



Agenda Sheet for City Council:

Committee: Public Safety **Date:** 12/01/2025

Committee Agenda type: Discussion

Council Meeting Date: 01/12/2026

Date Rec'd	11/26/2025
Clerk's File #	ORD C36820
Cross Ref #	
Project #	

Submitting Dept	MAYOR	Bid #	
Contact Name/Phone	ADAM 6779	Requisition #	
Contact E-Mail	AMCDANIEL@SPOKANECITY.ORG		
Agenda Item Type	First Reading Ordinance		
Council Sponsor(s)	PDILLON ZZAPPONE		
Sponsoring at Administrators Request	YES		

Lease? NO	Grant Related? NO	Public Works? NO
Agenda Item Name	ORDINANCE PROHIBITING THE SALE AND DISTRIBUTION OF KRATOM	

Agenda Wording

An ordinance prohibiting the sale and distribution of kratom products in the city of Spokane; adopting a new Chapter 10.83 of the Spokane Municipal Code.

Summary (Background)

Kratom is a psychoactive plant containing alkaloids including mitragynine and 7-hydroxymitragynine (7-OH) at low levels that can have stimulant and opioid-like effects. The U.S. Food and Drug Administration (FDA) has warned consumers not to use products containing 7-hydroxymitragynine because of the risk of "serious adverse events, including liver toxicity, seizures, and substance use disorder (SUD)". The Washington Poison Center has seen a "vertical spike" in kratom-related calls, including more calls related to 7-hydroxymitragynine and children. Kratom products and synthetic products containing 7-hydroxymitragynine (7-OH) concentrate can be found at local gas stations, smoke shops, and convenience stores. This ordinance prohibits the sale and distribution of kratom products in the city of Spokane.

FIRST READING OF THE ABOVE
ORDINANCE HELD ON
2/23/2026
AND FURTHER ACTION WAS DEFERRED
[Signature]
CITY CLERK

PASSED BY
SPOKANE CITY COUNCIL:
3/2/2026
[Signature]
CITY CLERK

What impacts would the proposal have on historically excluded communities?

How will data be collected, analyzed, and reported concerning the effect of the program/policy by racial, ethnic, gender identity, national origin, income level, disability, sexual orientation, or other existing disparities?

How will data be collected regarding the effectiveness of this program, policy, or product to ensure it is the right solution?

Describe how this proposal aligns with current City Policies, including the Comprehensive Plan, Sustainability Action Plan, Capital Improvement Program, Neighborhood Master Plans, Council Resolutions, and others?

Council Subcommittee Review

Fiscal Impact	
Approved in Current Year Budget?	N/A
Total Cost	\$
Current Year Cost	\$
Subsequent Year(s) Cost	\$
<u>Narrative</u>	
Amount	Budget Account
Select \$	#
Select \$	#
Select \$	#
Select \$	#
Select \$	#
Select \$	#
Funding Source N/A	
Funding Source Type	Select
Is this funding source sustainable for future years, months, etc?	
Expense Occurrence	N/A
Other budget impacts (revenue generating, match requirements, etc.)	
Approvals	
Dept Head	MCDANIEL, ADAM
Division Director	
Accounting Manager	BUSTOS, KIM
Legal	HARRINGTON,
For the Mayor	PICCOLO, MIKE
Distribution List	

ORDINANCE NO. C36820

An ordinance prohibiting the sale and distribution of kratom products in the city of Spokane; adopting a new Chapter 10.83 of the Spokane Municipal Code.

WHEREAS, kratom is a psychoactive plant containing alkaloids including mitragynine and 7-hydroxymitragynine (7-OH) at low levels that can have stimulant and opioid-like effects; and

WHEREAS, the U.S. Food and Drug Administration (FDA) has warned consumers not to use products containing 7-hydroxymitragynine because of the risk of “serious adverse events, including liver toxicity, seizures, and substance use disorder (SUD)”; and

WHEREAS, the University of Washington Addictions, Drug & Alcohol Institute reports 7-hydroxymitragynine (7-OH) as an emerging drug with a higher risk of overdose and use disorder than kratom; and

WHEREAS, the FDA reports kratom-related substance use disorder where individuals have cravings for kratom, use kratom for longer or more than intended, and experience withdrawal symptoms when kratom use is stopped; and

WHEREAS, the Washington Poison Center has seen a “vertical spike” in kratom-related calls, including more calls related to 7-hydroxymitragynine and children; and

WHEREAS, kratom products and synthetic products containing 7-hydroxymitragynine (7-OH) concentrate can be found online and at local gas stations, smoke shops, and convenience stores; and

WHEREAS, the sale and distribution of kratom products have been regulated or prohibited in many states and municipalities, but are not yet regulated in Washington state; and

WHEREAS, based on the public health data, prohibiting the sale and distribution of kratom products containing 7-hydroxymitragynine until regulated by the state or federal government may reduce the risk of accidental overdose, substance misuse, and long-term health impacts, and protect the public health, safety, and welfare of residents and visitors to Spokane.

NOW, THEREFORE, the City of Spokane does ordain:

Section 1. There is enacted a new chapter 10.83 to Title 10 of the Spokane Municipal Code to read as follows:

Chapter 10.83 Sale or Distribution of Kratom Products Prohibited

- 10.83.010 Purpose and Intent
- 10.83.020 Definitions
- 10.83.030 Prohibition on Sale or Distribution of Kratom Products
- 10.83.040 Sunset Upon State or Federal Regulation

Section 10.83.010 Purpose and Intent

It is the purpose and intent of this ordinance to protect the public health and safety of Spokane residents by prohibiting the sale and distribution of kratom (*Mitragyna speciosa*) products to all individuals, including any products containing 7-hydroxymitragynine, mitragynine, or an extract, synthetic alkaloid, or synthetically derived compound.

Section 10.83.020 Definitions

Term	Definition
Distribute	Distribute means to furnish, give away, exchange, transfer, deliver or supply, whether or not for monetary gain.
Kratom Product	Kratom product means any kratom analogue, food product, food ingredient, dietary ingredient, dietary supplement, or beverage intended for human consumption which contains any part of the leaf of the plant <i>Mitragyna speciosa</i> or an extract, synthetic alkaloid, or synthetically derived compound of such plant and is manufactured as a powder, capsule, pill, beverage, or other edible form.
Kratom Retailer	Kratom retailer means any person that sells or distributes kratom products or that advertises, represents, or holds itself out as selling or maintaining kratom products within the city of Spokane.

Section 10.83.030 Prohibition on Sale or Distribution of Kratom Products

- A. A person shall not distribute, sell, or permit to be sold a kratom product to any person.
- B. Any person violating this section shall be guilty of a civil infraction and shall be subject to the escalating penalties and repeat offender provisions prescribed in [SMC 01.05.151](#).

C. Any kratom retailer found guilty of violating this section may have its business license revoked or denied under the procedures prescribed in SMC 08.01.321.

Section 10.83.040 Sunset Upon State or Federal Regulation

This chapter shall be effective until preempted by the State of Washington or the federal government.

Section 2. Severability. If any section, subsection, sentence, clause, phrase, or word of this ordinance should be held to be invalid or unconstitutional by a court of competent jurisdiction, such invalidity or unconstitutionality thereof shall not affect the validity or constitutionality of any other section, subsection, sentence, clause, phrase, or word of this ordinance.

Section 3. Clerical Errors. Upon approval by the city attorney, the city clerk is authorized to make necessary corrections to this ordinance, including scrivener's errors or clerical mistakes; references to other local, state, or federal laws, rules, or regulations; or numbering or referencing of ordinances or their sections and subsections.


PASSED by the City Council on March 2, 2026


Council President

Attest:


City Clerk

Approved as to form:


City Attorney


Mayor

Mar 9, 2026
Date



4/8/2026
Effective Date



7-Hydroxymitragynine (7-OH):

An Assessment of the Scientific Data and
Toxicological Concerns Around
an Emerging Opioid Threat

7-Hydroxymitragynine (7-OH):

An Assessment of the Scientific Data and Toxicological Concerns Around an Emerging Opioid Threat



FDA Center for Drug Evaluation and Research

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EXECUTIVE SUMMARY

Recent reports indicate increased availability and marketing of 7-hydroxymitragynine (7-OH) in the U.S., raising public health concerns due to its pharmacology. This report provides an overview on the chemical, pharmacological, and epidemiological data on 7-OH. It focuses on the characterization of 7-OH-containing products in the marketplace, the evidence of increasing human exposures, and the extensive body of preclinical studies in the scientific literature that indicate the predominant mu opioid agonist pharmacology of 7-OH. These data sources indicate that 7-OH is a potent opioid that poses an emerging public health threat, especially when considering the increasing availability of enhanced or concentrated 7-OH products in the marketplace.

7-OH is a naturally occurring substance in the kratom plant (*Mitragyna speciosa*), but only a minor constituent that comprises less than 2% of the total alkaloid content in natural kratom leaves. However, 7-OH demonstrates substantially greater mu-opioid receptor potency than kratom's primary alkaloid constituent mitragynine, as well as other classical opioids such as morphine. In vitro studies reveal 7-OH exhibits high binding affinity for mu-opioid receptors ($K_i = 7.2\text{-}70\text{ nM}$), with functional activity as a mu agonist. Animal behavioral studies demonstrate its rewarding effects from self-administration and conditioned place preference methods, consistent with its opioid properties. Critically, 7-OH produces respiratory depression, physical dependence, and withdrawal symptoms characteristic of classical opioids, such as morphine, fentanyl, oxycodone, and hydrocodone.

Recently, there has been a concerning proliferation of concentrated 7-OH products that are sold over the counter and online. The enhanced amount of 7-OH in these products is likely synthetically derived through oxidate chemical conversion of mitragynine isolates or kratom extracts. Given the trace amounts of 7-OH that are naturally present in kratom, direct extraction of 7-OH from plant material would simply be unfeasible economically.

Surveillance data from multiple sources, including America's Poison Centers National Poison Data System (NPDS), Drug Enforcement Administration toxicology testing programs, and social media monitoring, suggest increasing human exposure to these concentrated 7-OH products. Clinical presentations include euphoria, sedation, respiratory depression, and opioid-like withdrawal syndromes, with users acknowledging its significant addiction potential.

The pharmacological profile, abuse liability, and emerging patterns of non-medical use establish 7-OH as a dangerous substance. Current regulatory gaps have enabled widespread availability of these products despite their opioid-like properties and necessitate immediate policy intervention to address this emerging threat to American public health.

INTRODUCTION

The Context for 7-OH Concerns

7-Hydroxymitragynine (7-OH) is a component of the plant kratom (*Mitragyna speciosa*), a tropical evergreen tree in the Rubiaceae family that grows in the wetlands of Southeast Asia (Brown et al., 2017). Kratom leaves contain over 50 alkaloids, with mitragynine and 7-OH being the primary psychoactive constituents (Warner et al., 2016). Its leaves, consumed as a tea or in dry leaf form, have been used for centuries in both medicinal and recreational settings, largely due to the properties of its alkaloids mitragynine and 7-OH. Typically, 7-OH occurs in botanical kratom in amounts no more than ~.01-.04 percent by dry weight (Heywood et al., 2024). Medicinally, kratom has been used to treat headaches, diarrhea, insomnia, anxiety, opioid use withdrawal, and more, while in recreational use cases, it has been associated with feelings of euphoria (Hill et al., 2025). Currently, there are no FDA-approved drugs containing kratom or kratom-derived drug substances such as 7-OH for any therapeutic indications.

Kratom products have grown in popularity since the mid-2000's; however, kratom, mitragynine, and 7-OH have faced regulatory scrutiny in the United States due to concerns about their safety and potential for abuse. None of these substances are lawful when added to conventional foods, as dietary supplements, or as ingredients in any FDA-approved drug, and yet, these substances are still sold in various markets. At the state level, some jurisdictions have implemented restrictions on their sale and use. Until now, 7-OH has not been the sole target of a regulatory response but has always been addressed alongside the kratom plant and mitragynine.

FDA issued its first import alert for kratom in 2012. At the time, kratom was being marketed in various forms for human consumption despite a lack of approved drug uses or established safety as a dietary ingredient. In the years since, additional import alerts have been issued by the Agency. The Drug Enforcement Administration (DEA) and the Department of Health and Human Services (HHS) had given consideration to kratom, as well as its constituents, mitragynine and 7-OH, to determine whether these substances should be recommended for control under the Controlled Substances Act (CSA). Those actions were ultimately suspended in 2018, with the Assistant Secretary for Health at that time stating that the science was incomplete, and the available data were not adequate to support a recommendation to control these substances under the CSA.

Contemporary Outlook

Given the concerning trends with 7-OH and other kratom-related products, FDA has now determined that a more comprehensive assessment of available scientific and medical data on 7-OH is warranted. Many of the products available today, which are often associated with or advertised as kratom, no longer resemble botanical kratom. Instead, they contain "enhanced" or concentrated amounts of 7-OH and are formulated as powders, capsules, and liquid extracts designed to generate a stronger effect on users. Other products are explicitly advertised as 7-OH-containing products. One analysis of websites selling 7-OH products found that most (82.2%) were formulated as chewable/sublingual tablets, shots, or gummies and marketed specifically as 7-OH only products (92%). The mean cost per recommended dose/serving was \$3.97 (Hill et al., 2025).

As described below, research has shown that 7-OH is a potent mu-opioid receptor agonist, demonstrating pharmacological characteristics that define classical opioids like morphine and fentanyl. Based on its opioid pharmacology, there is significant potential for abuse of 7-OH. In fact, in various preclinical studies it has demonstrated greater potency than classical opioids. For example, 7-OH produces respiratory depression with more than 3-fold greater potency than morphine. Since the substance's therapeutic and psychoactive effects are mediated through the same mu-opioid receptor pathways as classical opioids, it can be considered to have opioid properties warranting similar regulatory consideration (Hill et al., 2025; Obeng et al., 2021).

In this report, FDA presents its new assessment of the available scientific data and literature on 7-OH, as well as more recent law enforcement data and the rapidly evolving trends in kratom-related products. FDA still has concerns about the safety of kratom products more broadly and the unlawful marketing of them under several regulated product categories in the Federal Food, Drug, and Cosmetic Act. However, there is a recognized need for more immediate action to address 7-OH because it is a substance with potent mu opioid agonist properties and significant abuse liability.

ANALYSIS OF DATA ON 7-HYDROXYMITRAGYNE (7-OH)

7-OH Sources and Products vs. Kratom

The alkaloid 7-hydroxymitragynine (7-OH) is a naturally occurring substance in the kratom plant (*Mitragyna speciosa*), but only a minor constituent, described as early as 1994, when it was reported to comprise about 1.6% of the total alkaloid content of kratom leaves (Ponglux et al., 1994). This early reported value is in agreement with more recent assessments that have consistently demonstrated 7-OH as comprising less than 2% of the total alkaloid content in natural kratom as noted below.

7-OH has the chemical structure shown in Figure 1. Its IUPAC name is methyl (E)-2-[(2S,3S,7aS,12bS)-3-ethyl-7a-hydroxy-8-methoxy-2,3,4,6,7,12b-hexahydro-1H-indolo[2,3-a]quinolizin-2-yl]-3-methoxyprop-2-enoate, and it has the molecular formula $C_{23}H_{30}N_2O_5$, with a molecular weight of 414.40 amu.

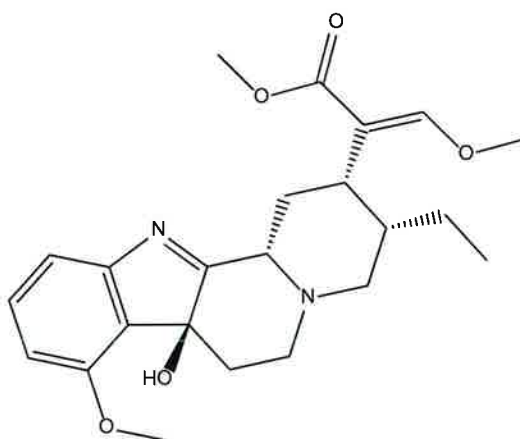


Figure 1. 7-Hydroxymitragynine Chemical Structure

Although details are not well-known, 7-OH is present in some products in amounts far exceeding its natural levels in the kratom plant. The 7-OH in these products is likely derived from the kratom plant. These 7-OH-enhanced products likely involve additional chemical synthetic steps by the producers of these products, converting the more abundant plant alkaloid mitragynine into 7-OH via chemical oxidation.

Data are available regarding 7-OH as a percentage of the total alkaloid content in kratom, and also as a percentage of dried botanical kratom leaf material and other kratom-derived products in the U.S. marketplace. One recent review reports 7-OH as comprising 2% of the total alkaloid content in kratom (Hossain et al., 2023) and this result can be extended to samples of kratom grown in the U.S. (Leon et al., 2009). In another analysis of 13 commercial products purported to contain kratom, the 7-OH content by weight ranged from 0.01-0.04% (Kikura-Hanajiri et al., 2009) a finding in agreement with others that have reported 7-OH to account for less than 0.05% by weight, substantially lower than reported mitragynine amounts (Kruegel et al., 2019). A more

recent study used ecological momentary assessment to evaluate the motivations and patterns of use of adult U.S. kratom consumers (Smith, Panlilio, Feldman, et al., 2024; Smith, Panlilio, Sharma, et al., 2024). As part of the study, subjects provided samples for quantitative testing of their own kratom products that they obtained and were self-administering. Across the 341 samples, the 7-OH content (expressed as a percentage by weight/weight or weight/volume, as indicated) ranged from below the limit of quantitation ($< 0.005\%$) to a maximum of 0.21% with a mean of 0.01% (Sharma et al., 2025). These data suggest 7-OH is present in botanical kratom (i.e., leaf) at relatively low or trace amounts and may be a postharvest oxidative derivative of mitragynine (Karunakaran et al., 2024).

Common forms of kratom sold online include powders, capsules, resin extracts, crushed leaves, and tablets, although loose powder and prepared capsules have been reported to be the most frequently used formulations (Garcia-Romeu et al., 2020; Smith, Panlilio, et al., 2024). While kratom use characteristics are complicated by the diversity of products in the marketplace, survey studies have reported on consumption patterns. Garcia-Romeu collected data from regular kratom users and found that most users reported using 1-3g (49%) or 4-6g (33.4%) of botanical kratom per consumption (Garcia-Romeu et al., 2020). In other survey studies, the self-reported average consumption of kratom powder was 4-5 g per serving with serving sizes ranging between 2.6- 7.5 g (Rogers et al., 2024; Smith et al., 2022). When quantifying the amount of mitragynine consumed through the use of kratom, individuals self-reported consuming an average of 31.3 mg of mitragynine/serving and a range of 78.3 – 134.6 mg of mitragynine per day (Sharma et al., 2025).

Mitragynine, as the most abundant alkaloid in kratom, accounts for about 66% of the *total alkaloid content* of kratom and less than 2% of dried leaf content *by weight*, although there are reports of regional and seasonal variability in the tree's alkaloid composition (Arndt et al., 2011; Leon et al., 2009; Sengnon et al., 2023). For example, Chear and colleagues collected fresh kratom leaves from different locations in Peninsular Malaysia and determined their alkaloid profiles. The mitragynine concentration ranged from 9.38 to 18.85 mg/g or 0.38% to 1.89% of dried leaf weight while the 7-OH concentration ranged from 0.05 to 0.15 mg/g or 0.005% to 0.015% (Chear et al., 2021).

Despite the low amounts of 7-OH in botanical kratom, there are reports of its more-enhanced presence in commercial kratom-related products (Grundmann et al., 2024), although some products have been identified in reports from nearly a decade ago. For example, Lydecker and colleagues tested eight commercially available kratom products for their alkaloid content(s). In seven of the eight products tested, they found levels of 7-OH to be 109-509% higher than expected, based on naturally occurring levels of 7-OH reported in the kratom plant (Lydecker et al., 2016). More recently, the Tampa Bay Times purchased twenty kratom-derived products from local stores. One of those products consisted of pressed pills and contained 15 mg/pill of 7-OH, an amount far greater than observed in any botanical kratom preparation to date (Ogozalek, 2023). In addition to the verified amounts of 7-OH in the products obtained by Lydecker et al. and the Tampa Bay Times, other products *labeled* and/or *purported* to have high levels of 7-OH appear to be readily available for purchase online.

In summary, the low amounts of 7-OH in natural botanical kratom products is well-established as a percentage of alkaloid content, as a percentage of dried kratom leaf material, and in products representing other dosage forms made from natural kratom and consistent with its natural

composition. However, there are also a concerning and increasing number of products being sold that have unexpectedly and unnaturally high levels of 7-OH. This poses a threat to public health that is more clearly understood based on the pharmacological properties and effects of 7-OH, discussed in the preclinical data section below, and also in the limited information available on known patterns of human use and resulting harms discussed below. These sections will present and discuss the evidence in the available data that establishes the mu opioid agonist pharmacology associated with 7-OH in particular.

Patterns of 7-OH Use, Human Exposures, and Law Enforcement Data

There are several sources of information to characterize the current patterns of 7-OH use and the resulting harms to individuals who knowingly or unknowingly are exposed to 7-OH at significant doses from 7-OH-enhanced products, as described in the subsections below.

National Drug Early Warning System (NDEWS)

The National Drug Early Warning System (NDEWS) provides real-time surveillance from sentinel sites across U.S. to detect early signals of potential drug epidemics using novel (e.g., street reporting, web monitoring) and traditional data sources (e.g., OD deaths, treatment admissions).

NDEWS analyzed Reddit posts mentioning 7-OH during January to September 2024 and found that posts increased over this time. These posts are broad and can vary in content but have included warnings from Reddit users about respiratory depression, potency, dependence and long-lasting withdrawal (NDEWS, 2024).

Social Media

A variety of social media outlets were assessed for mentions and/or discussions of 7-OH. Websites included:

- erowid.org - a member-supported organization providing access to information about psychoactive plants, chemicals, and related issues;
- bluelight.org - an international message board that educates the public about responsible drug use by promoting free discussion, advocating harm reduction, and attempting to eliminate misinformation;
- reddit.com - online forum that functions as a vast collection of user-driven communities, known as sub-Reddits, each centered around specific topics.

It is important to note that all considerations of these social media sources are, at best, anecdotal in considering the risks and abuse potential associated with 7-OH products. However, it is clear that there is fairly widespread understanding of the availability of products specifically targeting high levels of the substance 7-OH, distinct from kratom products generally. In analyzing these social media posts, some relevant themes have been identified and include mention of the following: euphoria and an opioid-like “buzz”/high as motivation for consuming 7-OH; availability of “candy-like” formulations which users acknowledge as having a risk of overconsumption to their own detriment; perceptions of therapeutic value of 7-OH in self-treating pain and anxiety; concerns over loss of access to these products if they were to be banned; acknowledgement that use of these products could lead to overdose and serious

outcomes including death; and acknowledgement that use could lead to addiction and has caused users to experience withdrawal symptomology much like that produced by other commonly abused opioids.

Drug Enforcement Administration Toxicology Testing Program (DEA TOX)

The Drug Enforcement Administration Toxicology Testing program (DEA TOX) conducts analyses of voluntarily submitted leftover or previously collected biological samples from drug overdose victims to identify novel psychoactive substances (NPS) and other drugs of abuse in subjects with fatal and nonfatal overdose. The DEA TOX database was queried for reports of mitragynine, 7-OH, or mitragynine pseudoindoxyl from 2019-2025. A total of 103 cases, some fatal and some non-fatal, were identified in this selected sample; this database does not include all overdose cases, and the number of samples voluntarily submitted for analysis may vary year to year based on unknown factors.

It is notable that the utility of the DEA TOX data is limited because it generally cannot be discerned whether deaths are related to mitragynine, 7-OH, or mitragynine pseudoindoxyl, or some combination thereof. In addition, although 7-OH and mitragynine pseudoindoxyl are not typically found in appreciable amounts in fresh kratom leaves (Hill et al., 2025), both are metabolites of mitragynine, complicating forensic assessments of causality (Kamble et al., 2020). These are significant limitations in making inferences from these data; however, the number of fatal overdose cases in which one or more of these substances were detected for 2023 to 2025 are approximately three-fold higher than for the years 2019 through 2022, coinciding with the more recent entry of more-concerning kratom-related products in the marketplace, such as 7-OH.

Human Exposures in Pharmacokinetic Studies

Pharmacokinetic (PK) data for 7-OH are sparse, as to our knowledge, no clinical studies have been performed using isolated or purified 7-OH. Nonetheless, there are 7-OH PK data derived from a small number of studies using botanical kratom. Most available clinical PK data for 7-OH are variable, which may be for several reasons such as genetic differences in kratom plants, different formulations (e.g., teas, capsules, etc.), and methods of analysis. Much of the data is also from non-controlled studies making it difficult to interpret the results. Huestis and colleagues conducted a randomized, between-subject, double-blind, placebo-controlled dose escalation study of 500-4000 mg encapsulated dried kratom leaf powder corresponding to mitragynine doses of 6.65-53.2 mg. Twelve subjects enrolled in the study (n=12). Blood plasma levels of mitragynine and 7-OH were assessed after a single dose, and then again after 15 days of continuous dosing. According to the study authors, peak plasma levels of 7-OH (i.e., C_{max} values) and exposure (i.e., area under the curve, (AUC)) were lower than mitragynine but increased in a dose proportional manner and ranged from 3.6 to 22.7 ng/mL while the time to peak plasma levels (i.e., T_{max} values) ranged from 1.2 – 1.8 h. The half-life of 7-OH increased with increasing dose and ranged from a mean of 1.7 to 4.7 hours. During the multiple dose phase of the study, 7-OH steady state was reached in about 7 days (Huestis et al., 2024).

In another study examining the PK properties of 7-OH, sixteen healthy subjects (n=16) received kratom tea containing 23.6 mg of mitragynine. Subjects were administered tea in two sessions: once with tea alone, and in a second session following pretreatment with itraconazole, a

CYP3A4 inhibitor. The 7-OH C_{max} was 12.81 ± 3.39 ng/mL which occurred 1.7 h after administration (T_{max}). In the second session after pretreatment with itraconazole (200 mg), the C_{max} decreased 56% with a concomitant 43% decrease in AUC. These data describe the PK of 7-OH and demonstrate that the metabolism of mitragynine to 7-OH is heavily dependent on CYP3A4 (Mongar et al., 2024).

Tanna et. al., assessed the PK of a single orally administered dose of kratom (2 g), in the form of a tea, to healthy adult subjects (n = 5 completers). According to the authors, there were only trace amounts of 7-OH (< LOQ) in the starting product, therefore, the assumption was made that 7-OH was generated from the metabolism of mitragynine *in vivo*. The authors identified a PK difference between enantiomers of kratom alkaloids in either the 3S or 3R configuration. 7-OH has a 3S configuration which, according to the authors, leads to a shorter T_{max} , lower exposure (AUC), longer terminal half-life, and a higher volume of distribution during the terminal phase compared to the 3R alkaloids. Measured 7-OH in plasma samples demonstrated that 7-OH had a $C_{max} = 16.1$ nM, $T_{max} = 1$ h, half-life = 5.67h, and an $AUC_{0-120h} = 103nM \times h$.(Tanna et al., 2022).

Epidemiological Data Sources

Limitations with the Epidemiological Data Sources

Because 7-OH appears to be a novel, emerging public health threat, the ability of public health surveillance systems to monitor 7-OH specific risks may be limited. For example, large national surveys such as the National Survey on Drug Use and Health include questions about use of kratom, but not 7-OH. Additionally, there may be a lack of awareness among consumers of kratom-related products that they are obtaining 7-OH enhanced products, and thus use of 7-OH would likely be underreported in data collected using self-report. Many forensic laboratories test for mitragynine as a marker of kratom use. In these cases, 7-OH overdose cases and fatalities may incorrectly be classified as kratom and/or mitragynine-related (Smith, Boyer, et al., 2024). Furthermore, toxicology reports documenting presence of 7-OH are difficult to interpret, because 7-OH is a known metabolite of mitragynine in humans. All of these issues complicate the real-world assessment of risks associated with use of 7-OH containing products as distinct from risks associated with kratom and other mitragynine-containing products.

FDA's Adverse Event Reporting System

Although FDA's Adverse Event Reporting System (FAERS) has documented cases reporting adverse events (13 cases, including 2 deaths) suspected to involve 7-OH, ambiguity about the contributory role of 7-OH from uncharacterized products or concomitant medications and underlying disease limits interpretation. Therefore, we do not include further analysis of these FAERS cases here.

America’s Poison Centers, National Poison Data System

National Poison Data System (NPDS) receives near real-time data from the nation’s poison centers (PC), providing information and assistance to callers on exposures to prescription drugs, over-the-counter medications, unapproved products, and other substances. PC healthcare professionals systematically follow up on exposure cases to document medical and clinical effects. Quality control measures are used to ensure data accuracy and completeness. Notably, 7-OH specific NPDS codes were only recently added (Feb-May 2025), and therefore the NPDS reporting period is limited to 2/1/2025-4/30/2025. As shown below, there were a total of 53 exposure cases involving 7-OH during this time period, the majority of which involved abuse-related reasons for use (i.e., “intentional abuse”). Most single-substance 7-OH exposure cases resulted in minor or moderate clinical outcomes, with several documented has having major clinical outcomes.

Table 1. National Poison Data System Closed Human Exposure Cases*, 2/1/2025-4/30/2025

	Number of exposure cases**	Number of abuse cases**	Single substance exposure cases	Single substance abuse cases
Total cases involving 7-OH	53	24	37	16
Reason				
Adverse drug reaction	4		2	
Intentional- abuse	24		16	
Intentional- misuse	4		3	
Intentional - Suspected suicide	2		0	
Other – Withdrawal	8		6	
Unintentional – general	4		4	
Unintentional- misuse	1		1	
Unintentional therapeutic error	4		3	
Unknown reason	2		2	
Related clinical outcomes				
Minor			6	3
Moderate			13	6
Major			3	1
Not followed, minimal clinical effects possible			5	3
Unable to follow, judged as potentially toxic exposure			1	0
Age				
<18 years	6	1	5	0
≥ 18 years	46	23	32	16
Unknown age	1	0	0	0
*Excludes cases classified as 'confirmed non-exposure'				
**Cases may involve other substances, besides 7-OH				
Related clinical outcomes include cases with clinical effects deemed “related” to exposure based on timing, severity, and assessment of clinical effects by Poison Center Specialists. Definitions available from America’s Poison Centers: NPDS Full Report 2023. Page 235.				

Note: This analysis used the case listing data in NPDS to identify and characterize cases documented as involving 7-OH. As of July 2025, an in-depth review NPDS case narrative data was ongoing; this further review may yield different numbers from those presented here.

Summary of Epidemiological Data and 7-OH Concerns

Available surveillance data indicate that abuse of 7-OH is occurring and is associated with serious harms; however, as noted previously, it is difficult to quantify the public health burden because surveillance systems do not provide estimates for the prevalence of 7-OH use and are only beginning to track the specific involvement of 7-OH enhanced products in exposure cases and overdoses. The current epidemiologic data on 7-OH exposures often lack sufficient detail to distinguish with confidence involvement of botanical kratom products from 7-OH enhanced products.

Preclinical Data Characterizing 7-OH Pharmacology

Although there are limited data from human studies to characterize effects of 7-OH in humans, as noted above, there is a large body of *in vitro* and animal studies that provide extensive evidence of 7-OH as a potent mu opioid agonist, as described in below subsections.

In Vitro Data

Receptor Binding Studies

7-OH has been shown to have affinity and activity at mu opioid receptors. In a study using human embryonic kidney (HEK) cells with cloned, human opioid receptors, 7-OH demonstrated high affinity for the mu opioid receptor ($K_i = 47 \text{ nM}$) relative to kappa ($K_i = 188 \text{ nM}$) and delta opioid receptors ($K_i = 219 \text{ nM}$) (Kruegel et al., 2016). In a second study using HEK 293 cells expressing human mu and other opioid receptors, 7-OH demonstrated high affinity for mu opioid receptors ($K_i = 16 \pm 1 \text{ nM}$) and its affinity was greater than mitragynine ($K_i = 238 \pm 28 \text{ nM}$) and lower than morphine ($K_i = 1.50 \pm 0.04 \text{ nM}$) (Todd et al., 2020). Using an *in vitro* radioligand binding assay with CHO cells expressing murine-derived opioid receptors, 7-OH demonstrated relatively high affinity for mu-opioid receptors ($K_i = 37 \pm 4 \text{ nM}$), relative to mitragynine ($K_i = 230 \pm 47 \text{ nM}$), although its affinity was lower than morphine ($K_i = 4.6 \pm 1.8 \text{ nM}$) (Varadi et al., 2016). Other studies conducted using whole brain homogenates of guinea pig brain tissue have also demonstrated that 7-OH has high affinity at mu opioid receptors ($K_i = 8.0 \text{ nM}$) relative to kappa ($K_i = 6.7 \text{ nM}$) and delta opioid receptors ($K_i = 6.8 \text{ nM}$) (Matsumoto et al., 2004). Obeng and colleagues evaluated the binding affinity of 7-OH using human recombinant HEK 293 cells expressing mu opioid receptors. Their results are in agreement with the data presented above where the authors found that 7-OH binds with high affinity ($K_i = 7.2 \text{ nM}$) to mu opioid receptors relative to delta ($K_i = 236 \text{ nM}$) and kappa ($K_i = 74.1 \text{ nM}$) receptor subtypes (Obeng et al., 2020). A number of additional binding studies are in keeping with the data described above, demonstrating the affinity of 7-OH for mu opioid receptors across a variety of binding assays (Chakraborty et al., 2021; Matsumoto et al., 2008; Obeng et al., 2021; Takayama et al., 2002).

The results of the receptor binding studies with 7-OH are in keeping with *in silico* receptor binding models that suggest 7-OH has high affinity for the mu opioid receptor. The *in silico* modeling results were subsequently confirmed with a radioligand binding assay where 7-OH demonstrated high affinity for cloned, human mu opioid receptors ($K_i = 70 \text{ nM}$). (Ellis et al.,

2020). Collectively, the available receptor binding data demonstrate the affinity and binding of 7-OH to mu opioid receptors.

Functional Studies

Many of the studies referenced above performed additional assessments of 7-OH to determine its functional activity after binding (i.e., agonist or antagonist effects). These studies have consistently demonstrated that 7-OH produces mu-opioid agonist effects. For example, Kruegel and colleagues examined the functional activity of 7-OH and mitragynine in HEK cells expressing opioid receptors using a bioluminescence resonance energy transfer (BRET) assay. Both mitragynine and 7-OH functioned as partial agonists, producing E_{max} values of 34% and 47% respectively and EC₅₀ values of 339 ± 178 nM and 34.5 ± 4.5 nM (Kruegel et al., 2016). Activation of the mu opioid receptor pathway was also investigated using forskolin-stimulated cyclic adenosine monophosphate (cAMP) accumulation in Chinese Hamster Ovary (CHO) cells expressing mu opioid receptors. In this assay, 7-OH produced a maximal activation (E_{max}) of 85.9%, a value similar to that produced by the positive control comparators DAMGO (86.2%) and morphine (86.9%). These data suggest 7-OH acts a full mu opioid agonist (Todd et al., 2020). Similarly, Matsumoto and colleagues concluded that 7-OH was “found to have an opioid agonist property on μ- and/or κ-opioid receptors” based on its ability to inhibit contraction of isolated guinea pig ileum. In this assay, 7-OH displayed approximately 13-fold greater potency than morphine and 46-fold greater potency than mitragynine. The inhibition was reversed by naloxone, suggesting the effects are mediated via mu opioid receptors (Matsumoto et al., 2004). Other functional assays produced results that are aligned with Matsumoto and colleagues. For example, using a cAMP mobilization assay as a measure of functional effects, 7-OH acted as a full agonist with an EC₅₀ of 7.6 nM, and was more potent than mitragynine (EC₅₀ 307.5 nM) (Obeng et al., 2020). Likewise, when evaluating the agonist activity of 7-OH in an electrically stimulated guinea pig ileum, 7-OH acted as a full agonist and was more potent than morphine (Takayama et al., 2002). Finally, using a [³⁵S] GTPγS functional assay, 7-OH produced an E_{max} of 77% with an EC₅₀ of 53.4 nM, further demonstrating its agonist effects (Varadi et al., 2016).

Animal Data on Behavioral and Physiological Effects

Conditioned Place Preference

Conditioned place preference (CPP) is a commonly utilized animal model to study the rewarding effects of drugs. In this paradigm, an animal is conditioned to associate a particular environment with a drug treatment, and an alternative environment with a non-drug condition. After repeated sessions, the animal is then observed under non-drug conditions to determine which environment the animal prefers. CPP is established if the animal spends more time in the drug-paired compartment vs. the vehicle-paired compartment (Mombelli, 2022; Prus et al., 2009). Many drugs of abuse produce CPP, though notably, it is not a direct measure of reinforcing effects.

Using the CPP paradigm, several studies have demonstrated the ability of 7-OH to produce rewarding effects and that it does so more potently than morphine. Gutridge and colleagues employed C57BL/6 mice and demonstrated the development of CPP after 3 mg/kg 7-OH. CPP was observed after both doses although 7-OH required more sessions (4 sessions) whereas morphine (6 mg/kg) was able to establish CPP in two sessions (Gutridge et al., 2020). Similarly,

other studies have demonstrated the ability of 7-OH (2 mg/kg) to produce CPP, and that it does so with greater potency than morphine (Matsumoto et al., 2008).

Drug Discrimination

Drug discrimination is an experimental method in which animals identify whether a test drug produces interoceptive effects similar to those produced by a drug to which the animals are trained to differentiate from placebo, and which has known pharmacological properties. If the known drug is one with abuse potential, drug discrimination methods can be used to predict if a test drug will have abuse potential in humans (Balster & Bigelow, 2003; Solinas et al., 2006).

For abuse assessment purposes, an animal is trained to press one bar when it receives a known drug of abuse (the training drug) and another bar when it receives placebo. A challenge session with the test drug determines which of the two bars the animal presses more often as an indicator of whether the test drug is more like the known drug of abuse or more like placebo. A test drug is said to have “full generalization” to the training drug when the test drug produces bar pressing >80% on the bar associated with the training drug (Ator & Griffiths, 2003; Swedberg, 2016; Walker, 2018; Young, 2009). A test drug that generalizes to a known drug of abuse will likely be abused by humans (Balster and Bigelow, 2003).

Male Sprague Dawley rats were trained to discriminate morphine (5.0 mg/kg i.p.) from saline using a 30 min pretreatment time and FR10 schedule of reinforcement. After successful training, substitution tests with 7-OH (0.3, 1.0 and 3.0 mg/kg) were performed. The highest dose of 7-OH (3.0 mg/kg) produced complete substitution for the morphine stimulus cue. Moreover, pretreatment with naloxone significantly reversed the 7-OH substitution and resulted in saline-like responding. Notably, in this study, 7-OH was more potent than morphine (Harun et al., 2015).

In a second study, the discriminative stimulus effects of 7-OH were examined in separate groups of rats trained to discriminate either morphine (3.2 mg/kg i.p., 15 min pretreatment) or mitragynine (32 mg/kg i.p., 30 min pretreatment) from saline. After successful acquisition of discrimination training 7-OH was administered in substitution tests. 7-OH was administered i.p., with a 15 min pretreatment time in a dose range of 0.1-17.8 mg/kg. In the morphine-trained rats, 7-OH produced complete substitution at doses above 0.56 mg/kg, with the 1.0 mg/kg dose producing 100% drug-lever-appropriate responding and a resultant ED₅₀ of 0.28 mg/kg. Notably, the dose-response curve was shifted to the left, demonstrating an increased potency of 7-OH relative to morphine. In addition, pretreatment with 0.032 mg/kg naltrexone shifted the dose-response curve to the right suggesting substitution was mediated via mu-opioid receptors (Obeng et al., 2021). Taken together, the drug discrimination data demonstrate the ability of 7-OH to substitute and mimic the stimulus effects of morphine, and that 7-OH is more potent in doing so. These data are a strong indication that 7-OH produces subjective effects in humans that are similar to opioids, along with an associated abuse potential.

Self-Administration

Self-administration is a method that assesses whether a drug produces reinforcing effects that increase the likelihood of behavioral responses in order to obtain additional drug (i.e., whether an animal will press a lever for a drug injection). Drugs that are self-administered by animals are

likely to produce rewarding effects in humans, which is indicative of abuse potential. Generally, a good correlation exists between those drugs that are self-administered by animals and those that are abused by humans (Balster & Bigelow, 2003; Brady et al., 1987; Johanson & Schuster, 1981; Panlilio & Goldberg, 2007). It is notable that self-administration is a behavior that is produced by drugs that have been placed into every schedule of the CSA. Additionally, rates of self-administration for a particular drug will go up or down if the available drug dose or the work requirement (bar pressing for drug) is altered. Positive results from a self-administration study provide an abuse potential signal, suggesting that a drug has rewarding properties, but not necessarily that it produces more rewarding effects than another drug in humans.

7-OH produces reinforcing effects and is self-administered by rodents. In the study, rodents were trained to self-administer morphine (100 µg/infusion) and faded to 50 µg/infusion once stable responding was achieved. Thereafter, extinction sessions were performed to confirm acquisition of the self-administration training prior to substitution tests. Substitution tests were performed with 7-OH doses of 2.5, 5, 10 and 20 µg/infusion. In the substitution tests, 7-OH produced an inverted U-shaped curve and the number of infusions for 5 and 10 µg/infusion of 7-OH were significantly greater than vehicle, demonstrating the reinforcing effects of 7-OH (Hemby et al., 2019).

The self-administration of 7-OH was blocked by both a mu opioid antagonist (naloxonazine) and a delta opioid antagonist (naltrindole), suggesting its reinforcing effects are mediated via opioid receptors. In addition, peak morphine self-administration occurred at 50 µg/infusion while peak 7-OH infusions occurred at 5 µg/infusion, demonstrating a substantially increased potency of 7-OH relative to morphine.

There are some pharmacokinetic (PK) data available from animal studies involving the administration of isolated, i.e., single entity, 7-OH. Following a single oral dose (1 mg/kg 7-OH) to beagle dogs, absorption was rapid, with a peak plasma concentration (i.e., C_{max}) of 56 ± 1.6 ng/mL 15 minutes post-dose. The elimination half-life was slower, producing a mean of 3.6 ± 0.5 h. No AEs were observed, and no abnormal laboratory findings were reported (Maxwell et al., 2021). In adult male and female mice, the PK parameters of 7-OH were investigated after a single oral dose of 50 mg/kg 7-OH. The tissue distribution of 7-OH was observed in descending order: liver > kidney > spleen > lung > brain. Plasma C_{max} values were 0.6 and 0.9 µg/mL in males and females with a T_{max} value of 0.5 hr. Area under the curve (AUC) values over 48 hours (AUC_{0-48} hr* µg/mL) were 1.4 and 2.9 in male and female mice (Berthold et al., 2022).

Antinociceptive Effects

The antinociceptive effects of 7-OH were investigated in mice using the tail flick and hot plate tests. These tests are commonly used to examine pain and analgesic effects in rodents (D'Amour & Smith, 1941). In these tests, rodents are subject to a heat stimulus and timed for the duration it takes to move their tail (i.e., tail flick) or produce a response such as jumping, licking, or shaking of limbs (i.e., hot plate).

In the tail flick test, subcutaneous administration of 7-OH (2.5 – 10 mg/kg) produced both time and dose-related antinociceptive effects. Notably, the dose-effect curve for 7-OH was shifted to the left, indicating a greater potency than the positive control comparator, morphine. Similar results were observed in the hot plate test, and when morphine and 7-OH were administered

orally. Naloxone (2 mg/kg s.c.) inhibited the effects of 7-OH and morphine in both tests (Matsumoto et al., 2004; Matsumoto et al., 2008). Concurrent results were observed by Obeng and colleagues using the hot plate test. In their study, 7-OH (0.0032 – 3.2 mg/kg, i.v.) produced maximum antinociceptive effects and was more potent morphine but less potent than fentanyl when administered intravenously. Likewise, naltrexone (0.1 mg/kg) reversed the antinociceptive effects of 7-OH suggesting the antinociception was mediated via mu opioid receptors (Obeng et al., 2020).

Respiratory Depression

A major risk of opioid exposure and cause of opioid-induced death is respiratory depression (Baldo & Rose, 2022; Bateman et al., 2023). To examine the respiratory effects of 7-OH in rodents, whole body plethysmography was used in freely moving, awake rats. Both morphine (10 and 32 mg/kg, i.v.) and 7-OH (1, 3.2, and 10 mg/kg, i.v.) induced significant respiratory depression as assessed by minute volume, tidal volume, and breathing frequency. The mu-opioid agonist naloxone (1.0 mg/kg i.v.) reversed these effects, a finding consistent with the mu opioid effects of 7-OH (Zuarth Gonzalez et al., 2025). These data highlight a potential risk factor of 7-OH exposure and suggest 7-OH may expose individuals to similar risks as classic opioids, including respiratory depression.

Physical Dependence and Withdrawal

It is well-established that chronic administration of opioids leads to the development of tolerance and physical dependence that may culminate into a withdrawal syndrome. In parallel with some of the hot plate tests described above, the ability of 7-OH to produce physical dependence and withdrawal was examined. Mice were treated with subcutaneous 7-OH (10 mg/kg b.i.d.) or morphine (10 mg/kg b.i.d.) for five days. Tolerance was assessed as a reduction of analgesia in the hot plate test. After five days of treatment, both morphine and 7-OH showed a decreased analgesic response on the hot plate test, demonstrating the development of tolerance. In addition, cross-tolerance was also observed between morphine and 7-OH suggesting a similar mechanism of action between the drugs. Finally, after five days of escalating doses of 7-OH and morphine (8-45 mg/kg b.i.d.) the development of withdrawal was assessed with a 3 mg/kg s.c., dose of naloxone injected two hours after 7-OH administration. Both morphine and 7-OH treatment produced signs of withdrawal such as jumping, rearing, urination, ptosis, forepaw tremor, and diarrhea (Matsumoto et al., 2005).

Summary of Preclinical Data

From the studies described above, 7-OH has high affinity for mu opioid receptors and functional activity as an agonist at these receptors. Consistent with this pharmacological activity, 7-OH is self-administered by animals, substitutes for morphine in drug discrimination studies, produces antinociception, and physical dependence leading to withdrawal when administered to rodents. Moreover, 7-OH has consistently demonstrated an increased potency relative to morphine in preclinical rodent studies. These observations suggest 7-OH has pharmacological properties representative of a full mu opioid agonist and an associated high potential for abuse.

CONCLUSIONS

The data described in this report indicate that 7-OH has a significant potential for abuse and associated harms. Conclusively, 7-OH has high affinity and agonist activity at mu opioid receptors. Consistent with this pharmacological mechanism of action, 7-OH demonstrates rewarding effects in that it is self-administered by animals and also produces conditioned place preference, two well-established animal behavioral models measuring rewarding effects as a predictor of abuse potential in humans. In animal drug discrimination studies, 7-OH substitutes for morphine with full generalization. 7-OH is also demonstrated to produce antinociception consistent with opioid pharmacology, and to produce physical dependence when administered to rodents, as evidenced by a classic set of withdrawal signs associated with opioid withdrawal upon discontinuation of opioid administration. Moreover, 7-OH in all above models has consistently demonstrated an increased potency relative to morphine.

Due to the fact that 7-OH is both a metabolite of mitragynine and naturally present in low amounts in botanical kratom, using toxicology results to identify 7-OH as a primary or sole contributor in human exposures is challenging. There is also a need for improved clinical awareness and population surveillance to better characterize patterns of 7-OH use, the products that people are obtaining, and individual treatment needs following 7-OH exposure. Additionally, questions on 7-OH are not generally included in national surveys, and other data sources that rely on self-reported use of 7-OH likely underestimate the number of 7-OH exposure cases, as individuals may be unaware of the distinction from kratom products. Nonetheless, since specific codes were added earlier this year to document 7-OH exposure cases, U.S. poison centers have identified multiple single-substance cases of 7-OH exposure resulting in serious adverse clinical outcomes. Also, although anecdotal, social media and online forums indicate growing awareness and use of 7-OH, and many testimonials of the negative opioid-mediated effects users have experienced, including 7-OH dependence, associated withdrawal syndrome, and addiction.

In the current marketplace in the U.S., 7-OH is increasingly being marketed over-the-counter and online, in concentrated forms or sufficient doses to cause harms to those individuals engaging, knowingly or unknowingly, in use of 7-OH. Based on demonstrated pharmacology, repeated or prolonged use of 7-OH would lead to tolerance, physical dependence, and potentially to opioid addiction—typical of mu opioid agonist drugs of abuse. This public health threat is troubling and requires immediate and impactful policies to educate consumers and take regulatory action that limits access to 7-OH containing products.

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Kratom & 7-OH: What do we know about use, safety, and overdose risk?



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Key Points

- Kratom is a plant from Southeast Asia. Two of its primary psychoactive components are mitragynine and 7-hydroxymitragynine (7-OH). These compounds have opioid-like properties.
- Data and reports suggest kratom use is relatively low in Washington State among those seeking substance use disorder treatment, with some cases of kratom physical dependence and use disorder.
- Kratom offers potential benefits to relieve pain, improve mood, ease opioid withdrawal, and manage symptoms of other substance use disorders, but its effects and safety profile are not fully understood.
- The most commonly reported side effects of kratom are typically mild, but it can also cause serious health issues like respiratory depression, seizures, liver toxicity, and arrhythmias.
- The number of overdose deaths involving kratom in WA State has increased but remain low. Most of these deaths involve other substances as well.
- 7-OH is present at low levels (about 2%) in kratom, however 7-OH is increasingly sold as a stand-alone and more potent product. Concerns about 7-OH's health impacts were expressed by the FDA on July 29, 2025.
 - Preliminary data indicate that 7-OH is much stronger than kratom and can cause severe respiratory depression leading to overdose, which *can* be reversed with naloxone.
 - Opioid use disorder involving 7-OH has been reported by WA State substance use treatment providers and has been successfully treated with buprenorphine.
 - Data on morbidity and mortality associated with 7-OH is very limited; however, treatment providers and the Washington Poison Center report increases in 2025.
- Kratom and kratom-related products such as 7-OH have not been approved by the Food and Drug Administration. More research is needed to evaluate their safety and efficacy and potential consumers should be cautious.
- Contact the Washington Poison Center or WA State Recovery Help Line for for additional information and support.

Introduction

Kratom is a psychoactive substance made from the leaves of the *Mitragyna speciosa* tree, native to Southeast Asia. Use and impacts of kratom are not well understood. Advertisements at gas stations are common, and there are occasional media reports of its involvement in deaths, leading to questions like: *What is kratom? Why do people use it? Is it dangerous? Can you be addicted to kratom?* We answer these common questions below.

Background

Kratom is reported to be used, and sometimes advertised, for its potential to relieve pain, increase energy, improve mood, and alleviate opioid withdrawal (opioids include substances such as morphine, oxycodone, heroin, and fentanyl). Kratom's components tend to produce stimulant-like effects at lower doses and depressant effects at higher doses.¹ The primary compounds in kratom, mitragynine and 7-hydroxymitragynine (7-OH), partially activate opioid receptors to relieve pain and induce mild euphoria. These compounds act on several neurotransmitter systems, suggesting potential antidepressant, anxiety-relieving, and antipsychotic effects.¹

While there are potential therapeutic benefits of kratom, there are also safety concerns. More research is needed to better understand kratom's effects, which may vary greatly depending on the amount taken, how it's processed, and varying quality and consistency of products.² Kratom is typically sold as dried leaves, commonly brewed into tea, or in capsules. It is more recently available as extracts or edibles, which may be considerably more potent.² The use of 7-OH as an isolated compound, typically available in tablet and liquid forms, has increased, and users report that it is much stronger than kratom. The FDA reports that 7-OH binds to opioid receptors three times stronger than morphine, indicating it has strong opioid effects.³ High potency kratom products and polysubstance use including kratom has been linked to deaths.

Kratom and 7-OH are not currently federally scheduled, meaning they are not illegal nor is a prescription required, but kratom has been banned in some states and local municipalities. These products are widely available and commonly sold online, at smoke shops, at gas stations, and in some bars. The Food and Drug Administration (FDA) has not fully evaluated or approved kratom or any kratom-related products and warns against their use.⁴

Common questions about kratom

How common is kratom use?

The number of people using kratom in the United States is difficult to determine. National surveys likely underestimate its use, with studies suggesting that 1-6 million Americans have tried kratom at some point. The American Kratom Association estimates significantly higher numbers, with 10-15 million people in the U.S. having tried kratom and about 5 million current regular users.² Evidence suggests kratom is most often used by current and former opioid users to manage pain, opioid withdrawal, and opioid cravings.⁵

How often do people in treatment for opioid use disorder report using kratom? How do they do in treatment?

Local data from Washington State's opioid treatment programs (OTPs) suggest kratom is rarely reported by clients as their primary substance of use; only 12 patients statewide among 19,162 individuals in care reported kratom as their primary substance used upon entering treatment (*Personal communication, Lauren Kula, Washington State Health Care*



Photo credit: Caleb Banta-Green.

Authority, August 4, 2025). However, improved data collection methods are needed to better understand local rates of use and use disorder.

Washington State health care and substance use disorder (SUD) treatment providers report low prevalence of kratom use among their patients, although most settings do not commonly or systematically screen for kratom. Consistent with existing research, these providers from across WA State find that patients typically use kratom to manage opioid withdrawal symptoms but then find that they become dependent on kratom or 7-OH as well. Health care providers report that the medications for opioid use disorder (OUD), methadone and buprenorphine, have worked well with people with OUD who are physically dependent on kratom or 7-OH.

We have two patients who were using heroin and then switched to exclusively using kratom for the next two years. It kept them from going into opioid withdrawal[,] so they were able to successfully get off heroin. They would feel withdrawal symptoms, however, if they didn't use the kratom for several days and came to us because they were afraid that if kratom ever became banned by the FDA, they would return to heroin (or now fentanyl). We placed them on Suboxone [a brand name for oral buprenorphine] as they dropped the kratom, and just recently they switched to Sublocade [a brand name for long-acting injectable buprenorphine]. They are doing fantastic. – Physician

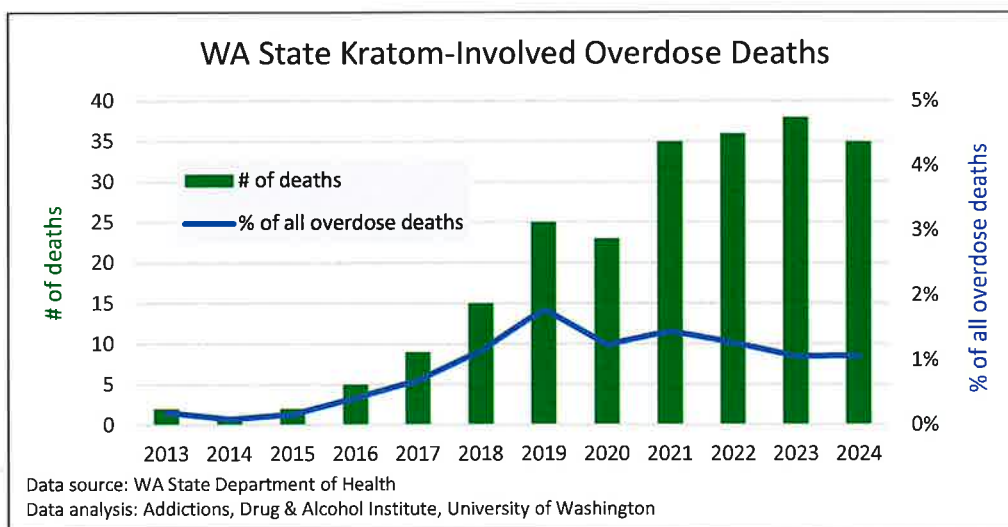
I have treated one patient for kratom dependence. They started opioids with oxycodone pills, did not like being hooked on that and found kratom kept withdrawal away...until they tried to stop that as well. [They] came to our clinic [and] did well on... [buprenorphine]. For a few years after that I would specifically ask about kratom use, including a question on our preliminary assessment form. No one else endorsed using [kratom]. I have since stopped asking. –Physician

We have been testing for kratom but rarely see it... I think it's a group that is used to 'harder' drugs and kratom just doesn't clear the bar. We have had 3-4 people come to us for medications for OUD for kratom dependence and have had fine outcomes with both buprenorphine and methadone. [In] my inpatient work we frequently see kratom dependence as a secondary problem to other use disorders and maybe one primary kratom dependence case in [about] 2 years. – Physician and OTP medical director

It's [7-OH] so much stronger than regular Kratom. Withdrawal is seriously difficult. We have treated 5 people with buprenorphine (which] works well.. – Substance use disorder professional

How often is kratom involved in overdose deaths?

The figure below shows the number of overdose deaths (also known technically as “poisoning”) in Washington State in which kratom was detected. Since 2013, when the first two deaths involving kratom were reported, the number of deaths has increased to 35 in 2024. The percent of all overdose deaths in which kratom was detected has been very low overall and only 1% in 2024. In comparison, in 2024 there were 2,275 overdose deaths in WA State involving “other synthetic opioids,” predominantly non-pharmaceutical fentanyl, representing 70% of all overdose deaths.⁶



Most drug overdoses involve multiple drugs. However, because of inconsistent coding of kratom in multiple drug categories, it is not possible to use International Classification of Diseases (ICD) coding to analyze death certificate data to identify all of the substances detected in a death. Therefore, to explore the presence of other drugs in kratom-involved overdose deaths, ADAI staff manually analyzed the “cause of death” text field from death certificate data for deaths in which kratom/mitragynine was reported in WA State for 2024. These data indicated that among the 35 kratom-involved deaths in 2024:

- 83% (n=29) involved at least one other drug
- 69% (n=24) involved at least one opioid
- 63% (n=22) involved fentanyl with or without other substances
- 17% (n=6) involved only kratom

For comparison, among 4,853 deaths in WA State in 2023 and 2024 that involved “other synthetic opioids” (mostly non-pharmaceutical fentanyl) and in which heroin was not present, 23% had no other drug detected.⁶ That is, there were many more deaths involving fentanyl than kratom, and a larger proportion of fentanyl deaths involved no other drug, suggesting kratom is a less lethal substance despite its widespread availability. It may be difficult to identify 7-OH involved overdose deaths, as it is unclear whether 7-OH would specifically be recorded on death certificates

A detailed analysis of kratom-involved deaths in Florida was conducted by the Tampa Bay Times. They analyzed data from 2013, when the first kratom-involved death was detected, through June 30, 2022. A minority of cases, 8%, involved kratom without any other substances present.⁸ Parallels with WA State data include that 2013 was the same year that a kratom-involved death was detected in both states, and both states report similar, low proportions of deaths involving just kratom.

Evidence on risks and benefits

Research on kratom, especially in the U.S., is limited, with most data coming from case studies and observation. Currently, there is not enough information on kratom to report more definitively on its impact on health and well-being. The National Institute on Drug Abuse (NIDA) notes on their webpage that “NIDA supports and conducts research to evaluate potential medicinal uses for kratom and related chemical compounds...NIDA also supports research towards better understanding the health and safety effects of kratom use. Rare but serious effects have been reported in people who use kratom.”⁹

Evidence suggests that kratom may be effective as an analgesic and may decrease the use of other drugs.¹⁰ People

who use kratom long-term report benefit in managing SUD symptoms (e.g., reducing cravings and use of other substances) and relief from withdrawal symptoms for alcohol, opioids, and other drugs.¹⁰ Results of preclinical studies in animals also strongly suggest that kratom/mitragynine is useful for alleviating pain and opioid withdrawal and has a lower risk of central nervous system effects and respiratory depression than conventional opioids.^{1,11} People who use kratom daily have also reported improvements in daily living and productivity, including reduced pain, improved mood, increased energy, and alertness. Euphoria or feeling “high” is less frequently reported.¹²

The most commonly reported side effects of kratom are typically mild and include agitation, irritability, tachycardia (high heart rate), nausea, vomiting, confusion, drowsiness, and hypertension. Kratom can also cause serious health issues like respiratory depression, seizures, liver toxicity, and irregular heart arrhythmias. Other adverse effects include hallucinations, delusions, depression, dizziness, difficulty sleeping, sweating, tremor, reduced appetite and anorexia, constipation, transient erectile dysfunction, difficulty sleeping, sweating, darkening patches of skin, and hair thinning. Higher doses of kratom and concentrated products are riskier. Using kratom with other substances can enhance the effects of those substances, which may increase negative effects.^{1,13} Furthermore, kratom’s unregulated status as a dietary supplement warrant concern for contamination, mislabeling, and varying quality and consistency, circumstances which have led to serious illness and death.^{1,9,13,14}

People who use kratom frequently can develop tolerance, dependence, and cravings, suggesting the potential for kratom use disorder. However, most users do not report social or functional impairment, a necessary component of a substance use disorder diagnosis.^{12,15} People who use kratom are more likely to have more severe symptoms of SUD related to other substances, but this does not imply that kratom *causes* this. Instead, it may be that people with severe SUD are more likely to use kratom, and thus are trying to stop the use of another, often illicit, substance.^{16,17} A small study showed regular kratom use did not significantly alter health measures, including blood chemistry, organ function, and vital signs of users over time.¹⁸

Less is known about 7-OH morbidity and mortality, but emerging pharmacological data suggests that it is more potent than kratom and mitragynine, and appears to have properties more similar to a pure opioid, including respiratory depression, thereby increasing overdose risk.¹⁹

Recommendations

More information is needed to better understand kratom’s impact. We make the following recommendations based on what is currently known:

- Individuals should carefully weigh the risks before deciding to use kratom and related products such as 7-OH and consider other approaches to manage emotional or physical pain, substance use disorders, and opioid withdrawal. Buprenorphine and methadone are highly effective in treating opioid use disorder, and access is expanding rapidly in WA State.
- Health care providers and SUD treatment providers should be aware of kratom's popularity and potential effects, risks, and medication interactions. Recent reviews of the clinical pharmacology of kratom are available.²⁰ Ask patients about all substances they use, including kratom, in a supportive and non-judgmental way to encourage open conversations. Talk with patients about how kratom may, or may not, fit into their recovery and/or harm reduction goals and strategies.
- Kratom offers potential benefits to relieve pain, improve mood, and manage SUD symptoms, but its effects and safety profile are not fully understood. Reports from WA State data and local health care professionals suggest kratom use is relatively low and primarily for managing opioid withdrawal symptoms. Some cases of kratom dependency and use disorder have been observed. The number of overdose deaths involving kratom has increased but remain low and rarely involve only kratom. Despite its therapeutic potential, concerns about safety call for a cautious approach.
- 7-OH is an emerging drug that appears to pose a higher risk for overdose and use disorder than kratom.

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