

EXHIBIT 11

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**UNITED STATES DISTRICT COURT
EASTERN DISTRICT OF LOUISIANA**

KATHLEEN MOLLER,

Plaintiff,

v.

**MARTIAN SALES, INC.; JOPEN, LLC;
JOHNSON FOODS, LLC; LP IND.,
LLC; CAG HOLDINGS, LLC; RMH
HOLDINGS, LLC; ABC INSURANCE
COMPANY and John Does 1-4,**

Defendants.

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CIVIL ACTION NO.

2:24-CV-228-WBV-DPC

THE HONORABLE WENDY B. VITTER

**MAGISTRATE JUDGE DONNA PHILLIPS
CURRAULT**

EXPERT REPORT OF EDWARD W. BOYER, MD PH.D

1. I have been asked by Kilpatrick Townsend & Stockton to review materials relevant to the medical event leading to the death of Harmony Moller on February 6, 2023, and to formulate opinions as to the cause of this event. I have therefore applied my education, training, and experience as a board-certified medical toxicologist and emergency medicine physician to address the role played by kratom, if any, in Ms. Moller's death on February 6, 2023. My fees for reviewing these materials, preparing this report, and testifying at trial are \$750/hour. My compensation is neither contingent on the outcome of this matter nor the conclusions reached in this report.

2. In preparing this report, I have relied on my personal academic and clinical expertise, some of which is described in the following paragraphs. I have attempted to highlight areas that are specific to my expertise in this case, and which permit me as a board-certified medical toxicologist to review, analyze and form the basis of my conclusions within a reasonable degree of medical certainty.

3. A copy of my current *curriculum vitae* is attached as Appendix A. A list of materials considered is attached as Appendix B.

QUALIFICATIONS

4. I am a board-certified emergency physician. Emergency medicine is an Accreditation Council for Graduate Medical Education (ACGME)-approved medical specialty that focuses on the diagnosis and management of any life-threatening condition. Emergency medicine practice, because of its intimate involvement with acute medical conditions, requires that the practitioner integrate patient complaint; presentation of illness; medical, family and social history; knowledge of anatomy; and the impact of physiology on structure and function of the

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human body to arrive at an accurate diagnosis and deliver targeted interventions. Emergency physicians utilize a broad range of tests, including ultrasound, echocardiograms, radiology, and laboratory studies, to arrive at diagnoses and to guide therapy. I have over 25 years of clinical practice experience (including internship, residency, and subspecialty training) in which I have treated hundreds of patients with acutely life-threatening conditions ranging from vascular catastrophes (e.g., aortic structural abnormalities and failure), heart attacks, strokes, and sepsis, among other conditions. As a result of my diagnostic skills, I was recognized as Master Physician by Brigham and Women's Hospital of Harvard Medical School.

5. I am a board-certified medical toxicologist. Medical toxicology is an Accreditation Council for Graduate Medical Education (ACGME)-approved medical subspecialty that focuses on toxicity, poisoning, substance abuse, overdose, adverse effects, and drug interactions from all manner of potential intoxicants, including dietary supplements, herbal products, and medications. As a medical discipline, medical toxicology is particularly concerned with the adverse effects of psychoactive substances. A significant portion of medical toxicology practice and experience is related to the clinical effects of substances with mu-opioid receptor ("MOR") activity such as kratom and other psychoactive substances, as well as the signs, symptoms and outcomes of diversion, misuse, abuse and withdrawal of each of these agents.

6. I have over 25 years of clinical practice experience (including internship, residency, and subspecialty training) in which I have treated several thousand patients with a wide variety of adverse drug events, drug interactions, toxicities, and withdrawal symptoms. I have treated anticipated and unanticipated side effects of xenobiotics, including prescription medications, illicit substances, illegal drugs, dietary products, herbal remedies, and novel psychoactive substances, alone and in combination with each other. As a clinician focusing on the treatment of adverse effects of xenobiotics, I have treated several hundred patients with adverse effects, drug interactions, and toxicity from all manner of MOR agonists, including kratom. In particular, I have considerable clinical experience directly with kratom.

7. I have served on the medical staff of UMass Memorial Medical Center (2001-2016), Brigham and Women's Hospital (2016-2022), the Toxicology Service at Children's Hospital, Boston (1999-2021), and the Regional Center for Poison Control and Prevention Serving Massachusetts and Rhode Island (1999-2021). I now serve on the medical staff of The Ohio State University Wexner Medical Center (2021-present), Nationwide Children's Hospital (2024-present), and the Central Ohio Poison Control Center (2024-present).

8. I was a 2018 Fulbright Scholar funded to study the pharmacophysiologic outcomes in kratom users in Malaysia. In addition, I have received funding from the National Institute on Drug Abuse, part of the National Institutes of Health ("NIH"), to conduct original research investigations related to opioid analgesic medications (2001-present). I have also served as an NIH-funded research mentor for international trainees from Thailand, Malaysia, and Indonesia on kratom use. My research studies examine psychoactive substance use (including kratom), opioid abuse, innovative technologies, and HIV.

9. In addition, I have served as an expert consultant to the Community Epidemiology Working Group, the National Institute on Drug Abuse organization that monitors trends in

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substance use, including kratom (2004-2007). I have served on the scientific advisory board for the National Drug Early Warning System (2016-2020). I have conducted research for National Institutes on Drug Abuse subcontractors related to opioid analgesic overdose, toxicity, misuse, and abuse (2004).

10. I have also served as a research mentor directing mentees performing a wide variety of investigations in several scientific and medical specialties. My doctorate in synthetic organic chemistry prepared me for my postdoctoral investigations into protein design; in this area I studied the structure, cellular handling, folding, organization and activity of cellular proteins. My research supervision and mentoring has spanned investigations into cardiology, cardiac remodeling, cardiac arrest management, cardiac arrhythmia detection, and hepatology. My research mentoring work has also extended to behavioral science, addiction medicine, infectious disease, injury prevention, medical toxicology, and emergency medicine studies.

11. Because of my expertise, I have been invited to deliver expert addresses including lectures on kratom and the clinical effects of alkaloid components of kratom at national conferences, including the American Society of Addiction Medicine (2009), College on Problems of Drug Dependence (2018), Northeastern University's conference on Chemistry and Pharmacology of Drug Abuse (2019), to members of the United States Congress, and the University of Florida's International Kratom Symposium (2025). In 2024 I was invited to author the Kratom card for UpToDate, the leading medical information database. Finally, I was the 2008 NIH Distinguished Scientist in Natural Products Neuroscience for my formative work describing the clinical effects of kratom.

12. I have extensive experience in assessing the quality of medical literature as well as in determining whether published findings apply to specific patient care questions. I served on the Massachusetts Medical Society's Committee on Publications, which oversees the Editorial Board of New England Journal of Medicine (2016-2021). I have served as associate editor for the Journal of Medical Toxicology and been a member of the editorial board of ToxED, an online clinical database resource that assists clinicians in identifying adverse effects of numerous substances. In this capacity, I have reviewed the literature describing the toxicity, adverse events, and drug interactions of numerous substances, including kratom.

13. I have considerable expertise in the assessment of the scientific validity of research investigations. I have participated extensively in the NIH scientific review process—one of the most rigorous and objective scientific review processes in existence. I am a member of the College of CSR Reviewers, and in my service with NIH Scientific Review Groups I have examined grant applications submitted to NIH that are related to behavioral and social science, substance abuse, and HIV (2005-present). The FDA recognizes my experience in medical toxicology and invited me to review the scientific validity of applications for funding of clinical trials (2004, 2022). I have also assessed the scientific validity of grant applications submitted to the US Centers for Disease Control ("CDC") related to HIV testing in emergency departments (2006). Related to these activities are my service as an expert reviewer for journals such as New England Journal of Medicine; Neuropsychopharmacology; Drugs, Drug and Alcohol Dependence; American Journal on Addictions; Academic Emergency Medicine; Journal of Medical Toxicology; Chest – CRITICAL Care Medicine; Clinical Toxicology; Journal of Forensic Science; and Forensic

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Science International, among others.

14. I have expertise in the toxicology of MOR agonists including mitragynine, 7-hydroxymitragynine, mitragynine pseudoindoxyl, and other kratom alkaloids. This experience is drawn from education in medical school, internship, residency, post-graduate work, editorial assignments, continuous reading, teaching, and postgraduate instruction. I supervised the University of Massachusetts Medical Toxicology fellowship training program (2002-2015), where I was responsible for teaching fellows, residents, and clinical pharmacists from six different academic medical centers the adverse effects, drug interactions, and toxicity of all manner of potential intoxicants. I was a member of the Division of Toxicology of Harvard Medical School (2016-2021). In addition, I served as a core faculty member of the Brigham and Women's Hospital Department of Psychiatry Training Program in Addiction Medicine (2020-2021). I am now Professor with Tenure at The Ohio State University and a member of the staff of the Central Ohio Poison Control Center.

15. As part of these academic assignments, I teach basic and applied medical toxicology to medical students, residents, fellows, physicians-in-practice, behavioral science practitioners, Doctor of Pharmacy students, and pharmacy practice residents. I have taught courses on clinical presentations and outcomes of overdose at the Boston and Worcester campuses of the Massachusetts College of Pharmacy (1999-2003), and the Central Ohio Poison Center (2023-present). I have been invited to lecture to psychiatry physicians-in-training at Harvard Medical School teaching hospitals on pharmacology-related topics, including kratom toxicity (2005, 2019). As an instructor in CME courses offered by Harvard Medical School and Albert Einstein College of Medicine, I lectured on adverse effects of drugs with central nervous system ("CNS") activity, substance abuse, and addiction.

16. I have published in peer-reviewed journals. In particular, my work related to kratom has appeared in *Addiction*, *Drug and Alcohol Dependence*; *American Journal on Addictions*; *Journal of Medical Toxicology*; *Current Topics in Medicinal Chemistry*; *Journal of Psychoactive Drugs*, *Journal of Addictive Diseases*, *Frontiers of Psychiatry*, *Journal of Toxicology and Pharmacology*; *Malaysian Journal of Medicine and Health Sciences*; *International Journal of Drug Policy*; and *Journal of Ethnopharmacology*. My other original research, literature reviews, case reports, and perspectives have been published in the *New England Journal of Medicine*; *Journal of the American Medical Association ("JAMA")*; *Pediatrics*; *Drug and Alcohol Dependence*; and *Academic Emergency Medicine*, among others. Those articles have covered a range of issues pertaining to drug toxicity, substance abuse, drug side effects, and behaviors of patients with chronic pain.

17. My general understanding of toxicology is based upon the length and scope of my graduate and post-graduate education, including the study of scientific methodology at Vanderbilt and Columbia Universities, pre-clinical pharmacology and neurosciences as well as clinical courses at Columbia College of Physicians and Surgeons, and the scope of my post-graduate training in medical toxicology at Children's Hospital Boston/Harvard Medical School. My ongoing study of medical toxicology continues with literature assessment and epidemiological analysis, including those instances where medications are involved in overdose and toxicity.

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18. In my work as an academic physician, research scientist, and practicing toxicologist, I have compiled and analyzed data related to chronic pain and opioid analgesic overdose, toxicity, misuse and abuse, as well as the pharmacology and toxicology of numerous pharmacologic substances. This work has enabled me to render conclusions about the likely factors that explain, to a reasonable degree of medical certainty, the toxicology of substances such as kratom, alone and in combination with other substances.

19. Since 2021, I have previously testified as an expert witness in the following matters:

- a. *Denise Zane, as Special Administrator of the Estate of Lucille Stroh vs. Jeanne Wilson, MD, Rehabilitation Specialists Of Chicago, LLC, and The Ingalls Memorial Hospital*, Circuit Court of Cook County, Illinois County Department, Law Division. No.: 13 L 5182;
- b. *United States of America, Plaintiff vs Nine2Five LLC, Sebastian Guthrey*, Case No. 23-cr-1079-TWR (S.D. Cal.);
- c. *Susan Weber v. OPMS a/k/a Optimized Plant Mediated Solutions.*, Case No 23EV000485, ongoing in the State Court of Fulton County, Georgia;
- d. *Susan Eppard, et al. v. JML Wholesale, Inc., et al.*, Case No. 24-004438 – NP, Circuit Court of Muskegon County, Michigan;
- e. *John Zheng Yan Ching v. White Dragon Botanicals, LLC, et al.*, Case No. CVPO21-0198083, Travis County, Texas; and
- f. *National Collegiate Athletic Association v. Jennifer Finnerty*, Case No. 49D01-1808-CT-33896, Marion Superior Court, Indiana.

BACKGROUND

Kratom

18. Kratom (*Mitragynia speciosa* Korth) is a tree-like plant native to Thailand, Malaysia, Indonesia, and other parts of southeast Asia. Indigenous users create a “juice” by boiling fresh kratom leaves for several hours that is then diluted with hot water to make kratom tea. Kratom is used in traditional environments to treat diarrhea, hypertension, cough, fever, impotence, and other ailments. Kratom is used by manual laborers for its mild stimulant effects, a replacement remedy for individuals with problematic opioid use, and as a mild analgesic agent. In the west, many different kratom formulations are available, including tinctures, extracts, capsules, softgels, concentrated products, as well as dried leaf powders and cut leaf material.

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Kratom in western countries is most commonly used as a remedy for opioid addiction, chronic pain, depression, and anxiety.

19. The neurobehavioral effects of kratom are more subtle than the commonly described stimulant effects at low doses and opioid effects at higher doses. Low doses of kratom increase alertness, energy, and talkativeness. At doses greater than 5 grams, however, kratom users describe the persistence of heightened alertness coupled with analgesia. These effects account for the use of kratom as a mild stimulant for manual laborers, a replacement remedy for individuals with problematic opioid use, and as a mild analgesic agent.

20. Some of the biological effects of kratom arise from binding of alkaloidal components to a number of neurotransmitter receptors in the central nervous system, including the μ -opioid (MOR), κ -opioid, serotonergic (5-HT), and alpha (α 1) receptors, among others. MOR activity is particularly important. MOR activation mediates pain, respiratory rate, gastrointestinal transit time, nausea, and pruritis, among other effects. Each of these effects is mediated via distinct signaling pathways following binding of an agonist to the MOR receptor.

21. Classical opioid agonists (*e.g.*, morphine and fentanyl) are “unbiased” agonists in that they indiscriminately activate all biological effects mediated by MOR. Kratom is different. The most prevalent—and best studied—alkaloid in kratom is mitragynine. In contrast to classical unbiased MOR agonists, mitragynine is a “biased” agonist in that it produces some, but not all, effects that arise from receptor binding of a classical MOR agonist such as morphine or fentanyl.

22. For example, while mitragynine produces analgesia, it does not produce the stupor characteristic of classic, unbiased MOR agonists. Instead, kratom produces increased alertness. Furthermore, mitragynine does not produce the respiratory depression that is a leading cause of mortality from classical unbiased MOR agonist overdose (Macko 1972; Varadi 2016; Kruegel 2019; Boyer 2012).

23. In fact, the clinical features as well as the time course of the toxidrome from kratom has never been described. Testing for mitragynine is generally limited to commercial forensic companies and medical examiner offices. Mitragynine itself undergoes postmortem redistribution; the postmortem concentration of mitragynine measured by forensic laboratories is over three times higher than premortem blood.

24. In general, adverse events attributed to kratom have been derived from database studies, poorly controlled case reports, or small case series, each of which has serious deficiencies in patient background information, including medical, social, or family history; clinical evaluation; scene findings; or analytical technique. These poorly controlled studies at times associate kratom with agitation, seizures, liver injury, renal injury, ventricular dysrhythmia, and respiratory depression. Individually and collectively, these studies lack the scientific heft to establish causation between kratom use and any adverse event.

25. For example, case reports describing right ventricular failure in the setting of intermittent kratom ingestions are complicated by the distant use of kratom, the lack of clarity

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regarding intensity of kratom use, analytical testing to confirm kratom use, and the concomitant delivery of multiple interventions.

26. Virtually all adverse events associated with kratom use have occurred in individuals who can be proven to have taken substances in addition to kratom, or who have an underlying medical condition predisposing them to medical events attributed to kratom. The most systematic examination of the relationship between specific medical outcomes and kratom has been done in a large cohort of Malaysian kratom users who have undergone a number of medical tests to identify effects arising from kratom use.

Case Synopsis

27. Harmony Moller had no known significant issues in her medical history, though she used cannabis and ethanol.

28. On or about February 5, 2023, she complained that she did not feel well and developed vomiting. The next day, Ms. Moller collapsed and was found by her mother (Kathleen Moller), who called 911. First responders found Ms. Moller in cardiac arrest. Paramedics intubated Ms. Moller and administered naloxone with no effect. Paramedics made a field declaration of death.

29. Ms. Moller's autopsy was performed on February 8, 2023. The examining pathologist identified areas of nutmeg liver "consistent with chronic passive congestion." In thorax, the bronchi contained blood, and the pathologist noted that the "[p]ulmonary cut surfaces demonstrate hemorrhage, congestion, and edema." From her heart, the pulmonic valve was harvested for transplant, but the remainder was not used (St. Tammany Parish Coroner Records).

30. Ms. Moller's heart was shipped to the Jesse E Edwards Registry of Cardiovascular Disease in St Paul, MN. Pathologists at that facility identified an enlarged heart (cardiomegaly) with hypertrophy of both ventricles (mild biventricular hypertrophy) with mild dilation of the right ventricle. In the left ventricle, the pathologist identified that the left coronary artery "arises 5 mm above the left sinotubular ridge with an acute angle of origin. There is an inferior aortic wall flap over the origin of the left coronary artery." (Jesse E Edwards Registry of Cardiovascular Disease records). In addition, the left ventricular wall was thickened at 15 mm and the intraventricular septum was 17 mm.

31. On microscopy, pathologists noted hypertrophy of atrial and ventricular myocytes, with increased right ventricular interstitial and perivascular fibrosis. The posterior wall of the right ventricle contained hypertrophic myocytes. While the AV node was normal, the ventricular septum and the lateral wall of the left ventricle are hypertrophic with scattered hypereosinophilic myocytes. Interstitial and perivascular fibrosis were present. The anterior wall of the left ventricle had hypertrophic myocytes with interstitial and perivascular fibrosis.

32. Postmortem analyses were performed by two different laboratories. National Medical Laboratories measured a mitragynine concentration of 150 ng/mL, along with caffeine, naloxone, cannabis/metabolites, diphenhydramine, and promethazine (NMS Laboratories

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documentation). The Gerona lab at UCSF, as part of the DEA TOX program, measured the mitragynine concentration as 158 ng/mL with a concentration of 7-hydroxymitragynine that was below the lower limit of quantification (Clinical Toxicology and Environmental Biomonitoring Laboratory Report dated 4/18/2023).

33. The St. Tammany Parish medical examiner assigned a final diagnosis of abnormally high left coronary ostia takeoff exacerbated by mitragynine use.

BASES FOR OPINION

34. To form the basis of my expert opinion, I relied on all of the information forwarded to me as described in the section under document review. From my personal review, including my careful examination of Ms. Moller's medical, social, and family history as well as the pharmacology and medical toxicology of kratom, I am able to support the factual basis for the following statements:

- 35. Harmony Moller had no known significant issues in her medical history;
- 36. Harmony Moller ingested dietary supplements, including ashwagandha and caffeine;
- 37. Harmony Moller used cannabis and alcohol; and
- 38. Harmony Moller vaped.

In addition:

39. Harmony Moller on the day before her death felt ill and began vomiting (St. Tammany Parish Sheriff's Office records);

40. Death scene investigators found in Harmony Moller's bedroom cannabis products, drug paraphernalia, multiple empty liquor bottles, and vaping devices; and

41. Death scene investigators also identified dietary supplements and ayurvedic remedies.

Furthermore,

42. Postmortem examination revealed a high takeoff of the left coronary artery with an acute angle of origin;

43. Postmortem examination also revealed an aortic wall flap covering the opening of the left coronary artery;

44. Postmortem examination identified cardiac hypertrophy;

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45. Harmony Moller also had chronic liver congestion;

46. Histological analysis of the heart identified that Harmony Moller had interstitial and perivascular fibrosis, hypertrophic myocytes, and hypereosinophilic myocytes;

47. Organ procurement specialists harvested only the pulmonic valve from Harmony Moller's heart;

48. Organ procurement specialists declined to utilize the harvested aortic valve;

49. Pathologists initially identified the cause of death as "chronic congestion of liver" (Southern Eye Bank Donor Information worksheet).

Additionally,

50. Kathleen Moller reported on social media that Harmony Moller's death was "caused by a cardiac anomaly that caused her heart to suddenly stop beating from lack of blood flow in a cardiac artery."

51. Another listed cause of death was "chronic congestion of liver";

52. Harmony Moller's blood mitragynine concentration was measured by two separate toxicology labs at 150ng/mL and 158 ng/mL; and

53. The medical examiner determined Ms. Moller's cause of death was due to her high takeoff of the coronary artery complicated by mitragynine use.

However,

54. Harmony Moller had several medical conditions and behaviors that could have produced sudden cardiovascular collapse;

55. According to the medical examiner, Harmony Moller had a high takeoff of the left coronary artery with an acute angle of origin;

56. A high takeoff of the coronary artery with an acute angle of origin is associated with sudden cardiac death;

57. A high takeoff of the coronary artery with an acute angle of origin is associated with sudden cardiac death, even in the absence of mitragynine;

58. Harmony Moller had an inferior aortic wall flap overlying the origin of her left coronary artery that had an acute angle of origin;

59. An aortic wall flap can further limit blood flow into the opening of the left coronary artery, even in the absence of mitragynine;

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60. Occlusion of the coronary artery decreases blood flow through the coronary artery, even in the absence of mitragynine;

61. Decreased blood flow through the coronary artery leads to myocardial hypoperfusion, even in the absence of mitragynine;

62. Decreased blood flow to the heart leads to lethal cardiac arrhythmias, even in the absence of mitragynine; and

63. Lethal cardiac arrhythmias cause sudden cardiac death.

In addition,

64. Harmony Moller used dietary supplements that contained caffeine and ephedrezine (a proprietary product that includes yohimbine and the methylxanthines caffeine and theobromine, among others);

65. Caffeine increases heart rate;

66. Caffeine increases aortic stiffness;

67. Caffeine increases aortic blood pressure; and

68. Harmony Moller died from her congenital heart disease complicated by caffeine use.

Finally,

69. The toxidrome of kratom has not been determined;

70. Mitragynine undergoes postmortem redistribution;

71. The peripheral blood concentration of mitragynine approximately triples in the period between death and measurement at autopsy;

72. A postmortem blood mitragynine concentration of 150 ng/mL or 158 ng/mL is not elevated; and

73. Harmony Moller did not die because of her kratom use.

CONCLUSIONS

74. Based upon the information provided to me, I am able to assert my conclusions to a reasonable degree of medical and toxicologic certainty.

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75. No evidence supports the contention that Harmony Moller’s use of kratom contributed to her death. The postmortem concentration of mitragynine identified in Ms. Moller’s blood was 158 ng/mL as measured by the Gerona lab (St. Tammany Parish Coroner records); commercial clinical laboratories measured the mitragynine concentration as 150 ng/mL (St. Tammany Parish Coroner records). Mitragynine undergoes postmortem redistribution, a process where the concentration of a substance changes following death. The available scientific literature demonstrates that the postmortem concentration of mitragynine is approximately triple that of pre-mortem measurements (Osawa 2025). To a reasonable degree of medical certainty, therefore, Ms. Moller’s premortem concentration of mitragynine was approximately 50 ng/mL, a value that occurred in human clinical trials involving kratom. Notably, none of the participants in these studies developed cardiac toxicity, much less a life-threatening event. Ms. Moller did not die from her kratom use.

76. In fact, medical examiners initially noted that the cause of death was “chronic liver congestion.” Chronic liver congestion is a medical condition often associated with right heart failure; backup of blood (i.e., “congestion”) into the liver produces fibrotic changes that become apparent even on visual inspection of the liver. Notably, kratom has not been associated with chronic liver congestion, even among patients where right heart failure was putatively associated with distant and undefined kratom ingestion (Alameh 2025).

77. Although I agree with the medical examiner’s finding that Ms. Moller died from complications of her unrecognized congenital heart disease—that has a recognized association with sudden cardiac death—I find that the medical examiner errs in failing to consider adequately the pharmacophysiology of the full range of substances identified on postmortem testing.

78. I find that the medical examiner errs in determining that Harmony Moller’s kratom use contributed to her death. In fact, the pharmacophysiology of kratom is more likely to be *protective* against cardiac sudden death in a patient such as Ms. Moller who had a high takeoff of the coronary artery with an acute angle of origin complicated by an inferior aortic flap. Following ingestion, mitragynine exerts parasympathetic effects on the cardiovascular system (Saengmolee 2022; Prevete 2024). These effects, measured generally as a slight *decrease* in systolic and diastolic blood pressure even in the setting of greater alertness, are insufficient to contribute to morbidity and mortality of an individual with high takeoff of the coronary artery with an acute angle of origin of the coronary artery complicated by an inferior aortic flap (Saengmolee 2022; Prevete 2024).¹

79. I also find that the medical examiner errs in ignoring other substances more likely than mitragynine to worsen the congenital heart disease described in the medical record. Normally, the aorta has “elasticity,” meaning it can dilate slightly during a pulse of blood from the heart. Harmony Moller ingested caffeine. Caffeine is an over-the-counter stimulant that increases pulse rate and intra-aortic blood pressure, but caffeine also limits the elasticity (i.e., how much the blood vessel can expand in response to increased blood pressure) of the aorta. (Mahmud 2001,

¹ Ms. Moller’s mother also initially believed that her congenital heart disease alone caused her death. (Mande’s Restaurant Facebook account: <https://www.facebook.com/mandesrestaurant/posts/647328647396846>).

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Vlachopoulos 2003)). Caffeine's pharmacologic actions make it dangerous in a person with high takeoff of the left coronary artery with an acute angle of origin. Caffeine increases aortic blood pressure and decreases aortic elasticity. These direct pharmacologic effects of caffeine compress the opening of the coronary artery precisely because the coronary artery has an acute angle of origin that narrows as the aorta expands (Virmani 1984). Moreover, the inferior aortic flap is an additional obstruction to blood flow into the coronary artery as the increased aortic blood pressure caused by caffeine presses it against the wall of the aorta. Ms. Moller's caffeine use occluded the coronary artery and hindered blood flow into heart muscle. This decrease in cardiac perfusion caused a malignant arrhythmia culminating in sudden cardiac death. Caffeine, not mitragynine, exacerbated Ms. Moller's congenital heart disease and contributed to her death.

80. Even if Ms. Moller was not taking her caffeine supplements, she also had "Mini Thins," a weight-loss remedy. This product contains caffeine and yohimbine, among other plant products. Yohimbine is a presynaptic alpha-2 antagonist. The pharmacologic effects of yohimbine are to increase pulse rate and blood pressure. These direct pharmacologic effects of yohimbine, either with or without caffeine, compress the opening of the coronary artery precisely because the coronary artery has an acute angle of origin that narrows as the aorta expands (Virmani 1984).

81. Even if Harmony Moller did not die of her congenital heart disease complicated by her caffeine use, she had other, more plausible causes of death than mitragynine toxicity. For example, Ms. Moller's vomiting was so severe that Kathleen Moller administered multiple antiemetics without improvement. Harmony Moller was taking two different caffeine-containing supplements. Caffeine is a methylxanthine. The methylxanthine toxidrome involves vomiting, seizure, and ventricular dysrhythmias (Shimane 2017). Death in methylxanthine-poisoned individuals arises from seizures or from malignant ventricular dysrhythmias (Shimane 2017; Poussel 2013). The medical examiner failed to quantify the postmortem concentration of caffeine even though Harmony Moller's clinical presentation (i.e., severe vomiting despite multiple antiemetics before being found down with no anatomic cause of death other than her congenital heart disease) was consistent with lethal methylxanthine poisoning. This clinical presentation is also inconsistent with mitragynine toxicity.

82. Harmony Moller did not die from any contribution of mitragynine to her congenital heart disease.

83. Harmony Moller did not die from mitragynine toxicity

84. Harmony Moller did not die from her kratom use.

85. I am able to defend rigorously these conclusions, made within a reasonable degree of medical and toxicologic certainty. I reserve the right to supplement, amend or addend these opinions based on additional or new information, including other expert reports.

Rebuttal Opinions

Rebuttal to Christopher Williams's report

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86. I have reviewed the report generated by Plaintiff's expert Christopher Williams, PhD, a laboratory researcher and NIH-funded scientist. Dr. Williams acknowledges he cannot "identify cause of death" or provide "clinical diagnostic information."

87. Dr. Williams attempts to link together all negative aspects of opioids, kratom and kratom alkaloids. He attempts unsuccessfully to establish a toxic profile of kratom alkaloids, each with an atypical corynanthe structure, by drawing parallels with formal opioids having phenanthrene, anilidopiperidine, and diphenylpropylamine chemical structures.

88. In formulating his opinions on the effects of kratom alkaloids on health, Dr. Williams picks and chooses among the scientific literature while making numerous scientific omissions and factual errors. His literature review is insufficient to allow an understanding of the complexity of kratom pharmacology, behaviors following use of the plant, or pharmacophysiologic outcomes of kratom use.

89. Dr. Williams cannot explain why kratom might contribute to Harmony Moller's death from her congenital heart disease (that is lethal by itself), and he does not describe any putative physiologic or clinical effect of mitragynine that might directly affect Ms. Moller's congenital heart disease.

90. Dr. Williams highlights molecules such as 7-hydroxymitragynine (a metabolite of mitragynine) and mitragynine pseudoindoxyl (a rearrangement product of 7-hydroxymitragynine). But the concentrations of neither were measured, and the medical examiner did not identify any connection between 7-hydroxymitragynine or mitragynine pseudoindoxyl and Harmony Moller's death. Despite opining on opioid effects, Dr. Williams fails to explain how opioid activity relates to a high takeoff of the coronary artery with an acute angle of origin.

91. Dr. Williams also attempts to demonstrate that mitragynine, a metabolite (i.e., 7-hydroxymitragynine), and a rearrangement product all "rival the potency of clinically available opioids" without scientific support. Dr. Williams makes a fundamental scientific mistake by conflating 'potency' with 'affinity'.

92. In his report, Dr. Williams implies that large amounts of 7-hydroxymitragynine are produced following ingestion of kratom, with a mean ratio of 0.20-0.29. This conflates parent/metabolite ratios from *single* dose administration and *multiple* dose administration. Single dose administration produces the ratio cited by Dr. Williams. Repeated ingestion of kratom by individuals, such as Ms. Moller, produces substantially lower ratios from poorer conversion of mitragynine to 7-hydroxymitragynine (Heustis 2024). The ratio of conversion in Ms. Moller, based upon the scientific literature, is closer 0.15-.21 (Heustis 2024). Even if a higher conversion ratio occurred in Ms. Moller from intermittent use of kratom, the amount of 7-hydroxymitragynine is toxicologically irrelevant. (Chiang 2025; Hill 2021)

93. Dr. Williams states, "[T]he ingestion of Kratom yields significant amounts of both MG and 7HMG, and subsequently yields pharmacological activity associated with both compounds throughout the duration of exposure." (Williams Report, page 3) The term "significant" carries precise scientific meanings associated with specific mathematical

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comparisons which Dr. Williams never makes in his report. In addition, the term “pharmacological activity” can mean anything related to the body’s handling of a xenobiotic; that these activities arise throughout the duration of exposure is unsurprising since pharmacologic activities can only occur from the moment a xenobiotic is introduced until it is totally eliminated. Finally, powdered kratom, the sort of which was used by Ms. Moller, contain small amounts of mitragynine and a toxicologically negligible mass of 7-hydroxymitragynine (Chakraborty 2021; Zhang 2021; Smith et al., 2024).

94. While the toxicological behavior of a molecule can be explained by its pharmacology, “pharmacological activity” does not by itself equal or even connote *toxicological* activity. Dr. Williams cannot point to a single proven effect of kratom or any of its alkaloids that can exacerbate a high takeoff of the coronary artery with an acute angle of origin.

95. Dr. Williams claims that “cardiotoxicity has been clearly linked to . . . kratom, which includes QT prolongation,”² citing a single case report. (Williams Report, page 4). But human studies have demonstrated daily kratom use is not associated with prolongation of the QT interval (Abdullah J. Tox 2021). Additionally, a separate human subjects study found mitragynine blood concentrations below 9,400 ng/mL were not associated with prolongation of the QT interval, and even individuals whose blood mitragynine concentration exceeded 22,000 ng/mL demonstrated slight increases in the QT interval but exhibited no cardiotoxicity. (Abdullah Frontiers in Pharmacology 2021) Unlike these studies, the single case report on which Dr. Williams relies suffers from methodologic deficiencies, including not verifying the use of kratom and relying upon standard urine drug screens to disprove use of additional substances. Moreover, the patient in Dr. Williams’s case report³ received an intervention that is different than those used to treat Torsades de Pointes, the cardiac rhythm caused by QT prolongation (Abdullah, 2019).

96. In an attempt to highlight putative effects of kratom on the QT interval, Dr. Williams cites Krantz 2023. As recognized by Dr. Williams, Krantz relies upon the FDA Adverse Event Reporting System (FAERS). But there are profound shortcomings of this dataset that color the outcome of the study. These limitations include but are not limited to ambiguity about the role of the ingested xenobiotic, lack of characterization of the product, use of concomitant medications, and effect of underlying disease on outcome. Each of these conditions applies to Ms. Moller. Additionally, reporting of the FAERS data set is biased “by patient’s or reporters’ perception of the drug/natural product.” (Li 2024). In fact, the FDA in its recent report entitled “7-Hydroxymitragynine (7-OH): An Assessment of the Scientific Data and Toxicological Concerns Around an Emerging Opioid Threat” declined to even analyze FAERS data because of the striking limitations of the database. (https://www.fda.gov/files/drugs/published/7-hydroxymitragynin_7-oh_an_assessment_of_the_scientific_data_and_toxicological_concerns_around_an_emerging_opioid_threat.pdf).

² The QT interval is a measure of cardiac repolarization on the electrocardiogram that has been associated with a specific type of ventricular dysrhythmia known as Torsades de Pointes (<https://my.clevelandclinic.org/health/diseases/21915-torsades-de-pointes>).

³ No cardiac rhythm was described in the case report, only that the patient was “pulseless” (Abdullah 2019).

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97. Dr. Williams also claims there are “significant drug-drug interactions involving MG and 7HMG,” but he cannot point to any scientifically sound example where kratom or its alkaloids led to any pharmacodynamic interactions between kratom, its alkaloids, and other drugs.

Rebuttal to Josef Thundiyl’s report

98. I have reviewed the report of Dr. Thundiyl. Dr. Thundiyl disagrees with both the medical examiner and cardiac pathologists, disregarding important autopsy findings that inform the cause of death. For example, he reports the high origin of the left coronary artery, but he disregards the acute angle of origin and its recognized association with sudden cardiac death. Similarly, Dr. Thundiyl ignores entirely the anatomic flap that obscures the origin of the artery. Any anatomic feature that obstructs blood flow into an artery is likely to produce end organ damage. Depending on the location of the obstruction (e.g., near the takeoff of a major artery) and the organ that artery perfuses (e.g., the heart), death is a likely outcome—exactly as recognized by several publication in the medical literature (Rizzo 2021; Taylor 1992; Muriago 1997; Loukas 2016; Virmani 1984; Nishida 2014).

99. Dr. Thundiyl incorrectly states that a mitragynine blood concentration of 150 ng/mL is sufficient to be lethal. First, a blood concentration of 150 ng/mL is within the range of concentrations achieved in clinical trials where no adverse events occurred (Heustis 2024). Second, Dr Thundiyl disregards the known postmortem redistribution of mitragynine. Based on the existing literature, Ms. Moller’s likely premortem concentration of mitragynine is approximately 50 ng/mL (Osawa 2025).

100. Dr. Thundiyl recognizes the presence of caffeine as identified by the medical examiner, but he misses that the methylxanthine toxidrome could be present. Dr. Thundiyl also overlooks Ms. Moller’s use of multiple dietary supplements that contained caffeine and other vasoactive compounds for which there is evidence of use. Furthermore, Harmony Moller exhibited a well-recognized toxidrome of protracted vomiting unresponsive to antiemetics, had no anatomic cause of death other than her congenital heart disease that is known to be lethal by itself, and had a qualitative caffeine measurement.

101. Dr. Thundiyl correctly disagrees with Dr. Williams that neither diphenhydramine nor promethazine contributed to Ms. Moller’s death. As no pharmacodynamic interactions have plausibly been attributed to kratom use, the likelihood that one arose now, particularly involving multiple medications with minimal cardiovascular effects at such low concentrations, is low.

102. I am able to defend rigorously these conclusions, made within a reasonable degree of medical and toxicologic certainty. I reserve the right to supplement, amend or addend these opinions based on additional or new information including other expert reports.

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Sincerely,

/s/ Edward Boyer
Edward Boyer, M.D. Ph.D.