposure simulation studies: Their role in litigation. Presentation at the Defense Research Institute's Toxic Tort and Environmental Law Seminar, March 18-19, 2004. New Orleans, LA.

203. 2004 (January). Dose reconstruction and dose simulation: its current and future role in forensic toxicology and epidemiology. Lecture at University of California at Irvine, January 27, 2004. Irvine, CA.

204. 2004 (January). Dose reconstruction and dose simulation: its current and future role in forensic toxicology and epidemiology. Presentation at University of Michigan School of Public Health, January 16, 2004. Ann Arbor, MI.

205. 2003 (December). Environmental and occupational health hazards associated with the presence of asbestos in brake linings and pads (1900 to present) (with G. Brorby and B. Finley). Presentation at the Society for Risk Analysis Annual Meeting, December 7-10, 2003. Baltimore, MD.

206. 2003 (December). Residential exposures to elemental mercury due to releases from the removal of gas pressure regulators (with P. Williams, G. Brorby and P. Sheehan). Presentation at the Society for Risk Analysis Annual Meeting, December 7-10, 2003. Baltimore, MD.

207. 2003 (December). Evaluating occupational exposures to benzene at an industrial chemical plant (with P. Williams). Presentation at the Society for Risk Analysis Annual Meeting, December 7-10, 2003. Baltimore, MD.

208. 2003 (December). An evaluation of historical exposures of mechanics to asbestos from brake repair (with R. Richter and P. Sheehan). Presentation at the Society for Risk Analysis Annual Meeting, December 7-10, 2003. Baltimore, MD.

209. 2003 (September). The evolution of dose reconstruction analyses over time (with P. Williams). Presentation at the International Society of Exposure Analysis Annual Conference, September 21-25, 2003. Stresa, Italy.

210. 2003 (September). Elemental mercury releases associated with the removal of gas pressure regulators in homes (with P. Williams, G. Brorby, and P. Sheehan). Presentation at the International Society of Exposure Analysis Annual Conference, September 21-25, 2003. Stresa, Italy.

211. 2003 (September). Characterizing benzene air concentrations at an industrial chemical plant (with P. Williams). Presentation at the International Society of Exposure Analysis Annual Conference, September 21-25, 2003. Stresa, Italy.

212. 2003 (September). How the industrial hygiene profession can update the Occupational Exposure Limits. Invited speaker. Professional Conference on Industrial Hygiene, September 14, 2003. Palm springs, CA.

213. 2003 (August). Exposure of infants and children in the U.S. to the flame retardant decabromodiphenyl oxide (DBDPO). Presentation at Dioxin 2003, 23rd International Symposium on Halogenated Organic & Persistent Organic Pollutants, August 24-29, 2003. Boston, Massachusetts.

214. 2003 (March). Refining the PBPK model for chromium (VI) in humans (with S. Hays and D. Proctor). Presentation at the Society of Toxicology 42nd Annual Meeting, March 9-13, 2003. Salt Lake City, Utah.

215. 2003 (March). Evaluating the threat of chemical and biological warfare via mail or drinking water delivery systems (with B. Murphy). Presentation at the Society of Toxicology 42nd Annual Meeting, March 9-13, 2003. Salt Lake City, Utah.

216. 2003 (March). Assessing the hazard to children of low-level environmental exposures. Presentation at the Society of Toxicology 42nd Annual Meeting, March 9-13, 2003. Salt Lake City, Utah.

217. 2003 (March). Estimated children's exposure to decabromodiphenyl oxide in the U.S. Presentation at the Society of Toxicology 42nd Annual Meeting, March 9- 13, 2003. Salt Lake City, Utah.

218. 2002 (December). Historical overview of dose reconstruction analysis and general methodology. Presentation at the Society for Risk Analysis Annual Conference, December 9-11, 2002. New Orleans, Louisiana.

219. 2002 (December). Overview of children's exposure to environmental toxicants (with P.R.D. Williams). Presentation at the Society for Risk Analysis Annual Conference, December 9-11, 2002. New Orleans, Louisiana.

220. 2002 (December). Benzene Exposure Reconstruction for the Pliofilm Cohort (1936-1976) Using Monte Carlo Techniques (with P.R.D. Williams). Presentation at the Society for Risk Analysis Annual Conference, December 9- 11, 2002. New Orleans, Louisiana.

221. 2002 (October). Conducted short course at the International Conference on Contaminated Soils, Sediments and Water entitled "Recent Advances in the Practice of Risk Assessment" (with A. Madl and R. DeMott). October 21-24, 2002. Amherst, MA.

222. 2002 (September). Dose Reconstruction in the Occupational Setting (with P. Williams and A. Madl). 17th Annual Professional Conference on Industrial Hygiene (PCIH). Thriving in Unpredictable Times. September 28-October 1, 2002. Cincinnati, OH.

223. 2002 (August). Estimating historical benzene exposures (1936-1976) among rubber hydrochloride (Pliofilm) workers using probabilistic modeling techniques. (with P.R.D. Williams and J. Warmerdam) International Society of Exposure Analysis Annual Conference, August 12-15, 2002. Vancouver, British Columbia.

224. 2002 (June). Evaluation of elemental mercury releases associated with the removal of gas pressure regulators in homes. (with P.R.D Williams, P. Sheehan, and G. Brorby) American Industrial Hygiene Association Conference & Expo, June 3-6, 2002. San Diego, CA.

225. 2002 (June). Probabilistic exposure assessment of benzene for rubber hydrochloride (Pliofilm) workers (1936-1976). (With P. Williams) American Industrial Hygiene Association Conference & Expo, June 3-6, 2002. San Diego, CA.

226. 2002 (May). Applying Risk Assessment Principles to Chemical and Biological Terrorism: An Overview Plus Two Case Studies. NorCal SRA (Society of Risk Assessment) and NorCal SOT (Society of Toxicology). Chemical and biological terrorism: the role of risk assessment. May 22, 2002. Berkeley CA.

227. 2002 (March). Lung cancer mortality among workers exposed to airborne hexavalent chromium. (With D.M. Proctor). 41st Annual Meeting of Society of Toxicology, March 17-21, 2002. Nashville, TN.

228. 2002 (March). Dose-response assessment for lung cancer mortality of an occupational cohort exposed to airborne hexavalent chromium. (With D. M. Proctor). 41st Annual Meeting of Society of Toxicology, March 17-21, 2002. Nashville, TN.

229. 2002 (March). Lung cancer mortality among workers exposed to airborne hexavalent chromium (with D. M. Proctor). 41st Annual Meeting of Society of Toxicology, March 17-21, 2002. Nashville, TN.

230. 2002 (March). Is mercury in urine indicative of exposure to low levels of mercury vapor? (With J.S. Tsuji, P. Williams, M. Edwards and K.P. Avadhanam). 41st Annual Meeting of Society of Toxicology, March 17-21, 2002. Nashville, TN.

231. 2002 (March). Dose reconstruction of benzene exposure for pliofilm cohort (1936-1976) using Monte Carlo techniques. (With P. Williams). 41st Annual Meeting of Society of Toxicology, March 17-21, 2002. Nashville, TN.

232. 2002 (March). Rate of hexavalent chromium reduction by human gastric fluid. (With D. M. Proctor). 41st Annual Meeting of Society of Toxicology, March 17-21, 2002. Nashville, TN.

233. 2002 (March). Estimation of the oral bioaccessibility of Dioxins/Furans in weathered soil. US-Vietnam Scientific Conference on Human Health and Environmental Effects of Agent Orange/Dioxin, March 3-6, 2002. Ha Noi, Vietnam.

234. 2002 (March). Temporal trends in human TCDD body burden. Vietnam Scientific Conference on Human Health and Environmental Effects of Agent Orange/Dioxin, March 3-6, 2002. Ha Noi, Vietnam.

235. 2002 (March). A Proposed Reference Dose (Rfd) For Dioxin Of 5-10 Pg/Kg-Day: A Weight of Evidence Evaluation of the Human Studies. US-Vietnam Scientific Conference on Human Health and Environmental Effects of Agent Orange/Dioxin, March 3-6, 2002. Ha Noi, Vietnam.

236. 2001 (November). Presented 3-hour guest lecture at Emery University to a class on Environmental Science and Risk Assessment. Atlanta, GA.

237. 2001 (September). Reducing PCDD/PCDF formation and emission from a hazardous waste combustion facility-technological identification, implementation, and achievement. (With B. Sun). Dioxin 2001, September 9- 14, 2001. Kyongju, Korea.

238. 2001 (September). In vitro bioaccessibility study of low concentrations (50-350 ppt TEQ) of dioxin/furans in weathered soils (with K. Fehling and M.V. Ruby). Dioxin 2001, September 9-14, 2001. Kyongju, Korea.

239. 2001 (September). The United States USEPA Science Advisory Board report (2001) on the USEPA dioxin reassessment. Dioxin 2001, September 9-14, 2001. Kyongju, Korea.

240. 2001 (March). A probabilistic assessment of household exposures to MTBE from drinking water (with P. Williams and P. Sheehan). 40th Annual Meeting of Society of Toxicology, March 25-29, 2001. San Francisco, CA.

241. 2001 (March). Event by event probabilistic methodology for assessing health risks of persistent chemicals in fish: a case study. 40th Annual Meeting of Society of Toxicology, March 25-29, 2001. San Francisco, CA.

242. 2001 (March). Is hexavalent chromium carcinogenic via the oral route of exposure an evaluation of the state of the science and implications for drinking water regulations (with D.M. Proctor). 40th Annual Meeting of Society of Toxicology, March 25-29, 2001. San Francisco, CA.

243. 2001 (March). An alternative approach for toxicity testing of genetically modified foods (with J. Greene, P. Williams, and K. Avadhanam). 40th Annual Meeting of Society of Toxicology, March 25-29, 2001. San Francisco, CA.

244. 2001 (March). Contribution of individual truck operations to ambient diesel particulate matter (DPM) concentrations: implications for risk assessment and management (with A.K. Madl and B. Finley). 40th Annual Meeting of Society of Toxicology, March 25- 29, 2001. San Francisco, CA.

245. 2001 (March). Identification of a Proposition 65 no significant risk level for coal tar. 40th Annual Meeting of Society of Toxicology, March 25-29, 2001. San Francisco, CA.

246. 2000 (December). A case study of MTBE exposures and risk in California (with P.R.D. Williams). Platform Presentation at the 16th Annual Society of Risk Analysis Conference, December 3-6, 2000. Arlington, VA.

247. 2000 (October). MTBE ambient air and drinking water exposures in California (with P.R.D. Williams, P. Sheehan, and J. Warmerdam). 10th Annual International Society of Exposure Analysis, October 24-27, 2000. Monterey, CA.

248. 2000 (October). MTBE in California drinking water: A probabilistic assessment of exposures (with P. Sheehan and P. Williams). 10th Annual International Society of Exposure Analysis, October 24-27, 2000. Monterey, CA.

249. 2000 (October). Assessing risks to nursing infants whose mothers ingested fish containing persistent organic pollutants (POPs): Inadequacy of USEPA default methods and directions for the future (with N.D. Wilson, V.A. Craven, S.M. Hays, B.E. Finley, and B.D. Kerger). 10th Annual International Society of Exposure Analysis, October 24-27, 2002. Monterey, CA.

250. 2000 (August). Is dioxin a threshold carcinogen? A quantitative analysis of the epidemiological data using internal dose and Monte Carlo Methods (with C.R. Kirman, L.A. Aylward, N.J. Karch, B.L. Finley, and S.M. Hays). Dioxin 2000: 20th International Symposium on Halogenated Environmental Organic Pollutants and POPs. August 13-17, 2000. Monterey, CA.

251. 2000 (August). International concern over persistent organic pollutants (POPs) in foods: Risk assessment and public policy (with K. Connor). Dioxin 2000: 20th International Symposium on Halogenated Environmental Organic Pollutants and POPs, August 13-17, 2000. Monterey, CA.

252. 2000 (August). The formation of hepta- and octa-dioxins in feces of cows fed pentachlorophenol treated wood (with G. Fries, W. Luksemburg, M. Lorber, and J. Ferrario). 20th International Symposium on Halogenated Environmental Organic Pollutants and POPs, August 13-17, 2000. Monterey, CA.

253. 2000 (May). Alveolar-deposited airborne particles of beryllium as a predictor of the prevalence of disease in a beryllium processing facility. Platform presentation at the American Industrial Health Conference & Exposition (with M. Kent, T. Robins, A.K. Madl, and M. Goodman), May 22-26, 2000, Orlando, FL.

254. 2000 (May). Biomonitoring for beryllium in workers of a beryllium processing facility. Platform presentation at the American Industrial Health Conference & Exposition (with Y. Lowney, D. Deubner, S.M. Hays, P. Chapman, B. Kerger, and W. Shields), May 22-26, 2000, Orlando, FL.

255. 2000 (May). Chronic beryllium disease and beryllium sensitization at a beryllium mine and extraction facility. Platform presentation at the American Industrial Health Conference & Exposition (with M. Kelsh, D. Deubner, L. Maier, M Kent, B. Smith, P. Chapman, K. Zhao, and M. Kolanz), May 22-26, 2000, Orlando, FL.

256. 2000 (May). The role of extrapulmonary exposure pathways in the prevalence of chronic beryllium disease and beryllium sensitization. Platform presentation at the American Industrial Health Conference & Exposition (with D. Deubner, M. Kelsh, Y. Lowney), May 22-26, 2000, Orlando, FL.

257. 2000 (February). Using PBPK modeling in the regulation of toxic compounds. Presentation at the Toxicokinetic Modeling Symposium (with S. Hays), February 17- 18, 2000, The Hague, the Netherlands.

258. 2000 (March). Consideration of alternate exposure pathways in the possible relation to prevalence of chronic beryllium disease. Presentation at the Society of Toxicology Annual Meeting (with D. Deubner, M. Kelsh, Y. Lowney, and M. Kolanz), March 19- 23, 2000, Philadelphia, PA.

259. 2000 (March). The persistent, bioaccumulative toxics (PBT) project: risk-based intentions—toxicological limitations. Presentation at the Society of Toxicology Annual Meeting (with H. Estreicher and K.T. Connor), March 19-23, 2000, Philadelphia, PA.

260. 1999 (September). Fingerprinting analysis of PCDD/PCDF sources in a surface water outfall near a petroleum refinery. Paper presented at the 19th International Symposium on Halogenated Environmental Organic Pollutants and POPs (with M.L. Moore, M.K. Butcher, K. Connor, and D.B. Mathur), Venice, Italy.

261. 1999 (September). Risk assessment in industrial hygiene. Presented to Symposia in Risk Assessment Methodology for the Practicing Industrial Hygienist, New Orleans, LA.

262. 1999 (September). Joint ACGIH and Brush Wellman Symposia on "Understanding the Risks and Research Involving Beryllium." Washington, DC.

263. 1999 (March). Comparison of acute inhalation exposure levels for chemical irritants among six agencies in the United States. Paper presented at the 38th Annual Meeting of the Society of Toxicology (with A.K. Madl and R.M. Kalmes), New Orleans, LA.

264. 1999 (March). Evolution of safety evaluation in the 20th century. Paper presented at the 38th Annual Meeting of the Society of Toxicology (with D.G. Dodge, P.J. Sheehan, and T.C. Bernhardt), New Orleans, LA.

265. 1998 (December). Assessing the ecological risks of dam removal. Paper presented at the Meeting of the Society for Risk Analysis (with P.J. Sheehan, R.J. Wenning, B.D. Wright, N.M. King, and D.M. Becklin), Phoenix, AZ.

266. 1998 (December). Background risks associated with exposure to n-nitrosodimethyamine (NDMA): is something amiss in the regulation of this chemical? Paper presented at the Meeting of the Society for Risk Analysis (with P.J. Sheehan, D. Mathur, and D. Dodge), Phoenix, AZ.

267. 1998 (December). Background risks from dietary exposure to di(2- ethylhexyl) phthalate (DEHP): implications of regulations in the context of California's Proposition 65. Paper presented at the Meeting of the Society for Risk Analysis (with D.B. Mathur and D.G. Dodge), Phoenix, AZ.

268. 1998 (December). Is TCDD a threshold carcinogen? A quantitative analysis of the epidemiological data. Paper presented at the Meeting of the Society for Risk Analysis (with C.R. Kirman, S.M. Hays, L. Aylward, and N.J. Karch), Phoenix, AZ.

269. 1998 (December). Significant upcoming changes in the application and practice of risk assessment. Paper presented at the Meeting of the Society for Risk Analysis (with D.G. Dodge), Phoenix, AZ.

270. 1998 (May). Future trends in the practice of health risk assessment. Keynote Speaker at Southern California Society for Risk Analysis Annual Meeting, UCLA, Los Angeles, CA.

271. 1998 (March). Comparison of measured and model-estimated indoor concentrations of airborne chloroform from use of residential tap water. Paper presented at the 37th Annual Meeting of the Society of Toxicology (with R.O. Richter, D. Suder, G.E. Corbett, T.P. Flahive, and B.D. Kerger), Seattle, WA.

272. 1998 (March). Measurements of airborne concentrations of halogenated methanes due to showering and bathing. Paper presented at the 37th Annual Meeting of the Society of Toxicology (with B.D. Kerger, C.E. Schmidt, T.P. Flahive, G.E. Corbett, and R.O. Richter), Seattle, WA.

273. 1998 (March). The ever-changing environmental responsibilities of the golf course industry. Keynote speaker at annual meeting of the Ohio Professional Golf Course Owners Association, Columbus, OH.

274. 1998 (March). Update on methods to derive ambient air limits. Paper presented at the 37th Annual Mtg. of the Society of Toxicology (with J.E. Ryer-Powder and K. Gaynor), Seattle, WA.

275. 1998 (February). A methodology for evaluating the human health hazard posed by ingesting hexavalent chromium in drinking water. Invited lecturer at Harvard University School of Public Health, Boston, MA.

276. 1998 (February). Series of lectures on Hum. Ecol. Risk Assess. Invited lecturer for 4 days at the University of Kuwait: Sponsored by Kuwait Foundation for the Advancement of Science. Kuwait City, Kuwait.

277. 1998 (February). Using the risk assessment methodology to tackle a unique environmental issue: hexavalent chromium in groundwater. Invited lecturer at Yale University School of Public Health, New Haven, CT.

278. 1998 (January). How industrial hygienists can change the national risk priorities embraced by regulatory agencies. Invited lecturer as part of the University of Michigan School of Public Health "Experts in the Field" series: How Industrial Hygienists Can Change the National Risk Priorities Embraced by Regulatory Agencies. Ann Arbor, MI.

279. 1997 (December). Critical review and reassessment of USEPA's risk- based cleanup level for dioxin and relocation plan for the Escambia Superfund site in Pensacola, Florida. Paper presented at the Annual Meeting and Exposition of the Society of Risk Analysis (with T.C. Bernhardt, D.B. Mathur, G.R. Vishwanath, and R.J. Wenning), Washington, DC.

280. 1997 (December). Human ingestion of chromium (VI) in drinking water: Pharmacokinetics following repeated exposure. Paper presented at the Annual Meeting and Exposition of the Society of Risk Analysis (with B.L. Finley, D.B., Kerger, M.W. Katanoa, M.L. Gargas, and G.C. Corbett), Washington, DC.

281. 1997 (December). The effect of cooking practices on levels of DDT and PCB compounds in the edible tissue of fish. Paper presented at the Annual Meeting and Exposition of the Society of Risk Analysis (with N.D. Wilson and N.M. Shear), Washington, DC.

282. 1997 (November). Invited speaker to the 2nd Workshop on Guidance for Environmental Risk Assessment and Management of Great Britain (Institute for Environment and Health), Royal College of Physicians, London.

283. 1997 (November). Participant in discussion on "Dust is More than Dirt." 7th Annual Meeting of the International Society of Exposure Analysis, Research Triangle Park, NC.

284. 1997 (November). The historical practice of risk assessment and how the field will evolve over the next 10 years. Featured speaker at the annual Lone Star Section Meeting of the Society of Risk Assessment, San Antonio, TX.

285. 1997 (October). The practice of health risk assessment; 1975-1995 and 1995- 2005. Experts Guest Lecture Series of the Graduate Program at the Stanford University Civil/Environmental Engineering Department, Palo Alto, CA.

286. 1997 (August). Comparing the predicted uptake of TCDD based on exposure calculations with the actual uptake: a case study of residents of Times Beach. Paper presented at the 17th International Symposium on Chlorinated Dioxins and Related Compounds (with S. Hays, S. El-Sururi, P. Underwood, and R. Ku), Indianapolis, IN.

287. 1997 (August). Relative doses of 2,3,7,8-TCDD using alternative dose metrics: comparison of the NIOSH and ranch hand populations. Paper presented at the 17th International Symposium on Chlorinated Dioxins and Related Compounds (with L. Aylward, S. Hays, J. Czernec, B. Brien, and N. Karch), Indianapolis, IN.

288. 1997 (April). Lecture in the "Topics in Risk Assessment" course at Stanford University, directed by Dr. Warner North, Alto, CA.

289. 1997 (April). Scientific symposium on proper dose metrics for assessing the risks due to persistent chemicals like dioxin, furans, PCB, and DDT. Sponsored by International Life Science Institute, Resources for the Future, and the American Industrial Health Council 1997, Washington, DC.

290. 1997 (March). Desert Storm: Environmental contamination, litigation strategies and bankruptcy considerations. Paper presented at Toxic/Hazardous Substances and Environmental Law Committee Symposium, ABA Tort and Insurance Practice Section, Inserting Real Science into Mass Toxic Tort Actions (with B.D. Kerger and J.V. Dagdigian).

291. 1997 (March). Risk assessment and its role in remedy selection. Invited paper at the Information Network for Superfund Settlements (a group of 100 Superfund Attorneys), Denver, CO.

292. 1997 (March). Risk assessment isn't dead… it is just undergoing a rebirth: implications to the Portland Cement Association. Presented to the Portland Cement Association, Chicago, IL.

293. 1997 (January). The role of risk assessment in environmental decision making in the U.S. and in Europe in the year 2000. Third Annual International Symposia on US- EURO Environmental Management, Brussels, Belgium.

294. 1996 (August). PCDD/PCDFs in urban stormwater discharged to San Francisco Bay, California USA. Paper presented at the 16th International Symposium on Chlorinated Dioxins and Related Compounds (with D. Mathur), Amsterdam, The Netherlands.

295. 1996 (August). Transport of chlorinated dioxin and furan contaminants in pentachlorophenol-treated wood to milk and adipose tissue of dairy cattle. Paper presented at the 16th International Symposium on Chlorinated Dioxins and Related Compounds (with Drs. Mathur and Fries), Amsterdam, The Netherlands.

296. 1996 (April). The role of risk assessment in Brownfield initiatives. Invited Paper at the International Conference on Industrial Site Recycling, Pittsburgh, PA.

297. 1996 (April). Urinary chromium as a biological marker of environmental exposure: what are the limitations? Invited Paper at the 30th Industrial Health Foundation's Annual Chromium Symposium, Washington, DC.

298. 1996 (March). An analysis of inter-individual variability in uptake and elimination of chromium from human volunteers. Annual Mtg. of the Society of Toxicology, Anaheim, CA.

299. 1996 (March). A physiologically-based pharmacokinetics model for ingestion of chromium (III and VI) in drinking water: validation with human studies. 35th Annual Meeting of the Society of Toxicology, Anaheim, CA.

300. 1996 (March). Assessment of airborne hexavalent chromium in the home following use of contaminated tap water. 35th Annual Meeting of the Society of Toxicology, Anaheim, CA.

301. 1996 (March). Dermal uptake of hexavalent chromium in human volunteers measures of systemic uptake from immersion in water at 22 ppm. 35th Annual Meeting of the Society of Toxicology, Anaheim, CA.

302. 1996 (March). Dioxin - health risk assessment and analytical methodologies. Invited lecture to the San Francisco Bay Regional Water Quality Control Board, San Francisco, CA.

303. 1996 (March). Monte Carlo techniques and risk assessment. Four-hour professional development short course. 30th Annual Meeting of the Society of Toxicology, San Diego, CA.

304. 1996 (March). The chromium (VI) reductive capacity of household beverages: implications for risk assessment. 35th Annual Meeting of the Society of Toxicology, Anaheim, CA.

305. 1996 (March). Toxicologic and environmental hazards posed by CR(VI) in the environment. Chair of a 1-day national symposium. Sponsored by the University of Massachusetts. Newport Beach, CA.

306. 1996 (March). Pharmacokinetics of drinking water exposure to select chromium (III and VI) compounds in human volunteers. 35th Annual Meeting of the Society of Toxicology, Anaheim, CA.

307. 1996 (March). Reduction kinetics of hexavalent chromium in human blood. 35th Annual Meeting of the Society of Toxicology, Anaheim, CA.

308. 1996 (March). Testing for DNA-protein cross linking after drinking water exposure to chromium (III and VI) in human volunteers. 35th Annual Meeting of the Society of Toxicology, Anaheim, CA.

309. 1996 (February-March). The critical role of house dust in understanding the hazards posed by contaminated soils. Invited paper at the First National Conference on Exposure and Risk Assessment, Munich, Germany.

310. 1996 (February). Lessons learned in the United States about environmental regulations and risk assessment (1975-1995). Risk, Regulation, and Reason Conference (sponsored by the Santa Clara County Manufacturing Association), Palo Alto, CA.

311. 1996 (January). Risk assessment aspects of Proposition 65. California Bar Association, Sacramento, CA.

312. 1995 (October). The cost of identifying and evaluating airborne toxicants. Invited paper presented at the 1st East Asian Regional Air Toxics Conference (with R. Wenning), Hong Kong.

313. 1995 (September). Lessons learned in the United States about environmental regulations and risk assessment (1975-1995). Annual Meeting of the Business Council of Australia, Sydney, Australia.

314. 1995 (September). The Australian challenge regarding chlorinated chemicals: establishing a plan based on the U.S. experience. Australian/New Zealand Chlorine Work Group and PACIA, Melbourne, Victoria, Australia.

315. 1995 (August). Human vs. animal sensitivity to the immunological effects of TCDD: a preliminary comparison. 15th International Symposium on Chlorinated Dioxins and Related Compounds, Edmonton, Alberta, Canada.

316. 1995 (August). The relative susceptibility of animals and humans to the carcinogenic hazard posed by exposure to 2,3,7,8-TCDD: an evaluation of different dosimetric measures. 15th International Symposium on Chlorinated Dioxins and Related Compounds, Edmonton, Alberta, Canada.

317. 1995 (August). Validating dermal exposure assessment techniques for dioxin using body burden data and pharmacokinetic modeling. 15th International Symposium on Chlorinated Dioxins and Related Compounds, Edmonton, Alberta, Canada.

318. 1995 (May and June). Symposia on evaluation of the human health components of the life cycle assessment. Expert panel member. Sponsored by the International Life Sciences Institute, Washington, DC.

319. 1995 (May). Presented two invited lectures as part of a "Visiting Experts in Risk Assessment" series directed by Dr. Steve Hrudy. University of Calgary and University of Alberta at Edmonton.

320. 1995 (March). Chairman and key presenter at professional development course on risk assessment, 30th Annual Meeting of the Society of Toxicology, Baltimore, MD.

321. 1995 (March). Fundamentals of health risk assessment. Professional development short-course. 30th Annual Meeting of the Society of Toxicology, Baltimore, MD.

322. 1995 (March). New methods for assessing the risks of sites with contaminated soil. USEPA of Switzerland and the Kanton of Zurich, Zurich, Switzerland.

323. 1995 (February). Methods for improving risk assessments of sites contaminated with chemical carcinogens. 1st Australian Symposia on Carcinogens in the Environment, Melbourne, Australia.

324. 1995 (February). Risk assessment techniques for airborne toxics. Symposia sponsored by The Australian Clean Air Society, Melbourne, Australia.

325. 1995 (February). State-of-the-art exposure techniques: the use of Monte Carlo techniques. 1st Australian Symposia on Carcinogens in the Environment, Melbourne, Australia.

326. 1995 (January). Session chair (with Dr. Roger McClellan) on risk assessment methods at the 1st International Symposia on Butadiene and Isoprene, Seattle, WA.

327. 1994 (November). Presented a 4-hour short-course on Hum. Ecol. Risk Assess. Royal Academy of Engineering of South Australia, Adelaide, Australia.

328. 1994 (October). The proper role of epidemiology in regulatory risk assessment. Invited symposia participant. Harvard School of Public Health, Boston, MA.

329. 1994 (May). How realistic are current risk assessment procedures and criteria? Presentation at the Australian Waste Management Conference, Melbourne, Australia.

330. 1994 (May). Presented a 2-hour short-course on ecological risk assessment. New South Wales Environmental Protection Agency, Sydney, Australia.

331. 1994 (May). Presented an 8-hour course on advanced topics in health risk assessment at the Clean Air Society of Australia Meeting, Melbourne, Australia.

332. 1994 (February). Presented a lecture on ecotoxicology and risk assessment at the PPG Annual Environmental Conference, Lake Charles, LA.

333. 1994 (January). A comparison of generic human health and ecological risk-based soil remediation criteria in Canada and the United States. Paper presented at the 4th Annual ASTM Symposia on Environmental Toxicology and Risk Assessment (with B.L. Finley, M.J. Kangas, and P. Scott), Montreal, Canada.

334. 1994 (January). New methods for assessing the risks of airborne toxicants. Paper presented at the CIRCA/AWMA-OS Joint International Conference on Atmospheric Chemistry (with M. Gray and C. Curry), Toronto, Canada.

335. 1993 (October). Keynote speaker at the 1st U.S. Environmental Protection Agency Workshop on Physiologically Based Pharmacokinetics (PBPK) and Cancer Risk Assessment, North Carolina.

336. 1993 (October). Keynote speaker and invited U.S. representative to the 1st International Conference on Ecotoxicology, Melbourne, Australia.

337. 1993 (October). State-of-the-art in risk assessment. Invited speaker. Professional Conference on Industrial Hygiene, Cincinnati, OH.

338. 1993 (October). The historical use of risk assessment in the United States and how this experience can be utilized within the international arena. Presentation at the 1st International Environmental Risk Assessment Conference for Australia and New Zealand, Melbourne, Australia.

339. 1993 (September). Participated in a panel discussion on the pharmacokinetics of the dioxins at the 13th Annual International Dioxin Meeting, Vienna, Austria.

340. 1993 (August). Presented a lecture on setting a reference concentration for hexavalent and trivalent chromium at the USEPA Reference Dose Task Group meeting, Cincinnati, OH.

341. 1993 (March). An alternative to the USEPA's proposed inhalation reference concentrations for hexavalent chromium. Paper presented at the 32nd Annual Meeting of the Society of Toxicology (with B.L. Finley and D.M. Proctor), New Orleans, LA.

342. 1993 (March). Approaches to assessing human exposure to soil contaminants at wood preserving facilities. Paper presented at the 32nd Annual Meeting of the Society of Toxicology (with M.L. Gargas, P.K. Scott, and B.L. Finley), New Orleans, LA.

343. 1993 (March). Fundamentals of health risk assessment. Professional development course (4 hours). 32nd Annual Meeting of the Society of Toxicology, New Orleans, LA.

344. 1993 (March). Presented two, 1-hour professional development courses on recent improvements in exposure assessment at the 32nd Annual Meeting of the Society of Toxicology, New Orleans, LA.

345. 1993 (February). Health risk assessment and the practice of engineering. Presentation at the UCLA School of Public Health and Engineering, Los Angeles, CA.

346. 1993 (February). Using human patch-test data to establish health-protective concentrations of allergens in soils. Paper presented at the International Conference on Dermatology (with Drs. Finley and Nethercott), Honolulu, Hawaii.

347. 1993 (February). Understanding the hazard posed by xenobiotics in Passaic River sediments. Presentation at the SETAC Symposium on The Hazards posed by Dioxin in the Passaic River, Rutgers University, Piscataway, NJ.

348. 1992 (December). Historical reconstruction of benzene exposure for an occupational cohort of rubber workers. Paper presented at the Annual Meeting of the Society of Risk Analysis (with P.S. Price), San Diego, CA.

349. 1992 (December). Comparing the results of a Monte Carlo analysis with USEPA's reasonable maximum exposed individual (RMEI): a case study of a former wood treatment site. Paper presented at the Annual Meeting of the Society of Risk Analysis (with T.L. Copeland, M.A. Harris, and V. Lau), San Diego, CA.

350. 1992 (November). How to prioritize the national environmental agenda using risk assessment. Invited participant at a meeting of experts. Resources for the Future, Annapolis, MD.

351. 1992 (October). How to set a TLV for an irritant. Presentation at the Quarterly Meeting of the Northeast Section of the AIHA, Hartford, CT.

352. 1992 (October). The U.S. should adopt a risk-based environmental agenda. Presentation at the President's Forum at the Annual Meeting of the American Paper and Pulp Institute, Syracuse University, NY.

353. 1992 (August). A Monte Carlo analysis of exposures and cancer risks from 2,3,7,8-TCDD in soil at residential and industrial sites. Paper (Abstract #RSK-29) presented at the 12th International Dioxin meeting (with R. Wenning and V. Lau), Tampere, Finland.

354. 1992 (August). Chemometric analysis of potential sources of PCDD/PCDD in surficial sediments from Newark Bay. Paper (Abstract #13520) presented at the 12th International Dioxin Meeting (with R.J. Wenning), Tampere, Finland.

355. 1992 (August). Chemometric comparisons of PCDD and PCDF fingerprint patterns in environmental sources and marine and freshwater sediments. Paper (Abstract #ANA-9) presented at the 12th International Dioxin Meeting (with R.J. Wenning), Tampere, Finland.

356. 1992 (June). The rationale for the proposed TLV for formaldehyde. Presentation at the Annual American Industrial Hygiene Conference, Boston, MA.

357. 1992 (April). State-of-the-art approaches for assessing the risks of contaminated groundwater. Presentation at the Gulf Coast Hazardous Substance Research Center's Fourth Annual Symposium, Lamar University, Beaumont, TX.

358. 1992 (March). Regulatory actions with TCDD: what will happen in the 1990s? Presentation at the Fourth Annual Petroleum Contaminated Soils Conference, Newport Beach, CA.

359. 1992 (March). Use of risk assessment in setting TLVs. Presentation at the Northern California American Industrial Hygiene Association Annual Meeting, San Francisco, CA.

360. 1992 (February). Ecological risk assessment guidelines. Invited participant at the National Academy of Science's workshop, Fairfax, VA.

361. 1992 (February). Improving risk assessment through the use of Monte-Carlo analyses. Presentation given at the National Academy of Sciences', CRAM Committee, State- of-the-Art Approaches to Exposure Assessment, Washington, DC.

362. 1992 (February). State-of-the-art approaches to exposure assessment. Invited speaker and participant at the National Academy of Science's Symposia, held as part of USEPA's Committee on Risk Assessment Methods, Washington, DC.

363. 1992 (January). Environmental exposure assessment. Invited workshop participant. U.S. Environmental Protection Agency, Virginia Beach, VA.

364. 1991. Recent changes in regulatory toxicity rankings for wood-treating chemicals and their impact on risk assessment and remediation at wood- treating site. Paper presented at the American Wood Preservers Institute (with B. Finley and M. Harris), Scottsdale, AZ.

365. 1991 (November). A quantitative uncertainty analysis of the CAPCOA approach to risk assessment. Paper presented at the Western States Meeting of the Air and Waste Management Association (with T. Copeland), Seattle, WA.

366. 1991 (November). A state-of-the-art review of the human health hazards posed by benzene. Presentation at the First International Conference on the Health Hazards of Gasoline, Miami, FL.

367. 1991 (November). The role of risk assessment. Presentation at Problems Posed by Transferring Contaminated Properties (sponsored by Diepenback and Associates), Sacramento, CA.

368. 1991 (March). A comprehensive approach to setting soil standards. Presented at the 3rd Western States Contaminated Soil and Groundwater Conference, Newport Beach, CA.

369. 1991 (February). Future likely changes in the TLVs. University of Michigan School of Public Health, Ann Arbor, MI.

370. 1991 (February). Future trends in health risk assessment. Exxon Biomedical Research Laboratories, East Millstone, NJ.

371. 1991 (February). Risk-based remedial investigations. Presented to Phillips Petroleum Environmental Engineering Staff, Bartlesville, OK.

372. 1991 (January). The use of risk assessment to optimize the remedial investigation process. Presentation at Environment Remediation from Cradle to Grave (sponsored by Armstrong, Teasdale, Schlafly, Davis, and Dicus), St. Louis, MO.

373. 1991 (February). Future likely changes in the TLVs. University of Michigan School of Public Health, Ann Arbor, MI.

374. 1990 (December). The proper use of risk assessment to solve environmental problems. Presentation at the Northern Ohio Manufacturers Association (sponsored by the law firm of Squire, Sanders and Dempsey), Cleveland, OH.

375. 1990 (November). Practice of health risk assessment and its impact on the economy. Presentation at the Annual Meeting of American Institute of Chemical Engineers of New York State, Albany, NY.

376. 1990 (October). What does the risk assessment process tell us about the TLVs? Presentation at the 20th Professional Industrial Hygiene Conference, Vancouver, British Columbia, Canada.

377. 1990 (September). An evaluation of the inhalation hazard posed by dioxin-contaminated soil. Presentation at the 10th International Dioxin Meeting, Bayreuth, Germany.

378. 1990 (September). A re-evaluation of the tumor histopathology of Kociba et al. (1978) using 1990 criteria: implications for the risk assessment of 2,3,7,8-TCDD using the linearized multistage model. Presentation at the 10th International Dioxin Meeting, Bayreuth, Germany.

379. 1990 (September). Dioxin risk assessment of the Columbia River. Presentation at the 10th International Dioxin Meeting, Bayreuth, Germany.

380. 1990 (September). PCDDs and PCDFs in surficial sediments form the lower Passaic River and Newark Bay. Presentation at the 10th International Dioxin Meeting, Bayreuth, Germany.

381. 1990 (September). Risk assessment of 2,3,7,8-TCDD using a biologically-based cancer model: a re-evaluation of the Kociba et al. (1978) bioassay. Presentation at the 10th International Dioxin Meeting, Bayreuth, Germany.

382. 1990 (June). Using health risk assessment to set clean-up levels. Presentation at Management of Hazardous Waste Issues (sponsored by Executive Enterprises), San Francisco, CA.

383. 1990 (May). Development and field validation of a sampling and analytical method for airborne hexavalent chromium. Presentation at the Annual Meeting of the Air Pollution and Waste Management Association, Raleigh, NC.

384. 1990 (May). The assessment of health risks associated with indoor benzene vapor emitted from building foundation soil: a case study. Presentation at the Annual Meeting of the Air Pollution and Waste Management Association, Raleigh, NC.

385. 1990 (April). Criteria for using substitution as a control measure. Presentation at the 10th Annual Meeting of the Semiconductor Safety Association, Phoenix, AZ.

386. 1990 (April). Current procedures in the practice of health risk assessment. Team-taught 8-hour short course presented at the 12th Annual Hazmacon Meeting, Anaheim, CA.

387. 1990 (April). Fundamentals in risk assessment for safety engineers. Presentation at the Annual Meeting of the American Society of Safety Engineers (California Section), Long Beach, CA.

388. 1990 (April). Methodology for setting airborne emissions limits for chloroform, chlorine dioxide, and chlorine from paper and pulp mills. Presentation at the Annual Meeting of the Technical Association of the Paper and Pulp Industries, Seattle, WA.

389. 1990 (April). Risk assessments methods and compliance with AB-2588. Presentation at Santa Clara Manufacturers Association, San Jose, CA.

390. 1990 (April). Setting rational health-based water quality standards for dioxin: risk assessment for the Columbia River. Presentation at the Annual Meeting of the Technical Association of the Paper and Pulp Industries, Seattle, WA.

391. 1990 (April). The practice of health risk assessment in the 1990s. Presentation at the 4th Conference on Toxic Substances, Canadian Air and Waste Management Association, Montreal, Canada.

392. 1990 (February). Risk assessment approaches for petroleum contaminated soils. Presentation at the Annual West Coast Meeting on Petroleum Contaminated Soils, Anaheim, CA.

393. 1989 (November). Assessing the risks of environmental tobacco smoke. Presented at the International Symposia on Environmental Tobacco Smoke, Montreal, Canada.

394. 1989 (November). Conservation in the practice of health risk assessment suggestions for avoiding mistakes. Presented at the Annual Meeting of the Society of Risk Analysis, San Francisco, CA.

395. 1989 (November). The role of risk assessment in dioxin litigation. Presented at Dioxin Claims and Litigation (sponsored by Executive Enterprises, Inc.), Washington, DC.

396. 1989 (November). Setting a clean-up level for dioxin contaminated soil based on indirect routes of exposure. Invited paper presented at the symposium A Re-evaluation of the 1 ppb Standard for Dioxin in Soil, Washington, DC.

397. 1989 (September). Assessment of pesticide related hazards to birds in residential settings. Presented at the Annual Meeting of the Society of Environmental Toxicology and Chemistry, Toronto, Canada.

398. 1989 (September). Risk assessment methods for petroleum contaminated sites. Presented at the 4th Annual Petroleum Contaminated Soils Conference, Amherst, MA.

399. 1989 (August). Evolving science and risk assessment methods. Presentation at the Fifth Annual San Diego Conference on Environmental Compliance, San Diego, CA.

400. 1989 (June). Risk based clean-up. Presented at California Environmental Compliance Management (sponsored by California Business Law Institute), Los Angeles, CA.

401. 1989 (May). Case studies of less than perfect risk assessments. Presented at California Manufacturers Association (CMA), Los Angeles, CA.

402. 1989 (April and May). Risk assessment approaches for complying with California's toxic hot spots - AB 2588 Law. Presented as short courses sponsored by Latham and Watkins, San Francisco, Los Angeles, Burbank, and San Diego, CA.

403. 1989 (February). Setting clean-up levels; site, by site, by site. Presented at the Western States HAZMAT Meeting, Seattle, WA.

404. 1989 (February). Fundamentals of Hum. Ecol. Risk Assess. Professional development short course (4 hours). Annual Meeting of the Soc. of Toxicology, Atlanta, GA.

405. 1989 (February). The risk assessment of environmental and human health hazards. Presented at the Annual Meeting of the Society of Toxicology, Atlanta, GA.

406. 1988 (December). Use of risk assessment methods in negotiating clean-up standards. Presentation at the Peninsula Industrial and Business Association, San Mateo, CA.

407. 1988 (November). New approaches to assessing the risks posed by dioxin. Presented at the East Coast Section of the Society of Toxicology, Cadbury, NJ.

408. 1988 (September). The USEPA's proposed position on the health hazards posed by dioxin. Presented at the Center for Environment and Energy Management, Washington, DC.

409. 1988 (July). CAPCOA air toxics risk assessment. Presented to the Santa Clara County Manufacturing Group, Santa Clara, CA.

410. 1988 (July). How health standards for groundwater contaminants are established. Presented at the American Groundwater Trust workshop Solving Groundwater Problems, City of Industry, CA.

411. 1988 (May). Why the USEPA risk assessment guidelines need to be updated. Presentation at the Joint Meeting of the Northern California Society of Toxicology and Society of Risk Analysis, Letterman Toxicology Institute, San Francisco, CA.

412. 1988 (April). A comparative health risk assessment of land filling versus incineration. Presentation at the 1st Conference on Municipal Solid Waste Disposal, Amherst, MA

413. 1988 (April). Justification for USEPA's new position on dioxin. Paper presented at the Joint Meeting of the USEPA's Science Advisory Panel and Society of Toxicology session Assessing the Risks from Toxics in Northern California; What Science Can Tell Us, San Francisco, CA.

414. 1988 (April). Risk assessment approaches for developmental toxicants and other non-carcinogenic chemicals. Presentation at the Annual Meeting of the American Occupational Medical Association, New Orleans, LA.

415. 1988 (March). Risk assessment and Prop. 65: a challenge for industrial hygienists. Presented at the Southern California Chapter of the American Industrial Hygiene Association, Los Angeles, CA.

416. 1988 (February). A physiologically pharmacokinetics description of the tissue distribution and enzyme induction of 2,3 7,8-tetrachlorodibenzo-p-dioxin in the rat. Presented at the 27th Annual Meeting of the Society of Toxicology, Dallas, TX.

417. 1988 (February). Assessing the environmental and human health hazards of contaminated soil. Presentation at the 27th Annual Meeting of the Society of Toxicology, Dallas, TX.

418. 1988 (February). Risk assessment and Prop 65. Presented at the Annual Meeting of the Food Processors and Engineers, Santa Barbara, CA.

419. 1988 (February). Shortcomings in traditional approaches to risk assessment. Presentation at the New York City Bar Association (Environmental Law Section), New York, NY.

420. 1987 (December). Assessing the risks of your operations under Proposition 65. Short course at the California Manufacturers Association, Oakland and Anaheim, CA.

421. 1987 (December). Complying with Prop. 65 using risk assessment approaches. Presentation at the Hazardous Materials Conference, San Diego, CA.

422. 1987 (November). A methodology for calculating the level of dioxin contamination due to particulate emissions from incinerators. Presentation at the Annual Meeting of the Society of Environmental Toxicology and Chemistry, Pensacola, FL.

423. 1987 (November). A risk assessment of incinerator emissions as a potential source in foods of animal origin. Paper presented at the Annual Meeting of the Society of Risk Analysis (with G. Fries), Houston, TX.

424. 1987 (November). Risk assessment approaches for developmental and reproductive toxicants. Presentation at the Annual Meeting of the Occupational and Environmental Health Groups at Hewlett-Packard Corporation, Palo Alto, CA.

425. 1987 (October). A physiologically based pharmacokinetics model for 2,3,7,8- TCDD. Presentation at the 7th International Symposium on Dioxins and Related Compounds, Las Vegas, NV.

426. 1987 (October). Documentation of an occupational exposure limit for 2,3,7,8- TCDD. Presentation at the 7th International Symposium on Dioxins and Related Compounds, Las Vegas, NV.

427. 1987 (October). Evaluation of factors used to assess incinerator emissions as a potential source of TCDD in foods of animal origin. Presentation at the 7th International Symposium on Dioxins and Related Compounds, Las Vegas, NV.

428. 1987 (October). Oral bioavailability in the rat of soil bound TCDD. Presentation at the 7th International Symposium on Dioxins and Related Compounds, Las Vegas, NV.

429. 1987 (September). Evaluating the public health hazards of contaminated soil. Presentation at the 2nd Annual Conference on Petroleum Contaminated Soil, Amherst, MA.

430. 1987 (September). Improved risk assessment approaches for carcinogens and developmental toxins. Presentation at the Annual Meeting of the California Manufacturers Association, Santa Barbara, CA.

431. 1987 (September). Risk assessment methods for developmental and reproductive toxicants. Presentation at the USEPA Experts Workshop, Little Rock, AR.

432. 1987 (August). The fundamentals of toxicology and risk assessment. Presentation at the Silicon Valley Clean Water Alliance, San Jose, CA.

433. 1986 (November). The process of evaluating environmental health risks. Presentation at the Dioxins: From Soil to Receptor, Medical College of Virginia, Richmond, VA.

434. 1986 (October). The use of pharmacokinetics in risk assessment. Presentation at the National Academy of Science Workshop, Washington DC.

435. 1986 (September). A critical examination of assumptions used in risk assessments of dioxin contaminated soil. Paper presented at the 3rd Annual meeting of the International Society of Regulatory Toxicology and Pharmacology (with J. Murray), Washington, DC.

436. 1986 (May). Conducting an environmental risk assessment of contaminated soil. Presentation at the USEPA Region I, II and III Workshop on the Assessment of Health Hazards of Contaminated Soil, Andover, MA.

437. 1986 (April). Critical assumptions in the risk assessment of soil contaminated with 2,3,7,8-TCDD. Presentation at the 196th Annual Meeting of the American Chemical Society, New York, NY.

438. 1986 (March). An examination of critical assumptions in health risk assessments of 2,3,7,8-TCDD contaminated soil. Presentation at the 25th Annual Meeting of Society of Toxicology, New Orleans, LA.

439. 1985 (September). A critical review of assessments of the health risk associated with TCDD in soil. Presentation at the 5th International Symposium on Chlorinated Dioxins and Related Compounds, Bayreuth, Germany.

440. 1985 (May). Shortcomings in risk assessment approaches used in occupational health. Presentation at the Annual Meeting of the American Industrial Hygiene Association, Las Vegas, NV.

441. 1985 (April). Setting occupational exposure limits in high tech industries. Presentation at the Northern California AIHA Symposium on Industrial Hygiene Aspects of Emerging Technologies, Oakland, CA.

442. 1985 (March). A physiologically-based pharmacokinetics (PBPK) model for carbon tetrachloride. Presentation at the Annual Mtg. of the Society of Toxicology, San Diego, CA.

443. 1985 (March). Methods for setting exposure limits. AIHA and NSC Professional Development Course, Salt Lake City, UT.

444. 1985 (February). The shortcomings of most environmental risk assessments. Presentation to the Monsanto Corporation, St. Louis, MO.

445. 1984 (October). What does industry expect engineering graduates to know about environmental safety and health? Presentation to the Accreditation Board for Engineering and Technology, Knoxville, TN.

446. 1984 (April). Principles of occupational health engineering. NIOSH-Sponsored Course for Engineering Professors, Purdue University.

447. 1984 (April). The problem with most risk assessments. Presentation at the Risk Assessment at the New USEPA (sponsored by the Center for Environmental and Energy Management), Washington, DC.

448. 1984 (March). Methods for setting occupational exposure limits and conducting risk assessments. AIHA and National Safety Council Professional Development Seminar, Las Vegas, NV.

449. 1984 (March). The toxicology, environmental fate and ecological impact of pro- drone as a fire ant pesticide. Presentation at the Annual Fire Ant Symposium, Gainesville, FL.

450. 1983 (December). The successful interaction of the toxicologist and the industrial hygienist. Presentation at the 3rd Northeastern Occupational Health Conference: East Brunswick, NJ.

451. 1983 (November). An environmental risk assessment of pro-drone: insect growth regulator for the control of the imported red fire ant. Presentation at the Annual Meeting of Society of Environmental Toxicology and Chemistry, Washington, DC.

452. 1983 (November). Inhalation toxicology methods and data interpretation. Presentation at the Southern States Occupational Health Conference, Tulane University, New Orleans, LA.

453. 1983 (November). The regulatory and societal aspects of using pesticides for fire ant control. Presentation to the Connecticut Chapter of Sigma XI, Southbury, CT.

454. 1983 (October). Quantitative risk assessment and occupational health. Presentation at the Midwest Occupational Health Conference, St. Louis, MO.

455. 1983 (May). Approaches to establishing occupational exposure limits. Symposium chair at the Annual Meeting of the American Industrial Hygiene Association, Philadelphia, PA.

456. 1983 (February). Pharmacokinetics aspects of 12-hour per day exposure schedules. Presentation to the Exxon Environmental Health Center, East Millstone, NJ.

457. 1982-1984. Co-authored and presented with Dr. Tanner Stewart three case studies at the Darden Graduate School of Business, University of Virginia. The cases addressed the ethical and societal difficulties faced by executives who must deal with issues surrounding environmental and occupational safety and health. The cases were later used at several other prominent business schools.

458. 1982 (July). A new technique for educating engineering in occupation safety and health. Presentation at the 90th Annual Conference of the American Society for Engineering Education, Texas A&M University, College Station, TX.

459. 1982 (July). Health physics and industrial hygiene education and training at Purdue University. Presentation at the Annual Meeting of the Health Physics Society, Honolulu, HI.

460. 1982 (June). Industrial hygiene and health physics education at Purdue University. Paper presented at the Annual Meeting of the American Industrial Hygiene Association (with N. Zimmerman), Cincinnati, OH.

461. 1982 (June). The effect of the unusual work shift on the toxicology, disposition, and pharmacokinetics of carbon tetrachloride in the rat. Presentation at the Annual Meeting of the American Industrial Hygiene Association, Cincinnati, OH.

462. 1982 (June). The non-traditional work shift and human health. Editor of the symposia manuscript for the Annual Meeting of the AIHA, Cincinnati, OH.

463. 1982 (June). Toxicological implications of the 12-hour work shift and various models for adjusting occupational exposure limits. Presentation to the University of Texas School of Public Health, Houston, TX.

464. 1982 (April). The fate of carbon tetrachloride in the rat following exposure during the 8- and 12-hour work shift. Presentation at the Annual Meeting of the Soc. of Toxicology, Boston, MA.

465. 1982 (February). The effect of a 12-hour exposure regimen on the distribution, pharmacokinetics and toxicology of carbon tetrachloride in the rat. Presentation to the Rocky Mountain Center for Occupational Health, Salt Lake City, UT.

466. 1982 (January). Criteria for setting occupational exposure limits for unusual work schedules. Presentation to the University of Michigan, School of Public Health, Ann Arbor, MI.

467. 1981 (November). The need to educate engineers in occupational safety and health and a proposal for schools of engineering. Presentation at the Annual Meeting of the American Institute of Chemical Engineers, New Orleans, LA.

468. 1981 (July). The need for industrial hygiene engineering in engineering education. Presentation at the Second NIOSH Symposium, Texas A&M University, College Station, TX.

469. 1979 (October). Engineering control techniques in the chemical industry. Presentation at the New York State Section of the AIHA, Rochester, NY.

470. 1979 (May). The toxicology and pharmacokinetics of inhaled carbon tetrachloride during 8- and 12-hour exposure periods. Presentation at the Annual Meeting of the AIHA, Portland, OR.

471. 1978 (April). Reviewing new manufacturing facility designs in order to minimize future environmental health problems. Presentation at the Midwest Mgmt. Conf., Traverse City, MI.

472. 1978 (April). Nitrosamines: one corporation's experience with a suspect carcinogen. Presentation at the Midwest Management Conference, Traverse City, MI.

473. 1977 (December). The problems of accurate air sampling at the part per billion level. Presentation at the Midwest Safety Conference, Indianapolis, IN.

PUBLISHED ABSTRACTS

1. What Are the Acute Adverse Health Effects of Kratom, if Any: An Analysis of Case Reports. S. Litvin, S. Smallets, S. Kirsh, G. Abele, and **D. Paustenbach**. Poster Presentation at the College on Problems of Drug Dependence (CPDD) Conference. June 14-19, 2025. New Orleans, LA.

2. A Systematic Evaluation of the Literature Related to Alleged Kratom-Induced Seizures. T. Turla, G. Abele, C. Hua, **D. Paustenbach**. Poster Presentation at the College on Problems of Drug Dependence (CPDD) Conference. June 14-19, 2025. New Orleans, LA.

3. Usefulness of the Dust Lamp Method (Tyndall Scattering) for Characterizing Respirable Asbestos. M. E. Stevens, B. M. Deckard, and **D. J. Paustenbach**. Poster Presentation at Society of Toxicology 64th Annual Meeting. March 16-20, 2025. Orlando, Florida.

4. A Critical Review of EPA's OEV for Airborne Formaldehyde and a Proposed Alternative Occupational Exposure Limit. J. Siracusa, Q. Perry, M. Stevens, and **D. Paustenbach**. Poster Presentation at Society of Toxicology 64th Annual Meeting. March 16-20, 2025. Orlando, Florida.

5. An Evaluation and Risk Assessment of PFAS Exposures of Child, Teen, and Adult Athletes from Synthetic Turf Use. E. Denly, J. Siracusa, S. Berlinski, C. House, and **D. Paustenbach**. Poster Presentation at Society of Toxicology 64th Annual Meeting. March 16-20, 2025. Orlando, Florida.

6. Seven Misconceptions Regarding the Health Effects of PFOA, PFOS, and the EPA's 2024 PFAS Drinking Water MCLs. K. McCauley, **D. J. Paustenbach**, S. Smallets, J. Siracusa, M. Stevens, B. Deckard, D. Brew, M. Hua. Poster Presentation at Society of Toxicology 64th Annual Meeting. March 16-20, 2025. Orlando, Florida.

7. A Critical Analysis of the Comments Submitted to the EPA's Proposed 2023 National Primary Drinking Water Regulation PFAS Docket. M. Hua, K. McCauley, D. Brew, J. Heywood, J. Siracusa, M. Stevens, and **D. Paustenbach**. Poster Presentation at Society of Toxicology 64th Annual Meeting. March 16-20, 2025. Orlando, Florida.

8. An Historical Review of the Most Commercially Used Diisocyanates: MDI and TDI Production, Usage, and Occupational Health Hazards. K. McCauley, Q. Perry, and **D. Paustenbach**. Poster Presentation at Society of Toxicology 64th Annual Meeting. March 16-20, 2025. Orlando, Florida.

9. Risk Assessment of Trace Concentrations of Hydrazine in Drinking Water. **D. J. Paustenbach**, B. M. Deckard, K. McCauley, M. Hilsabeck, M. Stevens, D. Brew. Poster Presentation at Society of Toxicology 64th Annual Meeting. March 16-20, 2025. Orlando, Florida.

10. The Acute Adverse Health Effects of Kratom: An Evaluation of Case Reports. S. Litvin, S. Smallets, S. Kirsh, G. Abele, and **D. Paustenbach**. Poster Presentation at Society of Toxicology 64th Annual Meeting. March 16-20, 2025. Orlando, Florida.

11. Assessing Occupational Exposure to Cosmetic Talc During Barbering: A Simulation Study. D. Brew, J. Siracusa, and **D. Paustenbach**. Poster Presentation at Society of Toxicology 64th Annual Meeting. March 16-20, 2025. Orlando, Florida.

12. Toxicological Assessment of Mitragyna Speciosa. S. Smallets, J. Heywood, B. Langenbach, S. Litvin, M. Hilsabeck, **D. J. Paustenbach**. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3088. March 10-14, 2024. Salt Lake City, Utah.

13. Risk Characterization of NDMA in Pharmaceuticals. S. Brown, J. Heywood, M. Donnell, D. Eaton, **D. J. Paustenbach**. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #4715. March 10-14, 2024. Salt Lake City, Utah.

14. An evaluation of trends for mesothelioma mortality in women: Addressing the content of a recent Morbidity and Mortality Weekly Report (MMWR). B. P. Tuttle, M. E. Stevens, R. A. Shore, D. W. Brew, and **D. J. Paustenbach**. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3329. March 10-14, 2024. Salt Lake City, Utah.

15. The presence of erionite in North American soils and the estimated mesothelioma potency by region. M. E. Stevens, **D. J. Paustenbach**, N. J. Lockhart, D. E. Busboom, B. M. Deckard, and D. W. Brew. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #4014. March 10-14, 2024. Salt Lake City, Utah.

16. Proposed occupational exposure limits (OELs) for various asbestos mineral types based on updated mesothelioma dose-response data. **D. J. Paustenbach**, M. E. Stevens, D. W. Brew, and A. Korchevskiy. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3630. March 10-14, 2024. Salt Lake City, Utah.

17. Occupational Exposures to Asbestos in the Steel Industry: An Analysis of the AISI Sampling Campaign (1989-1997). M. E. Stevens, D.W. Brew, & **D. J. Paustenbach**. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3329. March 10-14, 2024. Salt Lake City, Utah.

18. Proposed Acceptable Daily Intake (ADI) for Humans for Chronic Oral Exposure to Hydrazine. **D. J. Paustenbach**, M. Hilsabeck, M. Stevens, B. Deckard, and D. Brew. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #5148. March 10-14, 2024. Salt Lake City, Utah.

19. 2022 (March). Current Thoughts on Risk Assessment Methodologies Used to Assess the Carcinogenic Risk of N-Nitrosodimethylamine (NDMA) and N- Nitrosodiethylamine (NDEA). Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3172. March 27-31, 2022. San Diego, CA.

20. 2022 (March). A Methodology for Estimating 4-ABP Dermal Intake via Contaminated Consumer Products. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3964. March 27-31, 2022. San Diego, CA.

21. 2022 (March). Historical Use of Laboratory Equipment as a Risk Factor for Mesothelioma: A Weight of Evidence Approach. Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3908. March 27-31, 2022. San Diego, CA.

22. 2022 (March). An Analysis of Historical Workplace Exposures to Asbestos at US Steel Facilities (1972-2001). Poster Presentation at Society of Toxicology Annual Meeting. Abstract #3961. March 27-31, 2022. San Diego, CA.

23. Drechsel, D.A., E.S. Fung, K.M. Towle, **D.J. Paustenbach** and A.D. Monnot. 2019. Evaluating the Phototoxic Potential of a Hair Cleansing Conditioner. Accepted Poster Presentation at Society of Toxicology Annual Meeting. March 10-14, 2019, Baltimore, MD.

24. Fung, E.S., D.A. Drechsel, K.M. Towle, **D.J. Paustenbach** and A.D. Monnot. 2019. A Tier-Based Skin Sensitization Testing Strategy for Personal Care Products. Accepted Poster Presentation at Society of Toxicology Annual Meeting, March 10-14, 2019, Baltimore, MD.

25. Monnot, A.D. S.S. Ahmed, A.M. Dickinson, D.A. Drechsel, K.M. Towle, **D.J. Paustenbach**, E.S. Fung. 2019. An In Vitro Human Assay for Evaluating Immunogenic and Sensitization Potential of Personal Care and Cosmetic Products. Accepted Poster Presentation at Society of Toxicology Annual Meeting, March 10-14, 2019, Baltimore, MD.

26. Towle, K.M., D.D. Drechsel, E.S. Fung, **D.J. Paustenbach** and AD. Monnot. 2019. Examination of the FDA Adverse Event Reporting System to Assess the Halo Effect and Potential Reporting Bias. Accepted Poster Presentation at Society of Toxicology Annual Meeting, March 10-14, 2019, Baltimore, MD.

27. Abelmann, A., J.R Maskrey, J. Lotter, A.M. Chapman, M.D. Nemhard, S. Schlaegle, B Bandli, R.J. Lee and **D.J. Paustenbach**. 2018. Asbestos Fiber Transport from Facilities into the Community: Results of Field Studies. Presented at American Industrial Hygiene Conference & Exposition (AIHce), Philadelphia, PA, May 20-23.

28. Lotter, J., A. Abelmann, J.R. Maskrey, A.M. Chapman, M.D. Nemdhard, J.M. Wilmouth, R.J. Lee and **D.J. Paustenbach**. 2018. Take-home exposures following cutting cement pipe. Presented at American Industrial Hygiene Conference & Exposition (AIHce), Philadelphia, PA, May 20-23.

29. Kovochich M, D. Fung, K.M. Unice, S. Mahoney, **D.J. Paustenbach**, and B.L. Finley. 2018. Understanding Divergent Outcomes in MoM-THA Patient Populations with Well-Fixed Components: A Critical Appraisal of Patient Management Protocols and Revision Trends (2000-2017). Presented at Society of Toxicology Annual Meeting, San Antonio, TX, March 11-15.

30. Kovochich M, E.S. Fung, S. Mahoney, K.M. Unice, D. Fung, **D.J. Paustenbach**, and B.L. Finley. 2018. Characterizing cytotoxic and inflammatory responses to metal-on- metal wear debris from normal versus edge-loading conditions. Presented at Society of Toxicology Annual Meeting, San Antonio, TX, March 11- 15.

31. Maskrey, J.R., A. Ablelmann, J. Lotter, A.M. Chapman, M.D. Nembhard, J.M. Wilmoth, R.J. Lee and **D.J. Paustenbach**. 2018. Airborne Chrysotile and Crocidolite Exposure to Workers and Bystanders during Use of a Powered Abrasive Saw to Cut Asbestos-Containing Cement Pipe. Presented at American Industrial Hygiene Conference & Exposition (AIHce), Philadelphia, PA, May 20- 23.

32. Keeton, K.A., S.M. Benson, R.M. Novick, G.M. Marsh and **D.J. Paustenbach**. 2017. The 2014 Crude 4-Methylcyclohexanemethanol Chemical Release and Birth Outcomes in West Virginia. Poster presentation at Society of Epidemiologic Research, June 20- 23, 2017, Seattle, WA.

33. Hollins, D.M., P.K. Scott, J.L Bare, C.A. Barlow, M. Nembhard, J.R. Maskrey and **D.J. Paustenbach**. 2017. Estimating Asbestos Emissions from Former Industrial Sites and Estimating Resulting Airborne Concentrations in the Surrounding Community: A Review of Methodologies. Abstract #3248. Late Breaking Poster Presentation at Society of Toxicology Annual Meeting, March 12-16, 2017, Baltimore, MD.

34. Finley, B.L., E.D. Donovan, M. Kovochich, **D.J. Paustenbach** and A.M. Urban. 2017. Perineal Use of Cosmetic Talc as a Risk Factor for Ovarian Cancer: A Weight-of-Evidence Evaluation. Abstract #1288. Poster Presentation at Society of Toxicology Annual Meeting, March 12-16, 2017, Baltimore, MD.

35. Fung, E.S., A.D. Monnot, B.E. Tvermoes, K.M. Unice, M. Kovochich, D.A. Galbraith, B.L. Finley and **D.J. Paustenbach**. 2017. Characteristics of Cobalt Related Cardiomyopathy in Metal Hip Implant Patients. Abstract #2142. Poster Presentation at Society of Toxicology Annual Meeting. March 12-16, 2017, Baltimore, MD.

36. Kovochich, M. E.S. Fung, E.P. Donovan, K.M. Unice, **D.J. Paustenbach** and B.L. Finley. 2017. Characterization of Wear Debris from Metal-on-Metal Hip Implants during Normal Wear versus Edge Loading Conditions. Abstract #2143. Poster Presentation at Society of Toxicology Annual Meeting, March 12-16, 2017. Baltimore, MD.

37. Monnot, A.D., R.M. Novick and **D.J. Paustenbach**. 2017. An Evaluation of Dermal Irritation Potential of Crude 4-methylcyclohexanemethanol (MCHM) in Humans in 48-Hour Patch Tests. Abstract #2950. Poster Presentation at Society of Toxicology Annual Meeting. March 12-16, 2017, Baltimore, MD.

38. Gaffney, S.H., B.D. Simmons, M.E. Grespin, L. Garnick, **D.J. Paustenbach** and A. Gauthier. 2016. Anthophyllite Asbestos: State of the Art Understanding of its Toxicological Properties. Podium Presentation at American Industrial Hygiene Conference & Exposition (AIHce), May 21-26, 2016, Baltimore, MD.

39. Abelmann, A., M.E. Glynn, J.S. Pierce, P.K. Scott, S. Serrano and **D.J. Paustenbach**. Historical ambient airborne asbestos concentrations in the United States - An analysis of published and unpublished literature. Abstract #1690. Poster Presentation at Society of Toxicology Annual Meeting, March 13- 17, 2016, New Orleans, LA.

40. Gloekler, L., E.C. Shay, N. Schmidt, N. Haghighat, J.M. Panko, D.M. Cowan and **D.J. Paustenbach**. 2016. Flame-retardants in upholstered furnishings: An assessment of health risk and fire-related deaths in the era of California Technical Bulletin (TB-117). Abstract #2669. Poster Presentation at Society of Toxicology Annual Meeting, March 13-17, 2016, New Orleans, LA.

41. Monnot, A.D., B.E. Tvermoes, R. Gerads, H. Gürleyükc and **D.J. Paustenbach**. 2016. Risks associated with arsenic exposure resulting from the consumption of California wines sold in the United States. Abstract #1264. Poster Presentation at Society of Toxicology Annual Meeting, March 13-17, 201, New Orleans, LA.

42. Novick, R.M., P.K. Scott, B. Winans, S.M. Green and **D.J. Paustenbach**. 2016. Estimate of 4-methylcyclohexanemethanol (MCMH) exposure with normal use of contaminated water during the Elk River spill. Abstract #1693. Poster Presentation at Society of Toxicology Annual Meeting, March 13-17, 2016, New Orleans, LA.

43. Tvermoes, B.E., K.M. Unice, B. Winans, M. Kovochich, W.V. Christian, E.D. Donovan, B.L. Finley, I. Kimber and **D.J. Paustenbach**. 2016. Evaluation of immune stimulation following exposure to metal particles and ions using the mouse popliteal lymph node assay. Abstract #3896. Poster Presentation at Society of Toxicology Annual Meeting, March 13-17, 2016, New Orleans, LA.

44. Winans, B., R.M. Novick, S.M. Green and **D.J. Paustenbach**. 2016. QSAR modeling toxicity predictions of the constituents of crude 4- methylcyclohexanemethanol (MCHM) and structurally related chemicals. Abstract #1445. Poster Presentation at Society of Toxicology Annual Meeting, March 13-17, 2016, New Orleans, LA.

45. Huntley-Fenner, G., A Bernal, **D.J. Paustenbach** and B.D. Kerger. 2015. Risks of Unintended Health Consequences Associated with Negative Halo Effects. Presented at the American Marketing Association's 2015 Marketing & Public Policy Conference, June 4-6, 2015, Washington DC.

46. Sahmel, J., H.J. Avens, P.K. Scott, A.M. Burns, C. Barlow, K.M. Unice, A.K. Madl, J. L. Henshaw and **D.J. Paustenbach**. 2015. Characterization of Chrysotile Asbestos Fiber Removal Rates from Air. Podium presentation at the American Industrial Hygiene Conference & Expo (AIHce), May 30-June 4, 2015. Salt Lake City, UT.

47. Christian, W.V., L.D. Oliver, **D.J. Paustenbach**, M.L. Kreider and B.L. Finley. 2015. Toxicology Based Cancer Causation Analysis of CoCr-Containing Hip Implants: A Quantitative Assessment of Genotoxicity and Tumorigenicity Studies. Abstract #2006. Poster presentation at the 54th Annual Meeting and Society of Toxicology (SOT) Meeting at the San Diego Convention Center, San Diego, CA, March 22-26, 2015.

48. Kerger, B.D, C.J. Ronk, M.E. Glynn, B.L. Finley, and **D.J. Paustenbach**. 2014. Age-Related Trends in US Pleural Mesothelioma and Soft Tissue Sarcoma Rates: Evidence for a Longevity Effect. Poster presented at Society of Toxicology (SOT) 53rd Annual Meeting and ToxExpo; Abstract Number 225/Poster Board 402; Phoenix, AZ; March 23-27, 2014.

49. Liong, M., M. Kovochich, B.L. Finley, **D.J. Paustenbach**, and A.K. Madl. 2014. Nanoparticles from the Wear of Cobalt-Chromium Alloy Metal-on-Metal Hip Implants: Physicochemical and Dose Analysis of Patient and Toxicology Studies. Poster presented at Society of Toxicology (SOT) 53rd Annual Meeting and ToxExpo; Abstract Number 603j/Poster Board 472; Phoenix, AZ; March 23- 27, 2014.

50. Monnot, A.D., W.V. Christian, B.L. Finley, and **D.J. Paustenbach**. 2014. Correlation of Blood Cr (III) and Adverse Health Effects: Application of PBPK Modeling to Determine Non-toxic Blood Concentrations. Poster presented at Society of Toxicology (SOT) 53rd Annual Meeting and ToxExpo; Abstract Number 2233/Poster Board 345; Phoenix, AZ; March 23-27, 2014.

51. Thuett, K.A., B.D. Kerger, B.L. Finley, and **D.J. Paustenbach**. 2014. Evaluation of Four Alpha-Diketones for Toll-Like Receptor-4 (TLR-4) Activation in Human Embryonic Kidney Cells. Poster presented at Society of Toxicology (SOT) 53rd Annual Meeting and ToxExpo; Abstract Number 1610/Poster Board 269; Phoenix, AZ; March 23-27, 2014.

52. Ronk, C.J., D.M. Hollins, M.J. Jacobsen, D.A. Galbraith, H.M. Bolstad, and **D.J. Paustenbach**. 2013. Pulmonary Obstruction among Workers at a Food Flavorings Manufacturing Facility. Abstract ID: 5315, Poster presentation at The International Society of Exposure Science (ISES), Basel, Switzerland, August 19- 23, 2013.

53. Bolstad, H.M., D.M. Hollins, C.J. Ronk, A.M. Foda, R.J. Ward, M.J. Jacobsen, and **D.J. Paustenbach**. 2013. Occupational Exposures Measured in a Food Flavoring Manufacturing Facility. Abstract ID: 5397, Poster presentation at The International Society of Exposure Science (ISES), Basel, Switzerland, August 19- 23, 2013.

54. Vishnevskaya, L., K.A. Thuett, W.D. Cyrs, P.S. Chapman, **D.J. Paustenbach**, and B.L. Finley. 2013. Evolution of Warnings and Labels on Encapsulated Asbestos- Containing Products (1930–1990). Session: Legal, Regulatory, Guidelines, Standards. Poster presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 21, 2013; 10:00 AM – 12:00 PM. Presentation Number: SR-403-08.

55. Abelmann, A, J.S. Pierce, M.E. Glynn, S. Serrano, C.J. Ronk, and **D.J. Paustenbach**. 2013. Historical Outdoor Airborne Asbestos Concentrations Associated with Emission Sources in the United States: A Review of Published and Unpublished Data. Session: Aerosols. Poster presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 20, 2013; 10:00 AM – 12:00 PM. Presentation Number: SR-401-03.

56. Sahmel, J., C.A. Barlow, A.M. Burns, P.K. Scott, A.K. Madl, J.L. Henshaw, and **D.J. Paustenbach**. 2013. Measurement of Airborne Asbestos Fiber Settling Rates in a Simulation Study of Clothes Handling. Session: Exposure Assessment Strategies. Podium presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 20, 2013; 3:00 PM – 3:30 PM. Presentation Number: SR-108-04.

57. Gaffney, S.H., B. Donovan, **D.J. Paustenbach**, J. Sahmel, C.A. Barlow, A.K. Madl, J.L. Henshaw, R.J. Lee, and D. Van Orden. 2013. Evaluation of Potential Para- Occupational Exposure to Chrysotile Asbestos during Laundering Activities through a Simulation Study. Session: Exposure Assessment Strategies. Podium presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 20, 2013; 3:30 PM – 4:00 PM. Presentation Number: SR-108-05.

58. Hollins, D.M. C.A. Ronk, A. Foda, R. Ward, M.E. Jacobsen, and **D.J. Paustenbach**. 2013. Inhalation Exposure to Food Flavorings at a Manufacturing Facility. Session: IH General Practice. Podium presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 22, 2013; 2:00 PM – 2:30 PM. Presentation Number: SR-131-03.

59. Ronk, C.A., D.M. Hollins, M.E. Jacobsen, A. Foda, D.A. Galbraith, and **D.J. Paustenbach**. 2013. Pulmonary Restriction among Workers at a Food Flavoring Manufacturing Facility; A Follow Up Study. Session: Occupational and Environmental Epidemiology. Podium presentation at The American Industrial Hygiene Conference & Exposition (AIHce) in Montreal, Quebec, Canada. May 23, 2013; 1:30 PM – 2:00 PM. Presentation Number: SR-146-03.

60. Finley, B.L., B.E. Tvermoes, K.M. Unice, J.M. Otani, **D.J. Paustenbach** and D.A. Galbraith. 2013. Cobalt Whole Blood Concentrations in Healthy Adult Volunteers Following Two-Weeks of Ingesting a Cobalt Supplement. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 12, 2013. Safety Assessment: Non pharmaceuticals; 1:00 P.M. to 4:30 P.M.; Exhibit Halls C&D. Abstract number 1555/Poster Board 616.

61. H.M. Bolstad, K.M. Unice, J. Maskrey, D.M. Hollins, B.D. Kerger and **D.J. Paustenbach**. 2013. Airborne Diacetyl from Cooking and Consumption of Microwave Popcorn: Estimation of Consumer Exposure with a Two-zone Near-field/Far-field Model. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 12, 2013. Exposure Assessment: New Characterizations, Methods, and Models; 1:00 P.M. to 4:30 P.M.; Exhibit Halls C&D. Abstract number 1268/Poster Board 230.

62. Kerger, B.D., R. Gerads, B.L. Finley and **D.J. Paustenbach**, DJ. 2013. Method: Measuring Protein-Bound and Free Cobalt (II) in Human Serum - Size Exclusion Liquid Chromatography with ICP-MS. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 12, 2013. Metals I; 9:00 A.M. to 12:30 P.M.; Exhibit Halls C&D. Abstract number 1168/Poster Board 620.

63. Monnot, A.D., S.H. Gaffney, **D.J. Paustenbach**, and B.L. Finley. Dose-response relationships for blood cobalt concentrations and associated health effects. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10- 14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 12, 2013. Metals II; 1:00 P.M. to 4:30 P.M.; Exhibit Halls C&D. Abstract number 1514/Poster Board 573.

64. **Paustenbach, D.J.**, B.E. Tvermoes, J.M. Otani, K.M. Unice, B.L. Finley, and D.A. Galbraith. 2013. Cobalt blood concentrations and health effects in adult volunteers during a 90-day cobalt supplement ingestion study. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 14, 2013. Late-Breaking Abstract Session; 8:30 A.M. to 12:00 P.M.; Exhibit Hall A. Abstract number 2556/Poster Board 144.

65. Ronk, C.J., D.M. Hollins M.J. Jacobsen, A.M. Foda, D.A. Galbraith and **D.J. Paustenbach**. 2013. Lack of an Association between Cumulative Exposure to Diacetyl and Changes in Pulmonary Health among Workers at a Food Flavorings Manufacturer. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 11, 2013. Epidemiology: Exposures and Associations; 1:00 P.M. to 4:30 P.M.; Exhibit Halls C&D. Abstract number 478/Poster Board 253.

66. Thuett, K.A., B.L. Finley, P.K. Scott and **D.J. Paustenbach**. 2013. Speciation of Chromium Released from Metal-on-Metal Hip Implants. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 12, 2013. Metals I; 9:00 A.M. to 12:30 P.M.; Exhibit Halls C&D. Abstract number 1166/Poster Board 618.

67. Tvermoes, B.E., B.L. Finley, J.M. Otani, K.M. Unice, **D.J. Paustenbach** and D.A. Galbraith. 2013. Effects of Cobalt Dietary Supplementation on Cobalt Body Burden, Steady-State Levels and Selected Biochemical Parameters. Presented at the Society of Toxicology's (SOT) 52nd Annual Meeting, March 10-14, 2013, at the Henry B. Gonzalez Convention Center in San Antonio, Texas. March 14, 2013. Late-Breaking Abstract Session; 8:30 A.M. to 12:00 P.M.: Exhibit Hall A. Abstract number 2555/Poster Board 143.

68. Barlow, C.A., J. Sahmel, A.K. Madl, B. Donovan, S. Gaffney, J. Henshaw, R.J. Lee, D. Van Orden, and **D.J. Paustenbach**. Evaluation of Chrysotile Fiber Adherence to Clothing Exposed to Known Airborne Asbestos Concentrations Before and After Handling and Shaking Out of the Clothing. Presented at the 2012 Society for Risk Analysis (SRA) Annual Meeting; December 9-12, 2012; San Francisco, CA.

69. Sahmel, J., C.A. Barlow, B. Donovan, S.H. Gaffney, A.K. Madl, J.L. Henshaw, R.J. Lee, D. Van Orden, and **D.J. Paustenbach**. Evaluation of Potential Take Home Exposure during Laundering Activities: A Simulation Study of Airborne Chrysotile Concentrations Associated with Handling Clothing Exposed to Known Levels of Airborne Asbestos. Presented at the 2012 Society for Risk Analysis (SRA) Annual Meeting; December 9-12, 2012; San Francisco, CA.

70. Tvermoes, B.E., J. Otani, K.M. Unice, B.L. Finley, **D.J. Paustenbach**, D.A. Galbraith. Investigation of Cobalt Steady-State Levels in Five Healthy Adult Volunteers Taking 14-Days of a Cobalt Supplement. Presented at the 2012 Society for Risk Analysis (SRA) Annual Meeting; December 9-12, 2012; San Francisco, CA.

71. Monnot, A.D., S.H. Gaffney, **D.J. Paustenbach**, and B.L. Finley. Derivation of a Chronic Oral Reference Dose for Cobalt. Presented at the 2012 Society for Risk Analysis (SRA) Annual Meeting; December 9-12, 2012; San Francisco, CA.

72. Hollins, D.M., M.J. Jacobsen, **D.J. Paustenbach**, D.A. Galbraith and C.J. Ronk. Comparing Pulmonary Function Data in Flavorings Manufacturing Workers. Submitted to the 45th Society for Epidemiological Research (SER) Annual Meeting. June 27 - 30, 2012, in Minneapolis, MN.

73. Avens, H.A., K.M. Unice, J. Sahmel, S.A. Gross, J.J. Keenan and **D.J. Paustenbach**. Analysis and Modeling of Airborne BTEX Concentrations from the Deepwater Horizon Oil Spill. Submitted to the "Assessing Exposure During Disaster Response: The Gulf Oil Spill Experience" roundtable. Presented at 2012 American Industrial Hygiene Conference & Expo (AIHce), June 16-21, 2012, in Indianapolis, IN.

74. Lew, M.G. R.M. Novick, J.J. Keenan and **D.J. Paustenbach**. Is dermal sensitization for 1,2-benzisothiazolin-3-one [BIT] in consumer products a cause for concern? Submitted to Society of Toxicology's 51st Annual Meeting, March 11–15, 2012, at the Moscone Convention Center in San Francisco, California.

75. Phelka A., Finley, B.L., Pierce, J.S., Adams, R.E., **Paustenbach, D.J.**, Thuett, K.A. and Barlow, C.A. Tremolite Asbestos Exposures Associated with the Use of Commercial Products. Presented at Society of Toxicology's (SOT) 51st Annual Meeting, March 11–15, 2012, at the Moscone Convention Center in San Francisco, California. March 14, 2012. Exposure Assessment: Case-Specific Characterizations; 9:00 AM - 12:30 PM; Exhibit Hall; 2115 Poster Board -539.

76. Novick, R.M., J.J. Keenan and **D.J. Paustenbach**. An Analysis of Historical Exposures of Pressmen to Airborne Benzene (1930s to 2006). Submitted to Society of Toxicology's 51st Annual Meeting, March 11–15, 2012, at the Moscone Convention Center in San Francisco, California.

77. Adams R.E., Finley, B.L., Pierce, J.S., Phelka, A., **Paustenbach, D.J.**, Thuett, K.A. and Barlow, C.A. Derivation of LOAEL and NOAEL for Tremolite Asbestos. Presented at Society of Toxicology's (SOT) 51st Annual Meeting, March 11–15, 2012, at the Moscone Convention Center in San Francisco, California. March 14, 2012. Exposure Assessment: Case-Specific Characterizations; 9:00 AM - 12:30 PM; Exhibit Hall; 2114 Poster Board -538.

78. Knutsen, J.S., B.D. Kerger, B.L. Finley, and **D.J. Paustenbach.** Human PBPK Modeling of Benzene Inhalation Based Chinese Worker Urinary Metabolite Data: Comparison of Human and Mouse Metabolism. Submitted to Society of Toxicology's 51st Annual Meeting, March 11–15, 2012, at the Moscone Convention Center in San Francisco, California.

79. Cowan, D.M., M. Anderle de Sylor, L.J. Lievense, A.J. Slocombe, M.G. Fromowitz, and **D.J. Paustenbach**. Product sustainability: The role of chemical watch lists in chemical deselection from products manufacturing processes. Society of Toxicology (SOT) Annual Meeting, March 6-10, 2011. Washington, D.C.

80. Gross, S. A., R.D. Irons, D. Galbraith and **D.J. Paustenbach**. A Case Control Study of Chronic Myelomonocytic Leukemia in Shanghai, China. Society of Toxicology (SOT) Annual Meeting, March 6-10, 2011. Abstract #1210, Poster Board #713. Washington, D.C. (Epidemiology and Exposure Evaluations)

81. Keenan, J., H. Avens, K. Unice and **D.J. Paustenbach**. Evaluation of Airborne Toxicant Concentrations from the Deepwater Horizon Oil Spill. Society of Toxicology (SOT) Annual Meeting, Tuesday, March 8, 2011. Abstract #1262, Poster Board #834. Washington, D.C. (Exposure Assessments and Biomonitoring Applications)

82. Kerger, B.D., P.K. Scott, M. Pavuk, M. Gough, and **D.J. Paustenbach**. Reverse Causation of Dioxin Dose-Response Trends for Risk of Diabetes Mellitus Type 2 Among Operation Ranch Hand Vietnam Veterans. Society of Toxicology (SOT) Annual Meeting, March 6-10, 2011. Abstract #2255, Poster Board #359. Washington, D.C. (Persistent Organic Compounds (POPs)

83. Tvermoes, B., Anderle de Sylor, M., Sahmel, J., Cyrs, W. and **D.J. Paustenbach**. An assessment of the bioavailability of cadmium in thin-film PV modules. Society of Toxicology (SOT) Annual Meeting, Thursday March 10, 2011. Abstract #2909, Poster Board #155. Exhibit Hall, Convention Center. Washington, D.C. (Grace Period Abstracts - Session IV)

84. Gaffney, S., J. Sahmel, J. Knutsen, **D.J. Paustenbach**. Determinants of Deathly Carbon Monoxide Exposure inside a Recreational Vehicle. American Industrial Hygiene Conference and Expo (AIHce), May 14 – May 19, 2011, Portland, Oregon. PO 109-3.

85. Sahmel, J., K. Devlin, **D.J. Paustenbach**, D.M. Hollins, S.H. Gaffney. Exposure Reconstruction in Occupational Human Health Risk Assessment: Current Methods and a Recommended Framework. American Industrial Hygiene Conference and Expo (AIHce), May 14 – May 19, 2011, Portland, Oregon. PO 126-4.

86. Sahmel, J., K. Devlin, A.M. Burns, T. Ferracini, M. Ground, and **D.J. Paustenbach**. Analysis of Historical Industrial Hygiene Data: A Case Study Involving Benzene Exposures at a Petrochemical Manufacturing Facility (1974- 1999). American Industrial Hygiene Conference and Expo (AIHce), May 14 – May 19, 2011, Portland, Oregon. PS 402-18.

87. Le, M.H. and D**.J. Paustenbach**. The impact of the Globally Harmonized System of Classification and Labeling of Chemicals (GHS) on the changing regulatory landscape of hazard communication. Presented at the Society for Risk Analysis (SRA) Annual Meeting, December 5-8, 2010. Abstract #M3-C.3, Salt Lake City, UT.

88. **Paustenbach, D.J.** Reflections on the role of risk assessment at OSHA over the 40-year journey: Has it been a big disappointment? Presented at the Society for Risk Analysis (SRA) Annual Meeting, December 5-8, 2010. Abstract #M3-A.4. Salt Lake City, UT.

89. Sahmel, J., K. Devlin, **D.J. Paustenbach**, D. Hollins, and S.H. Gaffney. The role of exposure reconstruction in occupational human health risk assessment: Current methods and recommended framework. Presented at the Society for Risk Analysis (SRA) Annual Meeting, December 5-8, 2010. Abstract #M3-E.2, Salt Lake City, UT

90. Unice, K.M., P.K. Scott, and **D.J. Paustenbach**. Review of exposure models assessing outdoor use of volatile consumer and industrial products. Presented at the Society for Risk Analysis (SRA) Annual Meeting, December 5-8, 2010. Abstract #T4-E.2, Salt Lake City, UT.

91. Avens, H.J., J.J. Keenan, K.M. Unice, and **D.J. Paustenbach**. Estimating the airborne concentrations of benzene and other relevant volatiles in boats operating in or near oil spills: A comparison between the Exxon-Valdez and Deepwater Horizon incidents. Presented at the Society for Risk Analysis (SRA) Annual Meeting, December 5-8, 2010. Abstract #W3-E.1, Salt Lake City, UT.

92. Donovan, E.P., B.L. Donovan, P. Chowdhary and **D.J. Paustenbach**. Possible Risks Associated with Coalbed Methane-Produced Water. Presented at 2010 Society of Environmental Toxicology and Chemistry (SETAC) Conference, Portland, OR. Monday November 8th, 2010, Exhibit Hall.

93. **Paustenbach, D.J.** 2010 Reflections on thirty years in chemical risk assessment and thoughts about the next ten years. Presented at the Conference on Environmental Decisions: Risks and Uncertainties – Switzerland. April 25-29, 2010, Centro Stefano Franscini, Monte Verità, Switzerland

94. Keenan, J.J., S.H. Gaffney, D.A. Galbraith, P. Beatty and **D.J. Paustenbach**. 2010. Gasoline: A complex mixture or a dangerous vehicle for benzene exposure? Chem-Biol Interact. 184(1-2):293-295.

95. **Paustenbach, D.J.**, J.S. Knutsen, D.M. Hollins, J.E. Sahmel, and A.K. Madl. 2010. Comparison of modeled and measured concentrations of airborne benzene from the use of petroleum-based solvents spiked with low levels of benzene. Chem-Biol Interact. 184(1-2):296-298.

96. Cowan, D.M., J. Sahmel, K. Unice, P. Scott, and **D.J. Paustenbach**. Potential for Occupational Exposures of Hairdressers to Vinyl Chloride in Hairspray (1967-1974). Presented at the 48th Annual Society of Toxicology's Meeting and ToxExpo, March 15-19, 2009. Abstract #833. Baltimore, MD.

97. Gaffney, S.H., A.K. Madl, J.L. Balzer, and **D.J. Paustenbach**. Mechanic and Bystander Chrysotile Exposures During Heavy Equipment Brake Removal. Presented at the 48th Annual Society of Toxicology's Meeting and ToxExpo, March 15-19, 2009. Abstract #1196. Baltimore, MD.

98. Keenan, J., M.H. Le, **D.J. Paustenbach**, and S.H. Gaffney. Lead Testing Wipes Contain Measurable Background Levels of Lead. Presented at the 48th Annual Society of Toxicology's Meeting and ToxExpo, March 15-19, 2009. Abstract #1477. Baltimore, MD.

99. Kerger, B.D., J.S. Knutsen, J.R. Kuykendall, and **D.J. Paustenbach**. PBPK Modeling of Benzene Metabolites in Bone Marrow of Humans with Varied Workplace Exposure Patterns and CYP2E1 Activity. Presented at the 48th Annual Society of Toxicology's Meeting and ToxExpo, March 15-19, 2009. Abstract #1265. Baltimore, MD.

100. Murbach, D.M., K.D. Devlin, K.S. Franke, and **D.J. Paustenbach**. A Review of Historical Ambient Airborne Asbestos Concentrations in Cities and Buildings: 1950s to the Present Day. Presented at the International Society for Environmental Epidemiology and International Society of Exposure Analysis Joint Annual Conference (ISEE/ISEA), October 12-16, 2008. Abstract #1274. Pasadena, CA.

101. Donovan, B.L., E.P. Donovan, J. Sahmel, B. Epstein, and **D.J. Paustenbach**. Evaluation of Bystander Exposure to Asbestos: Review of the Literature (1950s – Present). Presented at the International Society for Environmental Epidemiology and International Society of Exposure Analysis Joint Annual Conference (ISEE/ISEA), October 12-16, 2008. Abstract #1011. Pasadena, CA.

102. Sahmel, J., P.K. Scott, K.M. Unice, and **D.J. Paustenbach**. Potential Occupational Exposures of Hairdressers to Vinyl Chloride in Hairspray (1962- 1973). Presented at the International Society for Environmental Epidemiology and International Society of Exposure Analysis Joint Annual Conference (ISEE/ISEA), October 12-16, 2008. Abstract #1150. Pasadena, CA.

103. Sweet, L., K.M. Unice, J. Panko, and **D.J. Paustenbach**. Conducting Risk Assessments of Chemicals in Consumer Products. Presented at the International Society for Environmental Epidemiology and International Society of Exposure Analysis Joint Annual Conference (ISEE/ISEA), October 12-16, 2008. Abstract #922. Pasadena, CA.

104. Gaffney, S.H., M.L. Kreider, K.M. Unice, A.M. Burns, **D.J. Paustenbach**, L.E. Booher, R.H. Gelatt, and J.M. Panko. Benzene Exposure in Refinery Workers (1976- 2006). Presented at the International Society for Environmental Epidemiology and International Society of Exposure Analysis Joint Annual Conference (ISEE/ISEA), October 12-16, 2008. Abstract #864. Pasadena, CA.

105. Knutsen, J., J. Kuykendall, **D.J. Paustenbach**. Comparing Equal Delivered Doses of Airborne Benzene for 8 hr./day Steady Exposure vs. Peak Exposure Regimens Using a PBPK Model. Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Abstract #14. Minneapolis, MN.

106. Knutsen, J., D. Murbach, **D.J. Paustenbach**, A. Madl. Comparison of Modeled and Measured Concentrations of Airborne Benzene from the Use of Petroleum- Based Solvents Spiked with Low Levels of Benzene. Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Abstract #53. Minneapolis, MN.

107. Gaffney, S., M. McKinley, A. Madl, **D.J. Paustenbach**, J. Knutsen. Validation of Two Different Exposure Models Using the Results of a Simulation Study Involving Exposures to Methanol Vapors During the Cleaning of Semiconductor Wafers. Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Abstract #55. Minneapolis, MN.

108. Panko, J., A. Burns, K. Unice, M. Kreider, S. Gaffney, **D.J. Paustenbach**. Benzene Exposure in Refinery Workers: Baytown, TX (1978-2006). Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Abstract # 191. Minneapolis, MN.

109. Shay, E., P. Scott, **D.J. Paustenbach**. Residential and Occupational Indoor Surface Dust Criteria for and PAHs and PCDD/PCDF. Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Abstract #261. Minneapolis, MN.

110. Gaffney, S., E. Donovan, J. Clarke, **D.J. Paustenbach**. Analysis of Historical Air Monitoring Data for Copper Beryllium at a Manufacturing Plant (1964-2000). Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Abstract # 298. Minneapolis, MN.

111. Shay, E., K. Unice, J. Knutsen, J. Panko, **D.J. Paustenbach**. Groundwater Vapor Intrusion into a School – Modeled Concentrations vs. Monitoring Results. Presented at the American Industrial Hygiene Conference & Exposition (AIHce), May 31-June 5, 2008. Abstract #300. Minneapolis, MN.

112. Harris, M., J.A. Tachovsky, E.S. Williams, **D.J. Paustenbach**, L.C. Haws. Assessment of the Health Risks Posed by Benzene in Certain Soft Drinks. Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Abstract #697. Seattle, WA.

113. Phelka, A.D., J.A. Clarke, **D.J. Paustenbach**, B.L. Finley. The Importance of Asbestos Fiber Length as a Predictor of Potency for Asbestos-Related Disease. Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Abstract #1185. Seattle, WA.

114. Kreider, M.L., K.M. Unice, J.M. Panko, T.E. Widner, **D.J. Paustenbach**, L.E. Booher, R.H. Gelatt. S.H. Gaffney. Occupational Exposure to Benzene the ExxonMobil Refinery in Joliet, Illinois (1977-2006). Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Abstract #1471. Seattle, WA.

115. Finley, B.L., A.K. Madl, D.M. Murbach, K.A. Fehling, **D.J. Paustenbach**, G.C. Jiang. A Study of Airborne Chrysotile Concentrations Associated with Handling, Unpacking, and Repacking Boxes of Automobile Clutch Discs (circa 1950- 1980). Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Abstract #1458. Seattle, WA.

116. Jiang, G.C., A.K. Madl, **D.J. Paustenbach**, B.L. Finley. Detailed Asbestos Fiber Size and Morphology Analyses of Automobile Clutch Discs, Brake Pads, and Brake Shoes. Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Abstract #1468. Seattle, WA.

117. Kerger, B.D., W.J. Butler, **D.J. Paustenbach**, J. Zhang, S. Li. Cancer Mortality in Chinese Populations Surrounding an Alloy Plant with Chromium Smelting Operations (1960-1978). Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Abstract #1472. Seattle, WA.

118. Fehling, K., J.R. Kuykendall, S. Shaw, **D.J. Paustenbach**, D. Collins. The Possible Community Health Hazards Posed by Emissions from Automobile Traffic Tunnels: A Review of the Literature. Presented at the 47th Annual Society of Toxicology's Meeting and ToxExpo, March 16-20, 2008. Abstract #1794. Seattle, WA.

119. Chapman, P., P.R. Williams, J. Kuykendall, M.A. McKinley, K. Franke, D.M. Murbach, A. Tachovsky, **D.J. Paustenbach**. Meta-Analysis of Asbestos-Related Disease Among Skilled Craftsmen in Various Occupational Settings, October 14-18, 2007. Abstract #300. Durham/Research Triangle Park, NC.

120. Flack, S.M., T.E. Widner and **D.J. Paustenbach**. Potential Radiation Exposures to Residents of New Mexico from the World's First Test of a Nuclear Device (Trinity Site, July 16, 1945), October 14-18, 2007. Abstract #390. Durham/Research Triangle Park, NC.

121. Gaffney, S.H., E.C. Moody, M.A. McKinley, J.S. Knutsen, A.K. Madl, **D.J. Paustenbach**. Worker Exposure to Methanol Vapors During Cleaning of Semiconductor Wafers in a Manufacturing Setting: Results of a Simulation Study, October 14-18, 2007. Abstract #301. Durham/Research Triangle Park, NC.

122. Jiang, G.C.-T., A.K. Madl, D.M. Murbach, K.A. Fehling, B.L. Finley, **D.J. Paustenbach** 2007. Exposure to Chrysotile Asbestos Associated with Handling, Unpacking, and Repacking Boxes of Automobile Clutch Discs, October 14-18, 2007. Abstract #94. Durham/Research Triangle Park, NC.

123. Murbach, D.M., C.M. English, J.S. Knutsen, **D.J. Paustenbach**, A.K. Madl. Reconstruction of Airborne Benzene Exposures Associated with the Use of Commercial Products Containing Solvents, October 14-18, 2007. Abstract #357. Durham/Research Triangle Park, NC.

124. Robinson, K.D., **D.J. Paustenbach**, A.D. Phelka, A.K. Madl. 2007. Factors Influencing the Development of Warnings on Regulated and Non-Regulated Consumer Products, October 14-18, 2007. Abstract #359. Durham/Research Triangle Park, NC.

125. Widner, T.E., S.H. Gaffney, A.F. Javier, J.M. Panko, M.L. Kreider, J.R. Marshall, L.E. Booher, **D.J. Paustenbach**. Benzene Exposures of Dock Facility Workers Serving a Refinery and Chemical Plant: 1977-2005, October 14-18, 2007. Abstract #320. Durham/Research Triangle Park, NC.

126. Widner, T.E., K.D. Robinson, S.M. Flack and **D.J. Paustenbach**. Residential Are Development and Potential for Public Exposures around the Manhattan Project and Early AEC Sites, October 14-18, 2007. Abstract #298. Durham/Research Triangle Park, NC.

127. Flack, S.M., T.E. Widner and **D.J. Paustenbach**. A Review of the World's First Test of a Nuclear Device (Trinity Site, July 16, 1945) and Potential Radiation Exposures to Residents of New Mexico, July 8-12, 2007. Portland, OR.

128. Widner, T.E., K. Robinson, S.M. Flack and **D.J. Paustenbach**. A Review of Residential Areas Developed around Manhattan Project and Early AEC Sites and Potential Pathways for Public Exposures, July 8-12, 2007. Portland, OR.

129. Chapman, P., P.R. Williams, J. Kuykendall, **D.J. Paustenbach**. Meta-Analysis of Asbestos-Related Disease Among Skilled Craftsmen in Various Occupational Settings, March 25-29, 2007. Abstract #1991-210. Charlotte, NC.

130. Ferriby, L., M. Harris, K. Unice, P. Scott, L. Haws, **D.J. Paustenbach**. Development of PCDD/F and Dioxin-Like PCB Serum Concentration Reference Values for the General U.S. Population Using the 2005-WHO TEFs and the 2001- 2002 NHANES Data. Abstract #425-320. Charlotte, NC.

131. Williams, E.S., L. Ferriby, L.C. Haws, D.J. Paustenbach, M.A. Harris. Assessment of Potential Human Health Risks Posed by Benzene in a Commercial Beverage, Abstract #1446-217. Charlotte, NC.

132. Nguyen, L., D. Staskal, E.S. Williams, W. Luksemburg, L. Haws, L. Birnbaum, **D.J. Paustenbach**, M. Harris. Dietary Intake of PBDEs Based on Consumption of Catfish in Southern Mississippi, Abstract #712. Charlotte, NC.

133. Williams, P.R., J. Panko, K. Unice, J. Brown, **D.J. Paustenbach**. Occupational Exposures Associated with Petroleum-Derived Solvents Containing Trace Levels of Benzene, Abstract #715. Charlotte, NC.

134. Gaffney, S., and **D.J. Paustenbach**. Reliability of Spot Urine Samples. Abstract# P- 144 Presented at the International Conference on Environmental Epidemiology and Exposure, September 3-4, 2006. Paris, France.

135. Williams, P., J. Knutsen, C. Atkinson, A. Madl, **D.J. Paustenbach**. Airborne Concentrations of Benzene Associated with the Historical Use of Liquid Wrench. Abstract# MAB2-PD-08 Presented at the International Conference on Environmental Epidemiology and Exposure, September 4, 2006. Paris, France.

136. **Paustenbach, D.J.**, J.R. Kuykendall, J.M. Warmerdam, P. Moy, B. Finley. Factors Affecting the Bioaccessibility and Bioavailability of Hexavalent Chromium and Dioxin Contaminants in Soil, and Their Relative Risk Assessment. Abstract# MSI-05 Presented at the International Conference on Environmental Epidemiology and Exposure, September 4, 2006. Paris, France.

137. Murbach, D., P. Chapman, A. Madl, J. Brown, **D.J. Paustenbach**. Evaluation of Bricklayer Exposures to Airborne Asbestos in Steel Mills (1972-1981). Abstract# P- 609 Presented at the International Conference on Environmental Epidemiology and Exposure, September 5-6, 2006. Paris, France.

138. Murbach, D., P. Chapman, A. Madl, **D.J. Paustenbach**. Evaluation of Background Exposures to Airborne Asbestos on Maritime Shipping Vessels (1972-1992). Abstract# P-608 Presented at the International Conference on Environmental Epidemiology and Exposure, September 5-6, 2006. Paris, France.

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146. Haws, L.C., P.K. Scott, K.M. Unice, M. Gough, M.A. Harris, D.S. Staskal, **D.J. Paustenbach** and M. Pavuk. Are dioxin body burdens surrogates for other risk factors in associations between dioxin and diabetes? Abstract# O-071. Presented at the 26th International Symposium on Halogenated Environmental Organic Pollutants and POPs, August 21-25, 2006. Oslo, Norway.

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148. Williams, P., and **D.J. Paustenbach**. Reconstruction of Exposure of Skilled Craftsmen to Asbestos at the Beaumont, Texas Refinery (1946-2004). Abstract #128 Presented at the American Industrial Hygiene Conference & Expo (AIHce), May 13-18, 2006. Chicago, IL.

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150. Madl, A., E. Donovan, **D.J. Paustenbach**, M. Kelsh. Assessment of Exposure- Response Patterns for Beryllium Sensitization and Chronic Beryllium Disease. Abstract #275 Presented at the American Industrial Hygiene Conference & Expo (AIHce), May 13-18, 2006. Chicago, IL.

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162. Williams, P.R.D., and **D.J. Paustenbach**. A historical review of asbestos exposures among skilled craftsmen. Abstract #M-30p. Annual International Society of Exposure Analysis Conference (ISEA), October 30-November 3, 2005. Tucson, AZ.

163. Madl, A.K., M.A. Kelsh, **D.J. Paustenbach**. Exposure and the prevalence of chronic beryllium disease and beryllium sensitization. Abstract #M-31p. Annual International Society of Exposure Analysis Conference (ISEA), October 30-November 3, 2005. Tucson, AZ.

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166. **Paustenbach, D.J.** Incorporating the hormesis into the risk assessment paradigm. Abstract #21. 9th International Conference on Environmental Mutagens & 36th Annual Meeting of the Environmental Mutagen Society, September 3-8, 2005. San Francisco, CA.

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169. **Paustenbach, D.J.**, K. Fehling, M. Harris, P. Scott, B. Kerger. Identifying a soil clean- up criteria for dioxin in residential soils: How has 20 years of research and risk assessment experience impacted the analysis? Abstract #1922. 25th International Symposium on Halogenated Environmental Organic Pollutants and POPs, 20th International Symposium on Polycyclic Aromatic Compounds, August 21-26, 2005. Toronto, Canada.

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172. **Paustenbach, D.J.**, A. Madl, K. Clark, K. Fehling, T. Lee. Chrysotile Asbestos Exposure Associated with Removal of Automobile Exhaust Systems (Circa 1946- 1970) Abstract #410. Society of Toxicology's 44th Annual Meeting, March 6-10, 2005. New Orleans, LA.

173. Madl, A., K. Unice, P. Scott, K. Robinson, P. Scaramella, D. Pyatt, **D.J. Paustenbach**. Exposure Reconstruction of Historical Airborne Benzene Concentrations: Case study of a Deck Crewman on board Crude Oil and Chemical Tankers. Abstract #411. Society of Toxicology's 44th Annual Meeting, March 6-10, 2005. New Orleans, LA.

174. Juberg, D., **D.J. Paustenbach**. Current Regulatory and Scientific Views Regarding Chemical Hazards to Children. Abstract #1338. Society of Toxicology's 44th Annual Meeting, March 6-10, 2005. New Orleans, LA.

175. **Paustenbach, D.J.**, R.O. Richter, B.L. Finley, P.R.D. Williams, and P.J. Sheehan. Evaluating Asbestos Exposures Associated with Vehicle Brake Cleaning and Machining Activities using Short-Term and TWA Measurements. Abstract #T6.3. Annual Meeting of the Society for Risk Analysis (SRA), Dec. 5-8, 2004. Palm Springs, CA.

176. Fontaine D., **D.J. Paustenbach**, B. Landenberger, R. Budinsky, T.B. Starr. An Evaluation of the Toxic Equivalency Factor (TEF) for 2,3,4,7,8-PCDF (4-PCDF) using Data from the Recent NTP Dioxin Bioassays. Abstract #W8.7. Annual Meeting of the Society for Risk Analysis (SRA) Conference, December 5-8, 2004. Palm Springs, CA.

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180. **Paustenbach, D.J.**, A.K. Madl, K.A. Clark, K.A. Fehling, T. L. Carolina. Chrysotile Asbestos Exposure Associated with Removal of Automobile Exhaust Systems (Circa 1950-1974): Preliminary Findings of a Simulation Study. Abstract #W2D-03. International Society of Exposure Analysis 2004 Conference, October 17- 21, 2004. Philadelphia, PA.

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189. **Paustenbach, D.J.** Our Future is Tied to Updating Exposure Limits. Presented at the American Industrial Hygiene Association's 2004 Annual Meeting, June 2004. Oakland, CA.

190. Mowat, F., M. Bono, and **D.J. Paustenbach**. Occupational Exposure to Airborne Chrysotile Asbestos during Use and Removal of Mastics, Coating, and Adhesives (circa 1940's-present day). Abstract #525. Presentation at the Society of Toxicology's 43rd Annual Meeting, March 22-25, 2004. Baltimore, MD.

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192. **Paustenbach, D.J.** and G. Jepson. Consideration Relevant to Constructing a Human PBPK Model for Perfluorooactanoic Acid (PFOA). Abstract #1919. Presentation at the Society of Toxicology's 43rd Annual Meeting, March 22-25, 2004. Baltimore, MD.

193. **Paustenbach, D. J.**, G. Brorby, and B.L. Finley. Environmental and Occupational Health Hazards Associated with the Presence of Asbestos in Brake Linings and Pads (1900 to Present). Abstract #T14.6. Presentation at the Society for Risk Analysis Annual Meeting, December 7-10, 2003. Baltimore, MD.

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195. **Paustenbach, D.J.** and P.R.D. Williams. Evaluating Occupational Exposures to Benzene at an Industrial Chemical Plant. Abstract #T23.1. Presentation at the Society for Risk Analysis Annual Meeting, December 7-10, 2003. Baltimore, MD.

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200. Hays, S.M., Cushing, C. Pratt, D. Holiday, K. **D.J. Paustenbach**. Exposure of Infants and Children in the U.S. to the Flame Retardant Decabromodiphenyl Oxide (DBDPO). Presented at the 23rd International Symposium of Halogenated Organic & Persistent Organic Pollutants, August 24-29, 2003. Boston, MA.

201. Hays, S.M, D.M Proctor, and **D.J. Paustenbach**. Refining PBPK model for chromium (VI) in humans. Abstract #280. Presentation at the Society of Toxicology 42nd Annual Meeting. March 9-13, 2003. Salt Lake City, UT.

202. Duffy, J.S., J. Fessler, T. Gauthier, J. Greene, S. Medhekar, R. Mongia, B. Murphy, and **D.J. Paustenbach**. Evaluating the threat of chemical and biological warfare via mail or drinking water delivery systems. Abstract #799. Presentation at the Society of Toxicology 42nd Annual Meeting, March 9-13, 2003. Salt Lake City, UT.

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204. Cushing, C.A., K.C. Holicky, D.W. Pyatt, D. Staskal, B.L. Finley, **D.J. Paustenbach**, and S.M. Hays. Estimated children's exposure to decabromodiphenyl oxide in the U.S. Abstract #1906. Presentation at the Society of Toxicology 42nd Annual Meeting, March 9-13, 2003. Salt Lake City, UT.

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226. Williams, P.R., P.J. Sheehan, and **D.J. Paustenbach**. A probabilistic assessment of household exposures to MTBE from drinking water. Abstract #105. Final Program and Abstracts. 40th Annual Meeting of Society of Toxicology, March 25-29, 2001, San Francisco, CA.

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325. **Paustenbach, D.J.** 1989. Improvements in health risk assessment 1990s. 5th Annual HazTech Meeting, San Francisco, CA.

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328. **Paustenbach, D.J.** and C.K. Kasunic. 1989. Dioxin emissions from incinerators. Abstract #8A. 6th International Meeting on Dioxins and Related Chemicals, Toronto, Canada.

329. **Paustenbach, D.J.** and S. Ripple. 1989. Applications of risk assessment to toxic tort litigation. Abstract #3. Annual Meeting of the American Bar Association, Honolulu, HI.

330. Leung, H.W., A.P. Poland, F.J. Murray, **D.J. Paustenbach**, and M.E. Andersen. 1989. Dose-dependent pharmacokinetics of [125I]-2-IODO- 3,7,8- trichlorodibenzo-p-dioxin (I-TCDD) in mice. Abstract #946. 28th Annual Meeting of the Society of Toxicology, Atlanta, GA.

331. Sarlos, T., B. Fishman, and **D.J. Paustenbach**. 1989. Evaluation of emission rate and health risks of volatile gasoline components of soil. Abstract #P111. Annual Meeting of the Society of Environmental Toxicology and Chemistry, Toronto, Canada.

332. **Paustenbach, D.J.** 1987. Determining significant risk under Proposition 65. Abstract #2D. 3rd Hazmat West Conference, Long Beach, CA.

333. **Paustenbach, D.J.** 1987. Environmental and public health hazards of contaminated soil. Abstract 12. 2nd Conference on the Environmental and Public Health Effects of Petroleum Contaminated Soils, Amherst, MA.

334. Fries, G.F. and **D.J. Paustenbach**. 1987. A critical evaluation of the factors used in assessing incinerator emissions as a potential source of TCDD in foods of animal origin. Abstract #RC-05. 7th International Dioxin Symposium, Las Vegas, NV.

335. Fries, G.F. and **D.J. Paustenbach**. 1987. Evaluation of factors used to assess incinerator emissions as a potential source of TCDD in foods of animal origin. Abstract #279. 8th Annual Meeting of the Society for Environmental Toxicology and Chemistry, Pensacola, FL.

336. Leung, H.W. and **D.J. Paustenbach**. 1987. Documentation for an occupational exposure limit for 2,3,7,8-TCDD. Abstract #RC-04. 7th International Dioxin Symposium, Las Vegas, NV.

337. **Paustenbach, D.J.** and G.F. Fries. 1987. A risk assessment of incinerator emissions as a potential source of TCDD in foods of animal origin. Abstract #WAM-C4. 6th Annual Meeting of the Society of Risk Analysis, Houston, TX.

338. Leung, H.W., M.E. Anderson, R.H. Ku, and **D.J. Paustenbach**. 1987. A physiologically based pharmacokinetics model for 2,3,7,8-TCDD. Abstract #MA-02. 7th International Dioxin Symposium, Las Vegas, NV.

339. Leung, H.W., M.E. Andersen, R.H. Ku, and **D.J. Paustenbach**. 1988. A physiological pharmacokinetics description of the tissue distribution and enzyme induction of 2,3,7,8-TCDD in the rat. Abstract #618. Annual Meeting of the Society of Toxicology, Dallas, TX.

340. Shu, H., **D.J. Paustenbach**, J. Murray, L. Marple, P. Brunck, and D. Dei Rossi. 1987. Oral bioavailability in the rat of soil bound TCDD. Abstract #PS-40. 7th International Dioxin Symposium, Las Vegas, NV.

341. **Paustenbach, D.J.** 1986. An examination of critical assumptions in health risk assessments of 2,3,7,8-TCDD in contaminated soil. Abstract #1133. Society of Toxicology, New Orleans, LA.

342. **Paustenbach, D.J.** 1986. Critical assumptions in the risk assessment of TCDD in contaminated soil. Abstract #30. 191st National Meeting, American Chemical Society, New York, NY.

343. Sielken, R.L., F.W. Carlborg, **D.J. Paustenbach**, F.J. Murray, and H.S. Shu. 1986. Alternate approaches to mathematically analyzing the bioassay data for 2,3,7,8-TCDD. Abstract #1132. Society of Toxicology, New Orleans, LA.

344. **Paustenbach, D.J.** and J.T. Dufour. 1985. A critical review of cancer risk assessment methodologies. Abstract #219. American Industrial Hygiene Conference, Las Vegas, NV.

345. **Paustenbach, D.J.** and F.J. Murray. 1985. A critical examination of assessments of the health risks associated with 2,3,7,8-TCDD in soil. Abstract #135. 5th International Dioxin Symposium, Bayreuth, Germany.

346. **Paustenbach, D.J.**, et al. 1985. A physiologically based pharmacokinetics model (PBPK) for inhaled CCl4 in the rat. Abstract #288. Society of Toxicology, San Diego, CA.

347. **Paustenbach, D.J.** 1984. Unusual work schedules: an overview. Abstract #112. American Industrial Hygiene Conference, Detroit, MI.

348. **Paustenbach, D.J.** 1983. An environmental risk assessment of an insect growth regulator for control of fire ants. Abstract #224. Society of Environmental Toxicology and Chemistry, Washington, DC.

349. **Paustenbach, D.J.** 1982. Effects of exposure duration on the severity of toxic response. Abstract #188. Society of Environmental Toxicology and Chemistry, Boston, MA.

350. **Paustenbach, D.J.** 1981. A comparative study of the pharmacokinetics and toxic effects of exposing rats to CCl4 during a standard and normal workweek. Abstract #120. American Industrial Hygiene Conference, Portland, OR.

351. **Paustenbach, D.J.** 1981. The need to educate undergraduate engineers in occupational health and safety. Abstract #124. American Institute of Chemical Engineering, New Orleans, LA.

352. **Paustenbach, D.J.** 1980. A unit operations approach to engineering control of health hazards. Abstract #302. American Industrial Hygiene Conference, Houston, TX.

ADVISORY BOARDS TO INDUSTRY

1. Member of the Alcoa Environmental Technology Board of Directors (1993-1996)
2. Member of the Savannah River Environmental Board of Directors (1997-2001)

INVITED LECTURES AND/OR PRESENTATIONS AT UNIVERSITIES

2024: As part of my duties as an adjunct professor at University of Kansas Medical School (Kansas City), I gave one lecture entitled “Case studies in the application of risk assessment to current day problems” (April 2024).

2024: At the request of professors in the toxicology program at the University of Rochester, I gave a lecture entitled “Case studies in the application of risk assessment to current day problems” (Spring of 2024)

2023: Invited lecture at Texas A & M for risk assessment course. Lecture was entitled "Case studies in the application of risk assessment to current day problems" (March 2023)

2023: As part of my duties as an adjunct professor at University of Kansas Medical School (Kansas City), I gave one lecture entitled "Case studies in the application of risk assessment to current day problems" (April 2023).

2022: Invited lecture at Texas A & M for risk assessment course. The lecture was entitled "Case studies in the application of risk assessment to current day problems". (April 2022)

2022: As part of my duties as an adjunct professor at University of Kansas Medical School (Kansas City), I gave one lecture entitled "Case studies in the application of risk assessment to current day problems" (April 2022).

2021: As part of my duties as an adjunct professor at University of Kansas Medical School (Kansas City), I gave two lectures. One on "Understanding the challenges of applying risk assessment to toxicology challenges" and one on "Historical occupational diseases". (November 2021)

2020: Invited lecture at Texas A & M for risk assessment course. The lecture was entitled "Case studies in the application of risk assessment to current day problems". (April 2020)

2019: As part of my duties as an adjunct professor at University of Kansas Medical School (Kansas City), I gave two lectures. One on "Understanding the challenges of applying risk assessment to toxicology challenges" and one on "Historical occupational diseases". (November 2019)

2018: Invited lecture at the University of Florida School of Public Health (Gainesville). Comparing and Contrasting the Four Career Paths Available to Toxicologists (Nov. 25 and 26, 2018)

2017: Invited lecture at the University of Kansas Medical School (Kansas City). Understanding the challenges of consulting in applied risk assessment: four case studies (Sept. 14, 2017) [The Annual John Doull Lecture]

2015: Invited lecture at the University of Washington School of Public Health. Understanding the role of professional consultants in applied risk assessment (Feb 15, 2015)

2014: Lecture at the University of Rochester Medical School. Overview of Risk Assessment and Examples of Some Applications. (Spring 2014).

2014: Participated in the University of Rochester Toxicology Retreat by attending seminar presentations and judging graduate student/post-doc presentations. (Spring 2014).

2013: Presented a couple lectures at the UM School of Public Health. One in the course "Advanced Topics in Risk Assessment" and another on "Famous case studies in health risk assessment" in the basic graduate level course in risk assessment (Fall Semester of 2013)

2012: Presented four different lectures at the UM School of Public Health. One in Advanced Topics in Risk Assessment (Dr. Andrew Maynard was lead professor), and lectures involving five different types of case studies (foods, sediments, medical devices, occupational hazards, and water pollution) (Spring 2012) in the School of Public Health.

2011: Taught a double lecture at the University of Michigan, School of Public Health (Class 550). Current approaches to conducting simulation studies (part I) and ten case studies which convey special lessons in risk assessment (Part II) (Dr. Andrew Maynard's class) (April 10; Ann Arbor, MI) [3.0 hours].

2011: Presented two invited lectures at the University of Michigan. One in a basic toxicology course and another one in a graduate level risk assessment course (Fall of 2011).

2010: Lecture at the University of Michigan, School of Public Health (Class 550). Recent examples of complicated risk assessments which our firm conducted and later published in various journals. (Dr. Maynard's class) (April 7; Ann Arbor, MI).

2009: Lecture at the University of Michigan, School of Public Health (Class 550). An examination of five simulation studies that have changed the way science views the hazards of certain industries. (Dr. Jolliett's class) (March 27; Ann Arbor, MI). Other lectures were given that year in the risk course.

2008: Lecture at the University of Michigan, School of Public Health (Class 550). How risk assessment serves a critical role in litigation: especially with respect to Daubert and Frye hearing about the admissibility of scientific evidence. (Dr. Garabrant's class) (March 12; Ann Arbor, MI).

2007: Lecture at the University of Michigan, School of Public Health (Class 550). Current approaches to conducting simulation and dose reconstruction studies. (Dr. Jolliett's class) (March 15; Ann Arbor, MI).

2007: Presented series of lectures (five or six, 3-hour lectures) at the University of Michigan which provided 50% of the teaching in the risk assessment course SPH 550 for MS and PhD students in this specialty. Was jointly responsible for teaching this course.

2007: Invited lecturer at the Rand School of Graduate studies; graduate risk assessment course). Lecture focused on "Advanced topics in health risk assessment" (Dr. Graham's class). (March 5; Santa Monica, CA).

2007: Invited lecture at the National Aeronautics and Space Administration (NASA) at Ames Research Center (national presentation via internal TV network). Lecture was on "Role of industrial hygienists in conducting simulation and dose reconstruction studies." (February 14; Mountain View, CA).

2006: Invited lecture at the University of Michigan School of Public Health Center for Risk Science and Communication Symposia. The lecture focused on seven examples of risk assessments performed by the government that could have been improved upon. (Dr. Garabrant's class) (September 15-16; Ann Arbor, MI).

2006: Invited lecture at the University of North Carolina, School of Public Health How well conducted exposure assessments can improve epidemiology studies. (Dr. Swenberg's class) (October 23; Chapel Hill, NC).

2006: What Can the Pharmaceutical Industry Learn from Applications of Risk Assessment? A lecture on "Lessons learned over the past 30 years from applying risk assessment to understanding the hazards of chemicals in the environment". Invited presentation at the Institute of Medicine Drug Forum, National Academy of Science. (May 30-31; Washington, DC).

2006: The Roles of Dose Reconstruction and Simulation Studies in Understanding Historical Exposure to Asbestos. Invited lecture at the International Conference on Chrysotile. (May 23-24; Montreal, Quebec, Canada).

2006: Achieving a Sensible Approach to the Management of Contaminated Sites: Current Practices and Issues. Presentation at the International Enviro Conference, (May 9; Melbourne, Australia).

2005: Invited speaker on "Assessing Exposure of Workers Historically Exposed to Benzene" at the Harris-Martin Course on Benzene (September 29-30; Los Angeles, CA).

2005: Invited speaker at the University of Michigan School of Public Health, first annual symposia on Risk, Center for Risk Science and Communication Symposia (September 15-16; Ann Arbor, MI).

2004-2005: Presented guest lectures in the School of Medicine at UC Irvine as a clinical adjunct professor in clinical medicine on the topic of (a) rare occupational disease new to the late 20th Century, (b) occupational diseases which have captured the imagination in 2002-2005 (or where we have better characterized the risks). Double lectures were given in 2004 and 2005 in the classes ran by Dr. Fedoruk and Dr. Dean Baker.

2005: Invited participant on "Faculty Demonstration of the Direct and Cross Examination of the Defense Toxicologist" at the International Association of Defense Counsel 33rd Annual Trial Academy at Stanford Law School (August 5; Palo Alto, CA).

2005: Invited speaker on "A State-of-the Art Review of the Health Hazards Posed by Asbestos and Brake Dust" at a joint meeting of the Chrysotile Association and Russian Institute for Occupational Medicine (June 18; Moscow, Russia).

2005: Invited speaker on "Incorporating Hormesis into Regulatory Decision-Making: Possible Implications to the Water Industry" at the AWWA Research Foundation conference (June 13th; San Francisco, CA).

2005: Chaired a symposium at the national meeting of the Society of Toxicology entitled "Evolving knowledge regarding understanding the health risks to children posed by chemicals in our environment" (March, New Orleans, LA).

2004: Invited speaker on "Evaluation of Chemosensory Effects Due to Occupational Exposures" Round Table Discussion at the "Adversity Workshop" sponsored by the German MAK committee (November 16; Cologne, Germany).

2004: Served on the review committee for a peer-review report from the Environmental Carcinogenesis Division and the National Health and Environmental Effects Research Laboratory, Office of Research and Development, US Environmental Protection Agency (May 10-12).

2004: Invited speaker on "The Theory and Methods of Retrospective Exposure Assessment: An Industrial Hygienist's Point of View" at the Defense Research Institute's Toxic Torts and Environmental Law Seminar (March 18; New Orleans, LA).

2003: Invited speaker on "How the industrial hygiene profession can update the Occupational Exposure Limits" at the Professional Conference on Industrial Hygiene (September 14; Palm springs, CA).

2003: Chaired and presented a short course entitled "Fundamentals of Risk Assessment and Applications of Recent Methodologies to Difficult Problems" at the Society of Toxicology 42nd Annual Meeting (March 9-13; Salt Lake City, UT).

2003: Served as an expert for the National Academy of Sciences for Workshop on USEPA Assessment Factors for Data Quality (January 21; Washington, DC).

2002: Served as an expert on the USEPA Oversight Panel on World Trade Center (October 22-23; New York, NY).

2002: Continuing Professional Education Course at the 17th Annual Conference on Industrial Hygiene, AIHA Academy of Industrial Hygiene, "Dose Reconstruction in the Occupational Setting" (October 1; Cincinnati, OH).

2002: Chaired a one-half day symposium on "Risk Assessment of Dioxins" US-Vietnam Scientific Conference on Human Health and Environmental Effects of Agent Orange/Dioxin (March 3-6; Hanoi, Vietnam).

2001: Chaired a one-day symposium on "Current and future international trends regarding dioxins in foods" 21st Annual International Dioxin Meeting (September 10; Kyongju, South Korea).

2001: Served on Strategic Environmental Research and Development Program (SERDP) and Environmental Security Technology Certification Program of the Department of Defense (August 6-7; Washington, DC).

2000: Served on Governor's task force to evaluate health hazard posed by chromium (VI) in soils (June-August; Sacramento, CA). Resigned prior to public meeting.

2000: Chaired a one-day symposium on current and future international trends regarding dioxins in foods and its impact on trade 20th Annual International Dioxin Meeting (September; Monterey, CA).

2000: EPA Science Advisory Board member on the dioxin reassessment (December 2-3; Washington, DC).

2000: EPA Science Advisory Board member in the role of cost-benefit analysis in environmental decision-making (November 7-8; Washington, DC).

1996: Chaired a one-day national symposium on "Toxicologic and Environmental Hazards Posed by chromium (VI) in the Environment". Sponsored by the University of Massachusetts (March; Newport Beach, CA).

1996: Presented a four-hour professional development short course at the 30th Annual meeting of the Society of Toxicology entitled "Monte Carlo Techniques and Risk Assessment" (March; San Diego, CA).

1995: Expert panel member at the Symposia on Evaluation of the Human Health Components of the Life Cycle Assessment sponsored by the International Life Sciences Institute (May 4-6 and June 6-8; Washington, DC).

1995: Member of the CDC expert panel to evaluate ways to assess bioavailability of mercury in soil (August 29-30; Atlanta, GA).

1995: Presented professional development short course at the annual meeting of the Society of Toxicology entitled "Fundamentals of Health Risk Assessment." 250 members attended (March 4; Baltimore, MD).

1994: Invited peer-reviewer of the new National Academy of Sciences book entitled "Risk Assessment: Science and Policy" (January).

1994: Invited participant in Symposia "The Proper Role of Epidemiology in Regulatory Risk Assessment" at Harvard School of Public Health (October 13-14; Boston, MA).

1994: Member of the CDC Science Advisory Board assigned to review and comment on the toxicological profile for dioxin (August 10; Atlanta, GA).

1993: Invited peer-reviewer of the proposed Canadian Guidelines for Assessing Contaminated Soil (December).

1993: Invited peer-reviewer of the National Academy of Science document regarding their evaluation of the "Role of the National Health Effects Research Institute" (January 2; Washington, DC).

1993: Invited speaker on "State-of-the-Art in Risk Assessment" at the Professional Conference on Industrial Hygiene (October 23; Cincinnati, Ohio).

1993: Invited peer-reviewer of the risk assessment report of the committee assigned to evaluate the Great Lakes Initiatives (July).

1993: Keynote speaker and the only invited representative of U.S. to the 1st International Conference on Ecotoxicology; Melbourne, Australia (October 6-8).

1993: Keynote speaker at the first USEPA workshop on physiologically based pharmacokinetic (PBPK) models and cancer risk assessment (October 18-20; North Carolina).

1993: Presented a professional development course (4-hours) at the annual meeting of the Society of Toxicology entitled "Fundamentals of Health Risk Assessment" (March 4).

1992: Invited participant in USEPA's workshop on environmental exposure assessment (January 21-23; Virginia Beach, VA).

1992: Invited peer-reviewer of Congress' Office of Technology Assessment document "Risk Assessment Research Needs in the 1990s" (December 28; Washington, DC).

1992: Invited speaker and participant at the National Academy of Science symposia on state-of-the-art approaches to exposure assessment held as part of USEPA's Committee on Risk Assessment Methods (February 10 and 11; Washington, DC).

1992: Invited participant in the National Academy of Science workshop on developing ecological risk assessment guidelines (February 26-28; Fairfax, VA).

1992: Invited participant at a meeting of experts who discussed how to prioritize the national environmental agenda using risk assessment (November 10-11; Resources for the Future, Annapolis, MD).

1990: Selected as a charter member of the Council for the Health and Environmental Safety of Soils that operated under the auspices of the International Society for Regulatory Pharmacology and Toxicology (September 1987-1993). Objective of this group was to set national clean-up guidelines/limits for chemically contaminated soils. From 1989-1992, was the co-chairman of the Health Effects Group with Dr. Renate Kimbrough.

1989: Presented a professional development short course (four hours) at the annual meeting of the Society of Toxicology entitled "Fundamentals of Human and Ecological Risk Assessment."

1987: Invited peer-reviewer of USEPA's proposed risk-specific dose for dioxin (December).

1987: Selected as a member of the steering committee for the ACGIH Community Air Guidelines Committee. The objective of the group was to set community air standards for airborne toxics (dissolved in 1988).

1985: Presented a number of lectures at Stanford University as part of one of the first PhD level courses on risk assessment. Several other notable instructors team taught this course (including Warner North, Don Kennedy and Bill Lowrance).

1984: Member of the American Industrial Health Council's Scientific Committee and the Quantitative Risk Assessment Sub-Committee (1982-1984).

1983: A principal reviewer of the 1983 DOHS California Cancer Policy. Represented Chemical Industry Council of California in public hearing dealing with the proposed cancer policy.

1981: President of Indiana Section—American Industrial Hygiene Association (Indianapolis).

PRESENTATIONS AT LEGAL CONFERENCES (AND SOME PROFESSIONAL MEETINGS) [INCOMPLETE]

2025 (November 12). Approaches for Identifying a Virtually Safe Dose (VSD) for Asbestos: The Impact on Toxic-Tort Litigation. Presentation at the DRI Asbestos Medicine Seminar. November 12-14, 2025. Tampa, FL.

2025 (November 13). Mock Direct Examination of a Defense Expert. Presentation at the DRI Asbestos Medicine Seminar. November 12-14, 2025. Tampa, FL.

2024 (April 29). Misconceptions Surrounding PFAS & Drinking Water. The Implications for Public Health & Toxic Tort Litigation. PFAS Environmental Professionals. April 29- 30, 2025. Nashville, TN.

2024 (Jan. 22). Lessons Learned From 20 Years Of Study Of The Dioxins: Can It Help Inform EPA In Setting An MCL For PFOS/PFOA? Shubik Lecture at the Toxicology Forum Meeting. Washington, DC.

2023(Nov. 20). Lack of scientific data supporting claims that mesothelioma rates are increasing in women. Presentation at the DRI meeting on medical aspects of asbestos. Austin, Texas.

2022 (Nov. 17). Addressing claims about take-home exposure using simulations studies and basic risk assessment principles. Presentation at the DRI meeting on medical aspects of asbestos. New Orleans.

2020 (Nov. 15). Why the public doesn't always embrace the views of scientists: Case studies where they should have done so. DRI conference on medicine.

2019 (Nov. 6). A toxicologist's reflections on 35 years of assessing contaminated properties; and what to expect in the coming years. Surplus Property Roundtable (SPR). San Francisco, CA.

2019 (Sept. 22). A toxicologist's reflections on 40 years of work within the pharmaceutical industry. Meeting of the Occupational Toxicology Roundtable (OTC). This is a gathering of 120 toxicologists who work within the pharmaceutical community. Napa, CA.

2019 (Sept. 19). Estimating exposure to talc due to use in cosmetics: Is it really as complicated as court cases seem to indicate? Presentation at joint ISRTP and DRI scientific seminar on talc litigation. Sept. 19 and 20. Washington, DC.

2019. (Sept. 9). Carbon monoxide poisonings related to keyless ignitions: A changing landscape. Presentation with Emma Jacobson and Natalie Holden. Presentation at the DRI annual seminar on litigation and science associated with automotives. Columbus, OH.

2018 (Sept. 7). Ten Fallacies Regarding the Science of Plaintiff's Cases involving Cosmetic Talc. Presentation at the Defense Asbestos Litigation Seminar Conference. Sept. 7, 2017. Austin, TX.

2018 (Sept. 6). Overview of Monticello Meeting of Asbestos Experts (Oct. 15-20, 2017). Presentation at the Defense Asbestos Litigation Seminar Conference. Sept. 6, 2017. Austin, TX.

2017 (June) Asbestos Fiber Transport to Communities: Theory and Practice. DALS meeting. June 6, 2017. Orlando, FL.

2017 (June). Community exposure to trace concentrations of amphibole fibers: Next generation of asbestos claims. Presentation at the Defense Asbestos Litigation Seminar Conference. June 7, 2017. Orlando, FL.

2017 (January). Asbestos, Smoking, Lung Cancer and Synergy: Shortcomings. In Plaintiff's Claims. Presentation at the National Forum on Asbestos Claims & Litigation Conference. January 12, 2017. Philadelphia, PA.

2016 (November). Overcoming Unjustified Fear: Attempts to Bring More Science to The Courtroom. Annual meeting of the DRI. Paustenbach and Rasmussen. Nov. 10. New Orleans, LA.

2016 (November). A Very Brief Historical Overview on The Toxicity of Talc. Presentation at the Defense Research Institute Annual Meeting. November 9, 2016. New Orleans, LA.

2016 (October). Looking into the crystal ball regarding science and litigation with a focus on the railroad industry. Meeting of railroad litigators. Oct. 17, 2016. Kansas City, MS.

2016 (February). Some Suggestion About Derailments that Result in a Fire or Contamination of a Waterbody: Some Aspects Which Can Influence Possible Future Litigation. Annual meeting of railroad litigators. Feb. 20. Sun Valley, ID.

2015 (June). Scientific Testimony "that seems to stick" In Asbestos Litigation. Presentation at the Defense Asbestos Litigation Seminar Conference. June 4, 2015. Las Vegas.

2014 (Sept). Asbestos take-home, fiber settling, and bystander research data. Presentation at Perrin Conference on Sept. 10. San Francisco.

2014 (June). Scientific Testimony "that seems to stick" In Recent Lung Cancer Cases. Presentation at the Defense Asbestos Litigation Seminar. June 4, 2014. Las Vegas.

2013 (November). Factors Which Influence Take Home Exposure of Asbestos. Presentation at the Perrin Asbestos Defense Strategic Summit. November 5, 2013. New Orleans.

2013 (November) Understanding Take Home exposure: Recent advances. Presentation to the Union Pacific Railroads. Nov. 6, 2013) New Orleans, La.

2011 (March). Personal Reflections on Friction Litigation. Presentation at the American Bar Association Meeting. March 31, 2011. Phoenix.

2009 (March). Toxicogenetics and Toxicogenomics: Science Fiction, or the Future of Toxic Torts. DRI Toxic Torts Seminar. March 20, 2009. Phoenix, Ariz.

2009 (March). Manufacturing Doubt and Science: Two fabricated Side Shows that have changed the landscape. DRI Toxic Torts and Env Law Seminar. March 20, 2009. Phoenix.

2009 (March). Clever deception: Judging science by popularity of funding source instead of intellectual content. DRI Toxic Torts and Env Law Seminar. March 20, 2009. Phoenix.

2006 (May). What can the Pharmaceutical Industry learn from the History of Health Risk Assessment? Drs. Paustenbach and Galbraith. Institute of Medicine Drug Forum. May 30- 31, 2006. By invitation. Washington, DC

2003 (March). Invited presentation. Science vs. Politics and Influence: Fact or Fiction. American Bar Association. March 22. Phoenix.

EDITORSHIP

- Associate Editor; *Journal of Toxicological Sciences* (1998-2001)
- Editor-in-Chief; *Journal of Children's Health* (first issue published March 2003)

EDITORIAL REVIEW BOARDS

- *Risk Analysis* (1992-2004)
- *Journal of Toxicology and Environmental Health A* (1994-2011; 2015-Present)
- *Soil and Sediment Contamination* (formerly Journal of Soil Contamination) (1995-2011; 2015-Present)

- *Human and Experimental Toxicology* (England) (1995-2005)
- *Human and Ecological Risk Assessment* (1995-2008)
- *Regulatory Toxicology and Pharmacology* (1993-2011; 2015-2021)
- *Occupational and Environmental Medicine* (1998-2001)
- *Toxicology and Industrial Health* (2002-2011)

UNIVERSITY ADVISORY BOARDS

2025-2026: Served on the Dean's Advisory Board for the new School of Health Sciences (semi-annual meetings in West Lafayette).

2005-08: Served on the Dean's Advisory Board for Purdue University (semi-annual meetings in West Lafayette).

2002-14: Served on the Dean's Advisory Board for the University of Michigan, School of Public Health (semi-annual meetings in Ann Arbor). Remained an advisor to the board through 2014.

1996-98: Member of the Center Director's advisory board for the Harvard School of Public Health, Center for Risk Analysis (Boston, MA) (annual meetings).

RESEARCH AND PROFESSIONAL INTERESTS

- Quantitative Risk Assessment
- Risk Assessment of Carcinogenic and Non-carcinogenic Chemicals
- Fate and transport of types of chemicals in the environment
- Risk Communication
- Cost-Benefit Analyses
- Toxicology of Medical Devices
- Understanding the health risks of various industrial chemicals and metals
- Fate and transport of Airborne Particulates
- Pharmacokinetics of environmental contaminants
- Carcinogenicity of Industrial Chemicals
- Industrial Toxicology
- Environmental Toxicology
- Industrial Hygiene Engineering
- Setting Occupational and Environmental Exposure Limits
- Radon
- Chemical and Environmental Engineering
- Business Ethics
- Indoor Air Pollution
- Industrial Ventilation

- Occupational Health
- Unusual Work Shifts

COMMUNITY SERVICE

2024-2026: Served on the Board of Directors of the American Museum of Folk Art (AFAM) in New York City. Our goal was to create a permanent home for the museum.

2024-2025: At the request of the directors of the Shaker Museum of Enfield, New Hampshire, offered advice as to how to raise funds to restore the village to its original condition.

2017-2024: Co-Chairman of Committee to Establish a Foundation for Permanent Maintenance and Support of Shaker Village. Sabbathday Lake, Maine

2014-2022: Served on the Board of Directors of the Jackson Laboratory (Bar Harbor, Maine). This was an internationally recognized research group who has developed thousands of strains of mice which are used in cancer and other research programs around the world. Assistant chair of the development committee.

2004-2014: Served as the co-chair of the fund-raising committee for the Center for Risk Analysis and Communication for the University of Michigan School of Public Health (Ann Arbor, Michigan). We raised approximately $10M.

2008-2012: Served as advisor with respect to fundraising to the Executive Director (Grainger) of the Peninsula Bridge Program (San Mateo County, CA). This organization is dedicated to providing supplemental educational training to dedicated and gifted minorities between grades 5-8 so as to enhance their opportunities to attend first rate high schools and colleges (when only about 30- 50% of their peers even graduate from high school

2005-2008: Financial supporter and lecturer at the Center for the Study of the American South at University of North Carolina, Chapel Hill. This is the first center based at a major university devoted to preserving the material and musical culture of the South. Harry Watson is the Director.

2005-2008: Fundraising advisor and interim executive director to the Web History Center (Museum) which is dedicated to preserving the history of the evolution of the World WideWeb. Mountain View, California.

2003-2007: Fundraising advisor to the Dean for the University of Michigan Center for Risk Analysis and Communication (Ann Arbor, MI).

2003-2014: Served as an active member of Dean's Advisory Board for School of Public Health at Univ of Michigan (Ann Arbor, MI)

1997-2000: Member of the Bellarmine High School Board to identify the "vision" for the next 30 years. Part of the team responsible for raising $150M to revitalize the entire campus.

1995: Member of Bellarmine High School Board to review performance criteria for Office of the President.

1976-1977: Served as a psychologist/counselor in training (as part of MS degree requirements) at the Indiana State Institution for Juvenile Delinquents (Terre Haute, IN).

OTHER ACTIVITIES

2003: Presented lecture at Heart of County—National Antique Show entitled "The history of the food safe in America: An updated analysis" as part of their symposia on material culture (February).

1995: Presented lecture at Heart of County—National Antique Show entitled "The history of the food safe in America" as part of their symposia on material culture (February).

NON-PEER REVIEWED PUBLICATIONS

1. **Dennis Paustenbach** and David Brew. Speculation v. Fact: The Dangers of the Proposed EPA Risk Assessment on Chrysotile Asbestos. Asbestos Columns. Harris Martin. February 2021.

2. **Paustenbach, D.** 2016. An erosion of credibility: Embracing opinions of scientific or medical experts. For the Defense. April, pages 65-71.

3. Langard, S., B.D. Kerger, and **Paustenbach, D.J.** 1999. Cancer causation and assessment of relative contribution for known causes of human cancers: case examples involving hexavalent Chromium. Hazardous Waste Strategies Update. 10(2): 14-18.

PUBLICATIONS (NON-SCIENTIFIC)

1. **Paustenbach, D.J.** and L. Paustenbach. 1995. Pie safes: American furniture and folk art. Part VI: Safes of the Deep South (Georgia, Alabama, Mississippi, Louisiana, and Arkansas). Antique Review. March.

2. **Paustenbach, D.J.** and L. Paustenbach. 1994. Pie safes: American furniture and folk art. Part V: Safes of Virginia, Tennessee, and Kentucky. Antique Review. January.

3. **Paustenbach, D.J.** and L. Paustenbach. 1993. Pie safes: American furniture and folk art. Part IV: Safes of the Midwestern states. Antique Review.

4. **Paustenbach, D.J.** and L. Paustenbach. 1992. Pie safes: American furniture and folk art. Part III: Geographical differences among pie safes (New England, New York, Pennsylvania, Delaware, and Maryland). Antique Review. March: 21-240.

5. **Paustenbach, D.J.** and L. Paustenbach. 1991. Pie safes: American furniture and folk art. Part II: Various food safes in America. Antique Review. November: 33- 38.

6. **Paustenbach, D.J.** and L. Paustenbach. 1990. Pie safes: American furniture and folk art. Part I: The early history of food safes. Antique Review. August: 29-34.

NEWSPAPER ARTICLES

1. **Paustenbach, D.J.** 2005. The myth of dioxin. Editorial page. Akron Beacon Journal. December 26

APPENDIX A

PARTICIPATION IN PUBLIC MEETINGS

GTX, Inc. (Amelia, LA): In May of 1998, gave a testimony at an evidentiary hearing regarding a permit for a large combustor. The testimony addressed how the modifications to the incinerator were intended to prevent significant quantities of airborne emissions.

Hewlett Packard; Barron Park Site (Palo Alto, CA): This job involved conducting a small risk assessment of contaminated sediment and effect on plant life in Barron Park Creek. In short, solvents had leaked from a contaminated plume into a creek that passed through a residential community in Palo Alto, CA. Upon completion of the assessment in 1992, the results were communicated in a public hearing to more than 150 residents and regulatory agency staff. Due to the poor reception that state and local regulatory agencies had received at previous public hearings, I was asked to present the data and the interpretation at this meeting, which was covered by local television and newspaper reporters.

Southern Wood Piedmont (Augusta, GA): A wood treatment facility was operated in a small town in Georgia for many years. The primary treatment chemical was pentachlorophenol, which is known to be contaminated with 2,3,7,8-TCDD. As a result of the treatment process, a groundwater plume had contaminated a creek that ran adjacent to the facility. We conducted a risk assessment on this site and presented the results at a public meeting in 1991.

Stauffer Chemical (Houston, TX): In 1989, a number of chemical plants along the Houston ship channel had releases of hydrogen sulfide on a number of occasions. Due to the odors associated with this chemical, the local community had significant concern about the possible health effects of the emissions. Stauffer had installed a number of preventive measures as well as on-line continuous monitors for this chemical. Our job was to attend the public meetings wherein the renewal of their operating permit was discussed. As such, we needed to explain a number of scientific issues to a relatively hostile audience.

City of Detroit (world's largest municipal incinerator): In 1991, Mayor Coleman Young of Detroit had run into significant difficulty in maintaining the permits to operate Detroit's municipal incinerators. For a number of years, the Canadian government had expressed concern about the release of airborne chemicals by these incinerators and the drift of the plume toward the Canadian border. Due to dozens of questions which had been raised about the possibility that dioxins were being admitted from the incinerators, Mayor Young and the operators of the incinerators, wanted us to give presentations to the air pollution control district at their next public meeting and to answer the public's questions. I developed some teaching materials for that meeting and answered several hours of questions from the community (in early 1992). Nearly 500 people attended that meeting.

GSF Industries (Calumet City, IN): As a result of an accidental release of a scrubbing agent (selexol) from a gas recycling exhaust system, a number of cars besides this landfill had had their paint pitted. Because nearly 100 autos had been affected, local residents believed that it was likely that the particulate released during the accident would affect their health. Our client asked that we conduct a screening risk assessment and present our findings to residents in the area. In 1990, a public question/answer session took place in a largely poor-minority neighborhood outside Chicago for about 2.5 hours.

Syntex Agribusiness and Region VII USEPA (Springfield, MO): In 1978, dioxin contaminated oils were used as dust control for a number of towns in central Missouri. Most famous among these was Times Beach, Missouri. Although this site received most of the press coverage, a good deal of remediation activities was required at the two manufacturing sites from which the oils originated or were analyzed. As a result of those activities, USEPA and Syntex Agribusiness wanted to conduct remediation activities at both the Verona and the Springfield sites.

Because the townspeople had read literally hundreds of pages of information on dioxin for nearly 15 years, they were quite nervous about living near a remediation activity involving this chemical. At the request of the Region VII Administrator (Mr. Morris Kay), a public hearing was conducted in 1989 to explain the risks to humans and the environmental associated with the upcoming remediation activities. Fortunately, as a result of that meeting, the remediation process began to occur without significant resistance from either the community or local environmental action groups.

Maxus Energy (Kearny, NJ): Between 1890 and 1960 nearly three million tons of chromite- ore processing residue was used as landfills in the meadowlands outside Newark, New Jersey. In the late 1980s the New Jersey Department of Environmental Protection requested that plans be made to assess the health risks posed by the 150 sites where the residues were used as landfill, and which now were predominantly the surfaces of parking lots. After conducting nearly two years of research, Maxus requested that we conduct a public meeting wherein we would answer the questions posed by members of the community, including the Mayor and the Town Council. At least two meetings were held in 1990 - 1992 wherein our work was explained, and questions of the press and other groups were answered.

Occidental Chemical (Atlanta, IL): In the 1970s and 1980s, a pesticide formulation facility operated in a town of about 1,500 people in Illinois. During a remedial investigation of the plant site, it was concluded that persistent pesticides (cyclodienes) may have been distributed from a stack from the baghouse at the facility. The state environmental protection agency and townspeople were seriously concerned by the results of the remedial investigation and consideration was given to evacuating the town.

ChemRisk conducted a series of focused risk assessments to evaluate the potential hazard posed by the soils in the town which had elevated concentrations of several cyclodiene pesticides. Following our work, we were asked to give two town meetings, which were held in the high school gymnasium in 1989. These meetings were conducted jointly with the state environmental protection agency and the fish and game organization.

Appendix B

DOCUMENTS REVIEWED

- Case Documents:
  - Third Amended Complaint
  - Plaintiff's Initial Disclosures
  - Plaintiff's Answers to Defendant JOpen, LLC's First Set of Interrogatories, dated 1/2/2025
  - Plaintiff's Supplemental Responses to Requests for Production of Documents, dated 9/9/2025
  - Plaintiff's Supplemental Answers to Interrogatories, dated 9/9/2025
  - Plaintiff's Expert Reports, produced November 18, 2025
- Third Party Productions
  - St. Tammany Parish Sheriff's Office Incident Report, STPSO000001–18
  - St. Tammany Parish Coroner's Office Records
  - Crime Scene Photos produced by St. Tammany Parish Sheriff's Office
  - TRUEPILL000001–03
  - 911 call made by Kathleen Moller
  - NMS Labs Records
  - St. Tammany Fire District #4, Patient Care Record
  - Paradigm Health System (certification of lack of records)
  - ASPEN0000001–12
  - LIFENET000001–08
  - The following records from the Louisiana Organ Procurement Agency:
    - LNH Chart
    - Audio Recording of call between Kathleen Moller and LOPA Representative
    - Cardiectomy Report
    - Louisiana Organ and Tissue Donor Registry Verification
- HIPAA Compliant Authorization for Release of Protected Health Information/Records
- MOLLER_000023–24
- MOLLER_000025–26

- Obituary, Harmony Anne Moller, https://www.dignitymemorial.com/obituaries/covington-la/harmony-moller-11148925
- *Mother Blames Companies Behind Kratom for Daughter's Death*, LEGAL NEWSLINE, Chris Dickerson, https://www.legalnewsline.com/louisiana-record/mother-blames-companies-behind-kratom-for-daughters-death/article_7c3ae0f1-0293-548c-9556-fd7911ae275e.html (February 28, 2024)
- The following Facebook Posts
    - Mande's Restaurant, dated February 15, 2023, linking Harmony Moller Obituary, https://www.dignitymemorial.com/obituaries/covington-la/harmony-moller-11148925, and comments
    - Kathi Moller's post, dated August 6, 2023, and comments
    - Kathi Moller's post, dated February 8, 2023, and comments
    - Kathi Moller's post, dated April 13, 2024, and comments
    - Kathi Moller's post, dated April 12, 2024
    - Kathi Moller's post, dated December 5, 2023
    - Kathi Moller's post, dated November 12, 2023
    - Kathi Moller's post, dated April 7, 2023
    - Kathi Moller's comment on Mande's Restaurant's post, dated February 15, 2023
    - Kathi Moller's post, dated May 29, 2023, and comments

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Number	Date	Case Name	City/County, State	Alleged Exposure	Client	Trial or Deposition
67	**Nov 03, 2025**	Cary Delaune v. Kinder Morgan Bulk Terminals, LLC, et al. [Regarding Rubicon, LLC] [Alleged Hypersensitivity Pneumonitis]	Metairie, LA	Methylene diphenyl diisocyanate	Rubicon, LLC	Deposition
66	**Oct 30, 2025**	United States Public Interest Research Group Education Fund, Inc. v. Haier U.S. Appliance Solutions, Inc., D/B/A GE Appliances	Chicago, IL	Nitrogen oxides	General Electric	Deposition
65	**Oct 8-9, 2025**	Angeline Brown v. Beverly Hills Properties (BHP), LLC et al. [Regarding BHP] [Proposition 65 – Alleged Failure to Warn to Asbestos]	Los Angeles, CA	Proposition 65	Beverly Hills Properties	Trial
64	**May 28, 2025**	Jerome Golem and Andrea Golem v. 3M Company [Regarding United States Steel Corporation]	Ann Arbor, MI	Asbestos	U. S. Steel	Deposition
63	**May 01, 2025**	Shintech Incorporated v. Olin Corporation and Blue Cube Operations LLC	Chicago, IL	VCM; Chemical Turnatound practices (Chemical Engineering case, not a tox case)	Olin Corporation and Blue Cube Operations LLC	Deposition
62	**Apr 23, 2025**	Lila Maier, Individually and as successor-in-interest to Decedent Donald Maier v. A.W. Chesterton Company, et al.	Los Angeles, CA	Asbestos	Crown Cork & Seal Company	Deposition
61	**Apr 15, 2025**	Mathews v. City of Tacoma	Tacoma, Washington	Asbestos	City of Tacoma	Deposition
60	**Apr 09, 2025**	Shintech Incorporated v. Olin Corporation and Blue Cube Operations LLC	Richmond, VA	VCM; Chemical Turnatound practices (Chemical Engineering case, not a tox case)	Olin Corporation and Blue Cube Operations LLC	Deposition

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59	**Mar 31, 2025**	Steven Wann and Kimberly Wann v. Familian Corp, et al. [Regarding Ford Motor Company]	Walnut Creek, California	Asbestos	Ford	Deposition
58	**Mar 26, 2025**	Five Point Holdings, LLC and Cp Development Co., LLC, v. TETRA TECH, INC. et al., Case No. 3:20-cv-01481-JD CPHP Development, LP, et al., v. TETRA TECH EC, INC., et al, Case No. 3:20-cv-01485-JD Linda Parker Pennington, et al., v. TETRA TECH EC, INC., et al, Case No. 3:18-cv-05330-JD United States of America Ex Rel. Arthur R. Jahr, III, et al, Anthony Smith, & Donald K. Wadsworth, et al, v. TETRA TECH, INC., TETRA TECH EC, INC., et al, Case No. 13-cv-03835	Fort Myers, FL	Radionuclides	Tetra Tech Inc.	Deposition
57	**Mar 07, 2025**	Vera Haney, Plaintiff, v. Advance Stores Company Incorporated, et al. [Regarding Ford Motor Company] [Alleged Pleural Mesothelioma]	Indianapolis, IN	Asbestos	Ford	Deposition
56	**Feb 25, 2025**	Frederick J. Ayers, Jr. and Jo Anne Ayers v. American Biltrite, Inc., et al. [Regarding Crown Cork & Seal Company]	Walnut Creek, CA	Asbestos	Crown Cork	Deposition
55	**Feb 18, 2025**	Phyllis Barnett, as the Personal Representative for the Estate of Daniel Barnett, vs. Air & Liquid Corporation, et al.	King County, WA	Asbestos	Pyrotek Inc.	Deposition
54	**Jan 14, 2025**	Gwendolyn Samons Simpkins, Personal Representative of the Estate of Royce A. Samons, deceased v. Armstrong International, Inc., et al.	Miami, Florida	Asbestos	Ford	Deposition
53	**Jan 08, 2025**	Robert Vivian Rydalch vs. Industrial Supply Company, Inc., et al.	Salt Lake City, UT	Asbestos and Corporate Conduct	Kennecott Utah Copper LLC	Deposition
52	**Dec 16, 2024**	Roy H. Allen v. Exxon Mobil Corporation, et al.	New Orleans, LA	Asbestos	Fireman's Fund	Deposition
51	**Nov 25, 2024**	The State of Alaska, v. JUUL Labs, Inc.	Anchorage, AK	Nicotine	Juul	Trial
50	**Nov 19, 2024**	Shirley A. Johnson, Individually and as Personal Representative of the Estate of Lonnie Stubblefield, Sr. vs. Air & Liquid Systems Corporation, et al. (Regarding Lockheed Shipbuilding Company, LLC)	Seattle, WA	Asbestos	Lockheed Shipbuilding Company, LLC	Deposition

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49	**Oct 28, 2024**	James and Lori Taylor v. Mood Rite, LLC and Cobb Express41, LLC	Dekalb County, GA	Kratom	Mood Rite Kratom	Deposition
48	**Aug 20, 2024**	Brown, et al. v. Affinitylifestyles.com, Inc. d/b/a Real Water, et al.	Clark County, Nevada	Bottled Water	United Natural Foods, Inc.	Deposition
47	**Aug 14, 2024**	Jeffrey Ochoa v. 3M Company, formerly known as Minnesota Mining & Manufacturing Company, et al.	Los Angeles, CA	Asbestos	Ford	Deposition
46	**Aug 13, 2024**	The State of Alaska, v. JUUL Labs, Inc.	Anchorage, AK	Nicotine	Juul	Deposition
45	**Jul 12, 2024**	Emily Everett v. Air Products and Chemicals Inc., et al. [Concerning Level 3 Holdings]	New Orleans, LA	Asbestos	Level 3 Holding	Trial
44	**Jun 21, 2024**	Tristan Rose Perkins, Independent Administratrix of Succession of the Decedent Geraldine Rabb Perkins v. United States of America	Seattle, WA	Asbestos	United States Department of Justice	Trial
43	**Apr 10, 2024**	David S. Van Winkle and Deborah G. Van Winkle v. Akebono Brake Corporation, et al.	Seattle, WA	Asbestos	Arvin Meritor, Osh Kosh and Ford	Deposition
42	**Mar 12, 2024**	Glenn Hagan and Billie Hagan, his wife, v. Advance Stores Company, Inc., f/k/a Advance Auto Parts, Inc., et al. [Regarding Ford]	Key West, Florida	Asbestos	Ford	Deposition
41	**Mar 08, 2024**	Jerome Jones and Sheila Jones vs. Atlanta Sand & Supply Company, et al. (Regarding MSA)	Clinton, MS	Silica	Mine Safety Appliances (MSA)	Deposition
40	**Feb 21, 2024**	Tristan Rose Perkins, Independent Administratrix of Succession of the Decedent Geraldine Rabb Perkins v. United States of America	Washington, D.C.	Asbestos	United States Department of Justice	Deposition
39	**Feb 20, 2024**	Elizabeth K. Davis, Individually and as Ancillary Administratrix of the Estate of Gary S. Davis v. Hesco Parts, LLC, Et al. [Regarding Ford Motor Company]	Prospect, Kentucky	Asbestos	Ford	Deposition
38	**Feb 02, 2024**	Delos M. Blackwell v. Eagle, Inc., f/k/a Eagle Asbestos & Packing Co., Inc., et al. [Regarding Ford]	New Orleans, LA	Asbestos	Ford	Deposition
37	**Jan 25, 2024**	Ward G. Ladd Jr. and Carol W. Ladd vs. Brake Parts Inc. LLC, et al. [Regarding Ford Motor Company]	Washington, D.C.	Asbestos	Ford	Deposition
36	**Jan 03, 2024**	Rockney Monroe and Lisa Monroe vs. Applied Industrial Technologies, Inc., et al. (regarding Pyrotek Inc. and Wagstaff)	Portland, OR	Asbestos	Pyrotek Inc. and Wagstaff Inc.	Deposition

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35	**Dec 19, 2023**	Dorothy J. Jackson, Independent Administrator of the Estate of Thomas J. Jackson, Deceased vs. A. W. Chesterton Co., et al.	Cook County, Illinois	Asbestos and Industrial Talc	Bridgestone Americas, Inc.	Trial
34	**Nov 29, 2023**	Dorothy J. Jackson, Independent Administrator of the Estate of Thomas J. Jackson, Deceased vs. A. W. Chesterton Co., et al.	Akron, OH	Asbestos and Industrial Talc	Bridgestone Americas, Inc.	Deposition
33	**Nov 15, 2023**	Melvin L. Richard vs. Lamorak Insurance Company, et al.	New Orleans, LA	Asbestos	Fireman's Fund	Deposition
32	**Oct 30, 2023**	Hunwardsen et al. v. AffinityLifestyles.com, Inc. et al.	Clark County, Nevada	Bottled Water	United Natural Foods, Inc.	Deposition
31	**Oct 16, 2023**	Sheila Elijah vs. Port Authority Trans-Hudson Corporation	Jersey City, NJ	SARS-CoV-2	Port Authority Trans-Hudson Corporation	Trial
30	**Oct 06, 2023**	James E. Altman, v. A.O. Smith Corporation, et al. [Regarding United States Steel Corporation] [Pleural Mesothelioma]	Cook County, Illinois	Asbestos	U.S. Steel	Deposition
29	**Sep 13, 2023**	Clayton E. Rhodes, Jr. and Donna R. Rhodes vs. 3M Company, et al. (regarding Navistar (f/k/a International Harvester))	Pottawattamie County, IA	Asbestos	Navistar	Deposition
28	**Aug 18, 2023**	Samson Bareh and Gen Bareh, plaintiffs, v. Honeywell International, Inc. et al.	Los Angeles, CA	Asbestos	MBUSA	Deposition
27	**Jun 08, 2023**	Adele Waud, as a Personal Representative of the Estate of William L. Waud, Jr., Pltf. vs. Bennett Auto Supply, Inc., et al.	Miami, Florida	Asbestos	Ford	Deposition
26	**Jun 02, 2023**	Gallagher Set of Plantiffs vs. Affinitylifestyles.com, Inc.	Clark County, Nevada	Bottled Water	United Natural Foods, Inc.	Deposition
25	**Apr 04, 2023**	Jerry Packer vs. Eagle, Inc., et al.	New Orleans, LA	Asbestos	Fireman's Fund	Deposition
24	**Feb 17, 2023**	District of Columbia, by its Attorney General, Karl Racine, vs. Juul Labs, Inc.	Washington, D.C.	Nicotine	Juul	Deposition
23	**Dec 06, 2022**	CNOOC Petroleum North America ULC vs. 801 Seventh Inc., et al.	Calgary, Alberta	Asbestos	801 Seventh Inc.	Trial

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22	**Dec 05, 2022**	CNOOC Petroleum North America ULC vs. 801 Seventh Inc., et al.	Calgary, Alberta	Asbestos	801 Seventh Inc.	Trial
21	**Nov 01, 2022**	State of West Virginia ex rel. Darrell v. McGraw, Jr. Attorney General vs. Minnesota Mining and Manufacturing Company et al.	Lincoln County, West Virginia	Coal Dust	Mine Safety Appliances (MSA)	Deposition
20	**Oct 11, 2022**	State of Minnesota, by its Attorney General, Keith Ellison, v. JUUL Labs, Inc.	Washington, D.C.	Nicotine	Juul	Deposition
19	**Sep 26, 2022**	Michele Felix, as Personal Representative on behalf of the Legal Heirs of Raymond Felix, Deceased, Plaintiff, vs. Asbestos Defendants, Including United States Steel Corporation	Salt Lake County, Utah	Asbestos	U.S. Steel	Deposition
18	**Aug 23, 2022**	Leonard "Eric" Olson, as Executor of the Estate of Gary D. Olson, Plaintiff, vs. Air & Liquid Systems Corporation, et al.	King County, WA	Asbestos	Pyrotek Inc.	Deposition
17	**Aug 17, 2022**	Jose Diaz and Maria Diaz vs. Barretts Minerals, Inc., et al.	Los Angeles, CA	Asbestos	P.W. Stephens and Fireman's Fund	Deposition
16	**Jul 21, 2022**	Joanne Clement Reulet, et al vs. Lamorak Insurance Company, et al.	Middle District of Louisiana	Asbestos	Fireman's Fund	Deposition
15	**Jun 28, 2022**	Richard J. Boetsch, Plaintiff, vs. A.W. Chesterton Company, et al.	Los Angeles, CA	Asbestos	Ford	Deposition
14	**Jun 17, 2022**	Richard J. Boetsch, Plaintiff, vs. A.W. Chesterton Company, et al.	Los Angeles, CA	Asbestos	Ford	Deposition
13	**May 17, 2022**	Shirley Tubandt vs. Asbestos Corporation Ltd., et al.	King County, WA	Asbestos	Crown Cork	Deposition
12	**Mar 18, 2022**	Kimberlee Trishell Winder, Individually and as Personal Representative of the Estate of Stephen Allan O'Neal, deceased vs. Jerry Seiner Chevrolet, Inc., et al.,	Salt Lake County, Utah	Asbestos	Ford	Deposition
11	**Mar 11, 2022**	Jerry V. Campbell and Darlene R. Campbell, Plaintiffs vs. Armstrong International, Inc., et al.	Richland County, SC	Asbestos	Southern Insulation	Deposition

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10	**Mar 04, 2022**	Susan W. Moore and Christopher Moore vs. Criswell Chevrolet, Inc. et al.	Baltimore City, MD	Asbestos	Navistar	Deposition
9	**Feb 25, 2022**	Gregory Sullivan and Janey Sullivan, his wife vs. Air & Liquid Systems Corporation, et al.	Broward County, Fl	Asbestos	Navistar	Deposition
8	**Feb 09, 2022**	Francis M. Barnum, Jr. and Laurie Barnum vs. Autozone West, Inc. et al.	Los Angeles County, CA	Asbestos	Ford	Deposition
7	**Jan 14, 2022**	Joan P. Lee vs. A.O. Smith Corporation, et al.	Spokane County, Washington	Asbestos	Spokane Industries	Deposition
6	**Dec 10, 2021**	United States District Court, Northern District of California, Consolidated Class Action vs. Juul Labs, Inc.	Northern District of California	Nicotine	Juul	Deposition
5	**Dec 07, 2021**	Tommy D. Wannamaker, and Dale J. Wannamaker vs. 3M Company et al. & Stephen C. Horton and Gloria J. Horton v. Armstrong International Inc., et al.	South Carolina (Orangeburg County) & South Carolina (Richland County)	Asbestos	Southern Insulation	Deposition
4	**Oct 06, 2021**	Marian Murphy Dudley, as Executor of the Estate of Nicholas Leon Murphy, Deceased and Doris Anne Murphy, Individually vs. Covil Corp., et al.	Richland, SC	Asbestos	Southern Insulation	Deposition
3	**Jun 29, 2021**	Salvador Maciel and Laura Maciel, his wife, Plaintiffs vs. A.W. Chesterton Company et al.	Madison County, IL	Asbestos	U.S. Steel	Deposition
2	**Apr 06, 2021**	Kimberly Spitz, as Executor of the Estate of Arthur G. Whelan, and individual heirs of the Estate of Arthur G. Whelan vs. A.O. Smoth Corporation. et al.	Middlesex County, NJ	Asbestos	Ford	Deposition
1	**Feb 19, 2021**	State of North Carolina, ex rel. Joshua H. Stein, Attorney General vs. Juul Labs, Inc.	Durham County, NC	Nicotine	Juul	Deposition