

# **KRATOM CONSUMER PROTECTION AND SAFETY ACT**

## **sponsor: Senator Tammy Nichols**

Chairman, Vice-Chair and Committee Members, please find the following enclosures:

### **1. "Why States Should Adopt a Kratom Consumer Protection Act"**

Framework that 20 states, including Wyoming in March 2026, have enacted and why.

### **2. "7-OH IS NOT KRATOM"**

FDA explicitly not targeting kratom leaf, recommends DEA scheduling of 7-OH.

7-OH that is packaged and marketed as "advanced kratom" is not the natural leaf.

7-OH is a synthetic opioid largely imported from China.

### **3. "Survey of Adult Kratom Users in the U.S."**

- Johns Hopkins Medicine (February 2020)

91% of 2,798 surveyed indicated use for pain relief

61% female, average age: 40 years old

### **4. "FDA completes a kratom 'dose finding study' on humans"**

- FDA: Journal of Clinical Psychopharmacology (Peer-reviewed published March 16, 2026)

Findings:

No significant adverse effects were observed - even at high doses.

### **5. "Analysis of the autopsy report" (Kielee Rustici)**

- Dr. Edward Boyer MD, PhD., The Ohio State University

Findings:

No evidence of [kratom] plant matter in deceased's gastrointestinal tract - kratom was likely not responsible for her death.

### **6. "What is 7-Hydroxymitragynine?"**

- New Jersey National Guard (October 2025)

Public service announcement specifically distinguishes from 7-OH and natural leaf kratom.





# AMERICAN KRATOM ASSOCIATION

## POLICY BRIEF

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### Why States Should Adopt a Kratom Consumer Protection Act (KCPA) Protecting Consumers Through Regulation — Not Prohibition

*Kratom* (*Mitragyna speciosa*) is used by millions of Americans for pain relief, focus, or to ease withdrawal symptoms. Without regulation, however, untested or synthetically altered kratom products pose serious risks to public health. Rather than banning kratom and driving consumers toward unsafe, underground markets, a Kratom Consumer Protection Act (KCPA) establishes a balanced, science-based framework that ensures safety, transparency, and accountability while preserving access for responsible adult consumers.

Regulations work because they replace a chaotic, unmonitored market with clear safety standards and enforceable requirements. When vendors must comply with testing, labeling, and licensing laws, unsafe products disappear from legitimate commerce, consumers gain access to verified information, and enforcement becomes more targeted and effective.

#### What the KCPA Does: A Strong Regulatory Framework

A well-designed KCPA ensures consumer protection without prohibiting natural kratom. Key provisions include:

- **Ban on adulteration:** No controlled substances or non-kratom synthetic opioids may be added.
- **Prohibition of synthetic 7-OH:** Products with concentrated or synthetic 7-hydroxymitragynine are banned.
- **Testing & COAs:** Independent laboratories must issue Certificates of Analysis verifying alkaloid content (including total mitragynine / 7-OH) and confirming the absence of contaminants such as heavy metals, pathogens, and adulterants.
- **Labeling:** Clear, accurate labeling with manufacturer identity, ingredients, serving size, number of servings, and health warnings.
- **Age restrictions:** Typically 18+ or 21+, plus controlled placement (e.g., behind the counter).
- **Licensing / registration:** Processors and retailers must be licensed or registered for traceability and accountability.

#### States Leading with KCPA Protections

As of 2025, 19 U.S. states have adopted KCPA-style protections for kratom, establishing safety standards and enforcement mechanisms that protect both consumers and legitimate businesses. These states are:

**Utah, Georgia, Arizona, Nevada, Oregon, Colorado, Oklahoma, Texas, Nebraska, South Dakota, Kentucky, West Virginia, Virginia, New York, Maryland, South Carolina, Mississippi, Florida, and Texas.**

Adopting a Kratom Consumer Protection Act is the most effective and balanced way for states to protect their citizens. The KCPA replaces unregulated risk with enforceable safeguards—restricting synthetic and adulterated products, mandating testing and transparency, and ensuring responsible access.

# 7-OH IS NOT KRATOM

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The 7-OH industry duped consumers in 2023 when they first introduced their chemically manipulated opioid products by deceptively marketing them as kratom. Now they are trying to dupe legislators into thinking 7-Oh and natural kratom leaf products are exactly the same.

## THEY ARE NOT!

7-OH products have been recommended for classification as a Schedule I controlled substance – Natural Kratom Leaf products have not.

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### FDA Commissioner Makary:



“To be clear, the kratom plant leaf, which contains trace amounts of 7-OH and has been consumed for centuries, is not our focus at the FDA.” (<https://nypost.com/2025/07/29/opinion/beware-synthetic-kratom-7-oh-powers-a-new-opioid-crisis/>)

“Vape stores are popping up in every neighborhood in America, and many are selling addictive products like concentrated 7-OH. After the last wave of the opioid epidemic, we cannot get caught flat-footed again,” **said FDA Commissioner Marty Makary, M.D., M.P.H.** “7-OH is an opioid that can be more potent than morphine. We need regulation and public education to prevent another wave of the opioid epidemic.” (<https://www.fda.gov/news-events/press-announcements/fda-takes-steps-restrict-7-oh-opioid-products-threatening-american-consumers>)

It’s concentrated 7-OH, a synthetic byproduct of the kratom plant that binds strongly to the body’s opioid receptors — making it up to 13 times more potent than morphine. This addictive compound is ubiquitous, it’s being pushed deceptively to consumers, and its use is quietly growing. In previous waves of the opioid crisis — prescription opioids, heroin and fentanyl — the Food and Drug Administration realized too late that a public health crisis was raging, and got caught flat-footed. (<https://nypost.com/2025/07/29/opinion/beware-synthetic-kratom-7-oh-powers-a-new-opioid-crisis/>)

### HHS Secretary Kennedy:



“Today, we’re taking action on 7-OH as a critical step in the fight against opioid addiction,” **said HHS Secretary Robert F. Kennedy, Jr.** “We will protect the health of our nation’s youth as we advance our mission to Make America Healthy Again.” (<https://www.fda.gov/news-events/press-announcements/fda-takes-steps-restrict-7-oh-opioid-products-threatening-american-consumers>)





# Survey of Adult Kratom Users in the U.S.

Provides Insight Into Potential for Harm or Abuse

2,798 kratom users

## WHO:



84% at least some college  
40 years old on average

61% are women



## WHY:



91% pain relief  
67% treat anxiety  
64% treat depression  
41% treat opioid dependence<sup>1</sup>



Of those treating opioid dependence:

87% reported relief from withdrawal symptoms

35% were free from opioids >1 year



## SIDE EFFECTS:



19% mild  
1.9% serious<sup>2</sup>  
9.5% withdrawal

<10% met criteria for mild kratom substance use disorder

<3% met criteria for moderate or severe kratom substance use disorder



1. many people reported multiple reasons for use
2. including symptoms like anxiety, irritability, depression and insomnia



JOHNS HOPKINS  
MEDICINE





FEBRUARY 2024

## FDA completes a kratom “dose finding study” on humans where no significant adverse events were observed even at very high doses.



The FDA acknowledged at a scientific meeting the data showed no significant safety concerns in the ascending dose study on kratom use and that clears the way for the planned Human Abuse Potential study.

The FDA announced on January 16, 2024, it will accept proposals to conduct a Human Abuse Potential (“HAP”) study to assess the potential severity of a kratom dependency or addiction liability. The HAP study is authorized only because the dose finding study showed kratom can be safely ingested.

An FDA scientist reported on some of the results of the dose finding study at the Third Annual Kratom Symposium on February 14-15, 2024. Researchers report that some of the policy staff at the FDA were “profoundly disappointed” at the lack of adverse events that occurred among human participants in the dose finding study, where the ascending doses got to 12 grams of kratom material before just 2 of the participants experienced some nausea. That level of kratom consumption is extraordinarily high among current kratom consumers.

It is reported that the scientists at FDA accepted the safety data for its evidentiary value and are now preparing to make a public presentation on the results of the dose finding study at a scientific conference in the fall of 2024.

That dose finding study data cleared the way for the HAP study to be advertised and that is expected to be completed within 2 years. It is important to note that the dose finding study had to demonstrate kratom can be safely consumed before the HAP study could ethically be advertised.

Kratom researchers are excited that the next level of studies on the safety and addiction liability of kratom extract products, and safe consumption levels can be identified, and the limits on kratom plant constituents in a kratom product before it is deemed to be adulterated. Those needed dose finding and HAP studies for kratom extract products will take several years after the current HAP study is completed.

- i. <https://grants.gov/search-results-detail/351644>
- ii. <https://pharmacy.ufl.edu/third-international-kratom-symposium/>
- iii. <https://www.fda.gov/files/drugs/published/Botanical-Drug-Development-Guidance-for-Industry.pdf>



# A pilot, placebo controlled, dose-finding, pharmacodynamic and pharmacokinetic study of orally administered botanical kratom in non-dependent, recreational polydrug users with opioid experience under fed conditions

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## Abstract

FDA has warned consumers about kratom (*Mitragyna speciosa*), a plant endogenous to Southeast Asia containing alkaloids with affinity and activity at mu opioid receptors, sites known to be associated with abuse potential. Although kratom use is prevalent in the US, prospective clinical investigations have been limited. This pilot study evaluated the pharmacodynamic (PD) effects, safety, and pharmacokinetics (PK) of the kratom alkaloids: mitragynine, 7-hydroxymitragynine (7-HMG), panyanthine, specioygnine, and specioclatine following oral administration of botanical kratom.

## Introduction

Kratom is a member of the coffee family (Rubiaceae) and native to countries in Southeast Asia, including Thailand, Malaysia, New Guinea, and the Philippines. For centuries, the leaves of the kratom plant have been used for cultural, recreational, health-promoting, and medicinal purposes. Fresh kratom leaves are usually chewed or made into tea for use. Products prepared from kratom leaves are available through the Internet and via brick-and-mortar stores. According to the National Survey on Drug Use and Health, an estimated 1.7 million Americans aged 12 and older used kratom in 2021<sup>1</sup>. In addition, kratom-related exposures reported to poison control center increased from 2014-2019<sup>2</sup>. FDA has determined that kratom is not appropriate for use as a dietary supplement and has concluded that kratom is a new dietary ingredient for which there is inadequate information to provide reasonable assurance that such ingredient does not present a significant or unreasonable risk of illness or injury<sup>3</sup>. Kratom is a botanical product that contains a mixture of indole-based alkaloid compounds. At least 25 alkaloids have been isolated from kratom, mitragynine represents about 66% of the total extractable alkaloid content of kratom, with panyanthine, specioygnine, and 7-hydroxymitragynine being the next most abundant<sup>4</sup>. In vitro binding studies have demonstrated that mitragynine and 7-hydroxymitragynine (7-HMG) bind to  $\mu$  opioid receptors and that mitragynine has pharmacological activity at a variety of receptors including other opioid ( $\kappa$ ,  $\delta$ , and  $\sigma$ ), serotonergic, and adrenergic receptor subtypes<sup>5</sup>. 7-HMG may be the most potent of the alkaloids with regard to psychoactive effects and is self-administered by animals whereas mitragynine is not<sup>6</sup>. Thus, while mitragynine and 7-HMG are thought to mediate most of kratom's pharmacologic effects, the presence of other minor alkaloid compounds such as specioclatine may contribute to the overall effect of kratom through multiple pharmacologic pathways.

While there are extensive anecdotal reports regarding the use of kratom in the US, Malaysia, and Thailand, overall, the available clinical literature is sparse. Most published studies on kratom are not controlled or prospective, utilize varied kratom products, and there are few studies directly correlating specific doses of kratom with study endpoints. Within the case reports available, many describe subjects that had a history of chronic kratom use, consumed large amounts of kratom, and/or used multiple substances. Other studies reported in the literature are retrospective analyses of poison center databases, case studies, autopsies, and social media posts, as well as survey (self-reported information) and interview (conducted by trained professional) studies. There are only a few prospective clinical pharmacokinetic studies. Because of the dearth of properly controlled clinical investigations, the current study proposed to examine the pharmacokinetics and pharmacodynamic effects of botanical kratom. These data may be useful to help characterize the safety of kratom and inform future investigations of its pharmacological effects.

## Materials and Methods

This study was performed under an Investigational New Drug (IND) application. Botanical kratom was obtained from Sun Distribution, Super Organics. 40 healthy recreational polydrug users (8 subjects/cohort, 6 active 2 placebo) completed the study. To be included in the study, subjects had to have experience with opioids, defined as recreational use  $\geq 10$  times in their lifetime and  $\geq 1$  time in the past 12 weeks. In addition, subjects had to have used  $\geq 2$  or more perception altering drugs or stimulants on  $\geq 5$  occasions in their lifetime. Subjects were otherwise healthy with exclusions for significant diseases and a history of substance use disorder.

This study utilized a between-subjects design where subjects randomly received a single dose of placebo or kratom. The planned starting dose was 1g and subsequent doses of 3, 8, 10, and 12g were administered after interim safety and pharmacodynamic (PD) data reviews following the completion of each cohort. Once enrolled, subjects received orally administered kratom (made up of 500 mg/capsule) or placebo under double blind conditions. Subjects arrived at the research unit the day before dosing and study capsules were administered under fed conditions after a standardized high fat meal. After dosing, serial assessments of pharmacodynamic (PD) subjective effects using Visual Analog Scales (VAS) and blood samples were collected over 24 and 48 hours, respectively. Safety and tolerability were assessed throughout the study and subjects left the research unit 48 hours after dosing. Safety assessments included AE monitoring, laboratory tests, vital signs (blood pressure, pulse, respiratory rate, oxygen saturation, and body temperature), ECG assessments, physical examination findings, and C-SSRS.

A validated HPLC method using MS/MS detection was employed in determining sample concentrations of the kratom alkaloids in human plasma. Additional analyses of alkaloids are forthcoming.

## Results

Forty subjects (N=40) completed the study, with N=8 subjects/cohort. The composition of the botanical kratom appears in Table 1.

Alkaloid	Capsule Content (mg)
Mitragynine	5.07 ± 0.71
Specioygnine	0.92 ± 0.13
Specioclatine	1.98 ± 0.26
Mitracolatine	0.29 ± 0.04
7-Hydroxymitragynine	BLLO*
Panyanthine	1.28 ± 0.18
Corynantheidine	0.13 ± 0.02
Corynoxine A	0.04 ± 0.01
Corynoxine B	BLLO*
Mitraphylline	BLLO*

\*BLLO = below the limit of quantification (1 ng/ml equivalent to 12 ng/capsule)

Table 1. Alkaloid content of botanical kratom. Values represent alkaloid quantities per capsule administered.

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. AEs by System Organ Class (SOC) when total kratom events reported were greater than placebo appear below in Table 2.

SOC (AE)	Kratom (n/N, %)	Placebo (n/N, %)	Kratom (n/N, %)	Kratom (n/N, %)	Kratom (n/N, %)	Kratom (n/N, %)	Relative Frequency (R/F)	Relative Frequency (R/F)
Gastrointestinal Disorders	0	0	2 (33.3%)	1 (16.7%)	1 (16.7%)	0	0.00%	0.00%
Nervous System Disorders	1 (16.7%)	0	1 (16.7%)	1 (16.7%)	0	0	6.00%	0.00%
Psychiatric Disorders	0	0	1 (16.7%)	2 (33.3%)	0	0	0.00%	0.00%
Respiratory Disorders	1 (16.7%)	0	0	0	1 (16.7%)	0	6.00%	0.00%
General Disorders	0	0	1 (16.7%)	1 (16.7%)	0	0	6.00%	0.00%

Table 2. Adverse events by SOC.

When examining AEs by preferred term (PT), vomiting was the most common adverse event (AE) reported, with a total of five reports. Events by PT when total kratom frequency was greater than placebo and greater than one event appear below in Table 3.

Preferred Term (PT)	Kratom (n/N, %)	Placebo (n/N, %)	Kratom (n/N, %)	Kratom (n/N, %)	Kratom (n/N, %)	Relative Frequency (R/F)	Relative Frequency (R/F)
Vomiting	0	0	2 (33.3%)	1 (16.7%)	1 (16.7%)	0.00%	0.00%

Table 3. Adverse events by preferred term. Preliminary assessments of kratom alkaloid exposure(s) suggested orderly, dose-related effects. Mitragynine exposure increased as a function of dose. Analysis of plasma concentrations suggested a variable Tmax that occurred between 2 and 4 hours. Time course data for mitragynine appear below in Figure 1. Analyses of additional alkaloids are pending.

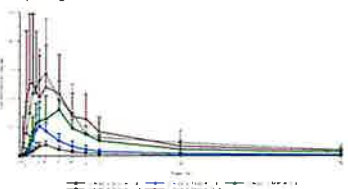


Figure 1. Mean (+SE) plasma concentration-time profile of mitragynine following oral administration of botanical kratom.

At doses > 1g Kratom appeared to produce mild pupillary constriction. This pupillary constriction appeared to have a dose-related component with maximum levels of occurring at doses  $\geq 3g$ . At all doses, pupil constriction was time dependent and resolved 12 h after dosing (Figure 2). Maximum, mean constriction was 2.42 mm after 12g.

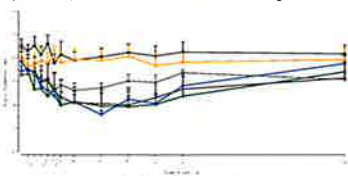


Figure 2. Mean (+SE) for pupil diameter measurements over time following a single, oral administration of botanical kratom.

No dose-related effects were observed on numerous study endpoints assessing the subjective profile of botanical kratom. For example, on measures of at the moment drug liking, kratom produced minor increases from baseline with substantial overlap and no apparent dose-response. The 8 and 12g doses appeared to produce the largest changes, although they did not appear to be significantly different from placebo (Figure 3).

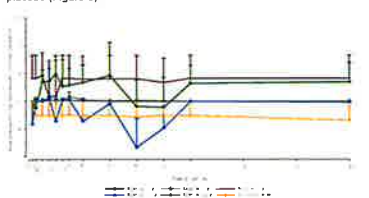


Figure 3. Mean (+SE) VAS measures of drug liking over time following oral kratom administration. Similarly, when examining maximum ratings (Emax) of VAS assessments of overall drug liking and take drug again 12 and 24 hrs after dosing, no apparent dose-related effects were observed following administration of kratom capsules (Figure 4).

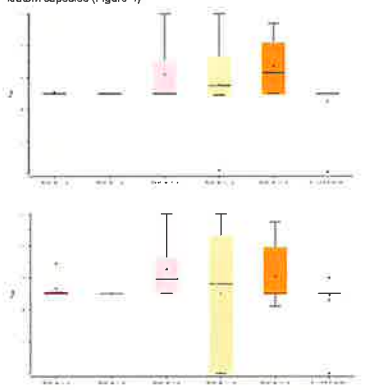


Figure 4. Box-and-Whiskers plot for maximum (Emax) ratings of overall drug liking (top) and take drug again (bottom).

However, kratom appeared to produce dose-related effects on a variety of subjective measures assessed as non-key secondary endpoints. For example, mean ratings of "high" (33.7) and "feeling drunk" (15.7) were highest at the maximum (12g) dose of kratom. These effects appeared to have resolved by 8 hour timepoint (Figure 5).

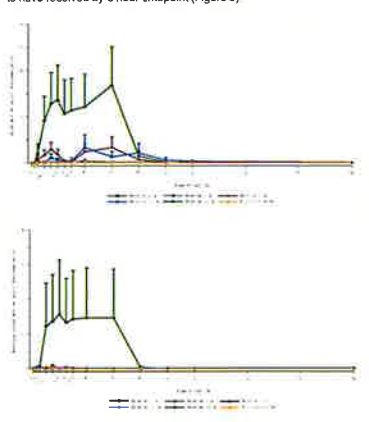


Figure 5. Mean (+SE) VAS ratings of feeling high (top panel) and feeling drunk (bottom panel) over time following oral administration of kratom.

## Discussion

Preliminary data are shown for this pilot, single ascending dose-response (SAD) study of botanical kratom. At the doses tested (1, 3, 8, 10, and 12g), no serious adverse events (SAEs) occurred. Vomiting was the most common AE and showed an increased trend in the higher dose range (i.e., 8, 10, and 12g) compared to the lower doses. Overall, the data suggest that at the doses tested, using the specific botanical kratom sourced for the study, and under carefully controlled clinical conditions (i.e., an inpatient study), kratom was well-tolerated. These data help inform the safety profile of botanical kratom for use in future investigations to more thoroughly characterize its pharmacological effects and abuse potential.

It is unclear how the single sourced, botanical kratom used in our study compares to the wide array of kratom-related products available on the marketplace. Though data are limited, the mitragynine and 7-HMG content of the kratom used in our study is consistent with that reported in the literature. However, more than 50 alkaloids exist in the kratom plant that may have pharmacological effects. Importantly, there is a growing trend of newly created kratom "extracts" that have enriched and increased levels of kratom alkaloids, including increased levels of mitragynine and 7-HMG<sup>7</sup>. For example, a recent investigation of commercially available kratom products found "pressed pills" purportedly derived from kratom with 7-HMG levels far greater than those in naturally occurring (i.e., "botanical") kratom<sup>8</sup>. It is unclear how these high levels of 7-HMG were achieved given the low levels of 7-HMG in naturally occurring kratom. However, the high 7-HMG concentrations may be achieved via chemical conversion of mitragynine to 7-HMG, resulting these semi-synthetic kratom products.

Initial assessments of the pharmacokinetic (PK) profile of the kratom alkaloids suggested orderly dose-effects. Maximum plasma levels (i.e., Cmax values) of mitragynine ranged from 250-300 ng/mL. Notably, Cmax values following 12g appeared to be greater than those after the 10g dose, suggesting dose-related PK effects. These data add to the existing kratom literature, as to our knowledge the 12g dose of kratom administered in this study is the highest dose reported in the literature to date. Additional analyses of 7-HMG and other kratom alkaloids are forthcoming.

Kratom produced pupillary constriction, a finding consistent with mu-opioid effects. However, this constriction was relatively mild (~2 mm maximal constriction), compared to other opioids such as morphine and oxycodone that have been shown to produce  $\geq 4$  mm of pupillary constriction at high doses<sup>9</sup>. Nonetheless, these data appear consistent with the pharmacological effects of mitragynine which has been shown to have affinity and activity at mu-opioid receptors, a finding consistent with the antioxiopietic effects produced by kratom<sup>11</sup>.

Kratom did not appear to produce clear, dose-related or significant effects on VAS-rated measures commonly associated with abuse potential including ratings of drug liking, overall drug liking, and assessments of take drug again. However, kratom did produce a constellation of effects commonly associated with drugs of abuse such as increases in subject-rated measures of good effects, high, and feeling drunk. These data warrant additional studies to more thoroughly assess the abuse potential of kratom.

## Conclusions

- This was a pilot, proof-of-concept study investigating the safety of single sourced botanical kratom. It did not have detectable 7-HMG levels found in some marketed kratom products, thus, the results might not be representative of drug effects associated with other kratom-containing products in the marketplace.
- The small sample size limits the interpretability and translatability of the data.
- The study was not a formal HAP assessment and there are methodological differences between this SAD and HAP studies. For example, a within-subjects design was not used and no qualification session preceded study subject enrollment into the study. In addition, there was no positive control administered for comparative purposes.
- This study utilized encapsulated, ground kratom leaf powder. It is unknown how the results of this study may extend to other kratom preparations such as teas that are commonly used to ingest kratom and may result in increased alkaloid dissolution and bioavailability. This may be significant, as the volume of kratom administered was relatively large (e.g., at the maximum dose, subjects ingested 24 g/200 capsules).
- Future studies more thoroughly examining the abuse potential of kratom and its associated alkaloids may be warranted.

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Analysis of the autopsy report performed on Kielee Rustici (date of death: March 2, 2025)

Gwendolyn Payton, Esq  
Kilpatrick Townsend & Stockton LLP

Dear Ms. Payton:

At your request, I have reviewed the autopsy report for Keilee Rustici dated April 10, 2025. My findings are below.

**Summary:** The cursory nature of the death scene investigation, coupled with errors by medical examiner, led to erroneous conclusions regarding the cause of death of Keilee Rustici. The conclusion that mitragynine toxicity contributed to death lacks a basis in scientific fact.

**Analysis:** No evidence points to the recent ingestion of kratom by Ms. Rustici. Overdose deaths overwhelmingly occur soon after ingestion of oral opioids; because opioids decrease gut motility, insoluble material such as the kratom plant matter ingested by Ms. Rustici would be expected to be found in the stomach. None was. The absence of kratom plant matter in Ms. Rustici's gastrointestinal tract is critical because scene investigators identified only kratom plant product in the home but no extracts, elixirs, or alternative formulations. Because Ms. Rustici's last kratom use was therefore likely distant to her death, kratom was likely not responsible for her death.

The rigorous assessment of a decedent's medical history, recognized as critically important in forensic investigations by the National Association of Medical Examiners and the American College of Medical Toxicology, was inadequate. Death scene investigators identified Ms. Rustici's noncompliance with medical care as one reason for obtaining an autopsy. Even though the ways in which Ms. Rustici was not compliant with her medical care were not described, the medical examiner failed to investigate other possible causes of death in his deliberations. That Ms. Rustici suffered from several chronic conditions (eg, Ehlers-Danlos syndrome, POTS disease, depression, nicotine use by vape) is well documented, yet forensic staff conducted only a cursory evaluation of Ms. Rustici's health history, including substance abuse history. The NAME/ACMT best practices publications indicate that a deep understanding of a deceased person's medical history is essential to eliminating other causes of death that are more plausible than putative opioid poisoning.

Similarly, Ms. Rustici had lived in the home where she died for only a short period of time. The death scene investigator noted that 'family problems' were the cause of her moving out of her own home, but no further investigation was performed to determine why her previous housing was unsustainable. The cursory evaluation into Ms. Rustici's social history further erodes confidence in the medical examiner's findings.

The medical examiner did not consider the pharmacokinetics and toxicokinetics of mitragynine in his assessment. Mitragynine undergoes postmortem redistribution so that concentrations of mitragynine measured at autopsy are approximately three times premortem values. Even if mitragynine concentrations do not increase after death, a concentration of 3100ng/mL is not elevated. A review of the peer-reviewed medical literature demonstrates that mitragynine concentrations as high as 22,800 are well tolerated, with no evidence of respiratory depression, cardiac abnormalities, nor neurotoxicity. In his assessment, therefore, the medical examiner makes the classic error of presuming that a concentration of a xenobiotic measured at

autopsy contributed to death when the scientifically correct approach is that the xenobiotic concentration is *associated* with death.

The postmortem concentration of opioid-like agents is useful for determining *exposure* but not *poisoning*. Stated another way, the presence of a substance on forensic testing suggests (because of the presence of false positive results) that a person used a substance, not that it killed him/her. In fact, NAME/ACMT developed their 'Best Practices' because medical examiners erroneously assumed that substances have a "lethal" threshold beyond which death must occur. This is simply untrue, because of the potential for tolerance. The medical examiner could not determine, based upon any concentration of mitragynine, that mitragynine was the cause of Keilee Rustici's death.

The medical examiner also failed to consider the recognized limitations of forensic testing. Laboratory tests, particularly when performed by commercial forensic testing corporations, cannot detect all substances used by a decedent. Research from Colorado found that even in deaths attributed solely to mitragynine, more rigorous testing found the presence of *additional* substances that more plausibly contributed to death.

For the medical examiner's contention—that mitragynine somehow contributed to Ms. Rustici's death—to be correct, the manner in which mitragynine produces death must be known.<sup>1</sup> The toxidrome of mitragynine remains unknown.<sup>2</sup> Because the toxidrome is unknown, the concentration of mitragynine capable of producing this hypothesized toxidrome—and, hence, death—is unknown as well. To suggest, therefore, that mitragynine toxicity contributes to death lacks a basis in scientific fact.

**Conclusion: The autopsy performed on Kielee Rustici lacked sufficient rigor and contained several errors that call into question the medical examiner's determination that the cause of death was mitragynine toxicity.**

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<sup>1</sup> The "toxidrome" is the consistent pattern of clinical findings associated with a particular class of drug. For example, the opioid toxidrome includes stupor and decreased ventilatory responsive to hypercapnia measured as decreased respiratory rate—none of which occurs even at striking mitragynine concentrations.

<sup>2</sup> Opioids affect the body's respiratory response to rising blood carbon dioxide concentrations; this is most commonly measured as a decreased respiratory rate culminating in apnea. Although mitragynine has been known for over 50 years to have limited effects on respiration, the most recent research proves that mitragynine stimulates—not decreases—respirations .

★ NEW JERSEY NATIONAL GUARD ★

# COUNTER DRUG

DRUG DEMAND REDUCTION OUTREACH

## 7-OH Kratom



### What is 7-Hydroxymitragynine?

7-OH is a potent, concentrated opioid compound derived from the kratom plant, *Mitragyna speciosa*. While it occurs in very small amounts in natural kratom, modern products—often marketed as “enhanced kratom”—contain highly concentrated synthetic or semi-synthetic versions of 7-OH. This creates a powerful, addictive, and unregulated substance that is far more dangerous than traditional kratom.

When mixed in drinks, it produces potent opioid-like effects and can lead to severe side effects, overdose, and even death. The visual characteristics of the drink depend on the form of 7-OH used, but in all cases, the primary impact is on the body, not the appearance of the beverage.

*These products are often sold as pills, gummies, drink mixes, and candies in gas stations, smoke shops, and online.*

### Effects and potency

- **High potency:** Studies indicate that 7-OH can be up to 13 times more potent than morphine by weight in its interaction with the brain's mu-opioid receptors.
- **Opioid-like effects:** By activating the same receptors as opioid drugs like heroin, 7-OH can produce pain relief, euphoria, and sedation.
- **Increased risk:** The high concentration and potency of 7-OH in synthetic products increase the risk of dependency, withdrawal, and overdose.
- **Brain function:** A 2025 study on rats found that low doses of 7-OH increase dopamine release, while high doses decrease it.



SCAN FOR MORE INFO

- Some people use 7-OH as a self-medication for chronic pain, believing it to be a more natural alternative to prescription opioids.
- Others use it to manage the symptoms of opioid withdrawal and reduce cravings.
- Products are also marketed for mood elevation, relaxation, and increasing focus.

