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# Therapeutic Potential of Mitragyna speciosa in Neuropathic Pain and Neurodegenerative Disorders: A Literature Review

Alfan Adika Rahman[1], Yogi Ertanto[2]
[1,2] Faculty of Military Pharmacy, Indonesia Defense University, Sentul, 16810, Indonesia.
alfanadika@gmail.com , joghie036@gmail.com

**Abstrak**
Kratom (Mitragyna speciosa) contains active alkaloids, primarily mitragynine and 7-hydroxymitragynine, which interact with the opioid, adrenergic, serotonergic, and cannabinoid systems. This review assesses primary evidence from 2020–2025 regarding neuropathic pain, cognitive or neurodegenerative effects, and pharmacokinetics and drug interactions. Three structured searches were conducted in PubMed/MEDLINE on June 16, 2026, yielding 90 records. After removing seven duplicates, 83 unique records were screened. A total of 29 reports were further assessed, and 17 primary studies met criteria. Citation and title searches added four reports, three of which met criteria, for a total of 20 studies. The evidence for pain comes primarily from animal models of chemotherapy-induced neuropathy and suggests that mitragynine may reduce allodynia through adrenergic, opioid, and cannabinoid mechanisms. Human evidence for analgesic effectiveness is observational and does not prove causality. In the cognitive domain, preclinical findings more frequently indicate impairments in learning, memory, and hippocampal plasticity with high-dose or prolonged exposure. Its therapeutic potential against Alzheimer's disease is supported by docking, in vitro AChE inhibition, and network pharmacology. Human pharmacokinetic studies indicate prolonged mitragynine exposure and potential interactions via CYP3A, while preliminary single-dose clinical data demonstrate short-term tolerability but do not yet address the safety of chronic use. Overall, kratom has consistent preclinical analgesic signals, but clinical evidence for therapeutic use in neuropathic pain and neurodegenerative disorders remains inadequate.

**Keywords**: Mitragyna Speciosa, Kratom, Mitragynine, Neuropathic Pain, Drug Interactions

## 1. Introduction

Kratom is a Southeast Asian plant whose leaves are traditionally used to increase stamina, relieve pain, and reduce opioid withdrawal symptoms. Mitragynine is the most abundant alkaloid, while 7-hydroxymitragynine is present in smaller amounts but has stronger opioid activity. The composition of kratom products varies greatly by variety, processing, and dosage form. Therefore, results from pure compounds cannot always be generalized to teas, leaf powders, extracts, or commercial products.

Interest in kratom as an alternative treatment for chronic pain has increased because preclinical studies have demonstrated antinociception via the μ-opioid receptor and non-opioid pathways. However, claims of neuroprotection in Alzheimer's or Parkinson's disease often stem from computational predictions and in vitro testing, while animal studies have also suggested possible cognitive impairment and synaptic plasticity with chronic exposure. This review clearly separates mechanistic, preclinical, observational, and clinical evidence to ensure conclusions do not exceed the strength of the data.

The primary review questions are: (1) How consistent is the evidence for the analgesic properties of kratom or its alkaloids in neuropathic pain? (2) whether there is evidence to support benefit in neurodegenerative disorders, and how these findings compare with signals of cognitive impairment, and (3) what are the pharmacokinetic, drug interaction, and safety implications for clinical translation.

## 2. Methods

### 2.1. Review Desain

This manuscript is a narrative review with a structured search and selection process. The identification and selection process is reported following the PRISMA 2020 terminology to enhance transparency [21,22]. Because the primary search used only one bibliographic database and the screening was performed by a single reviewer, this manuscript does not claim to be a full systematic review.

### 2.2. Eligibility Criteria

Table 1. Inclusion and exclusion criteria

| Component | Criteria |
|---|---|
| Population/Model | Human, mammalian in vivo, cell/in vitro systems, or computational models directly assessing kratom, M. speciosa extract, mitragynine, 7-hydroxymitragynine, or related natural alkaloids. |

| | |
|---|---|
| Intervention/Exposure | Kratom products are characterized by extracts, teas, leaf powders, or natural alkaloids. Synthetic analogues that are not natural constituents are excluded from primary synthesis. |
| Outcome | Neuropathic/chronic pain or analgesic mechanisms, cognition, neuroplasticity, Alzheimer's/neurodegeneration or pharmacokinetics, drug interactions, and clinical tolerability relevant to translation. |
| Design | Primary studies. Reviews are used only as background and are not counted as included studies. |
| Year/Language | Publication 2020–2025. English-language articles with abstracts and sufficient methodological information. |
| Exclusions | Ulasan, editorial, laporan kasus untuk sintesis efektivitas, abstrak konferensi tanpa artikel lengkap, model yang tidak relevan, dan artikel tanpa luaran sasaran. |

2.3. Search sources and strategies

A search was conducted in PubMed/MEDLINE between June 8 and 16, 2026, with publication dates ranging from January 1, 2020, to December 31, 2025. Three strings were used to cover the domains of pain, neurocognitive, and pharmacokinetics/drug interactions. Exact title and backward citation searches were performed to identify relevant reports not captured by the PubMed strings.

Table 2. Search log and actual number of results as of June 16, 2026

| Components | Criteria | Total |
|---|---|---|
| Q1 – Pain/Analgesia | Kratom/Mitragyna/mitragynine/7-OH + neuropathic pain, allodynia, chemotherapy neuropathy, cancer bone pain, antinociception, analgesia | 35 |
| Q2 – Neurocognitive | Kratom/Mitragyna/mitragynine/7-OH + Alzheimer's, Parkinson's, neurodegeneration, neuroprotection, neuroinflammation, cognition | 27 |
| Q3 – PK/Human Interaction | Kratom/Mitragyna/mitragynine/7-OH + pharmacokinetics, drug interaction, healthy volunteer. Filter Humans | 28 |
| Total before deduplication | Sum of Q1–Q3 results | 90 |
| Duplicates between queries | Deduplication based on PMID | 7 |
| Unique records | 90 – 7 | 83 |

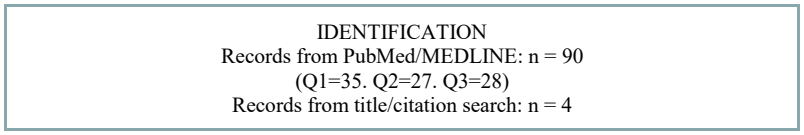
2.4. Selection, extraction, and critical appraisal

Records were merged and deduplicated using PMID. Titles and abstracts were screened against eligibility criteria. Potentially relevant reports were further assessed based on the full article or available methodological information. Extracted data included design, model/population, intervention type, outcomes, main findings, and limitations. Due to the heterogeneity of designs, no meta-analysis was performed.

Critical appraisal was conducted narratively and tailored to the design: the SYRCLE domains were used as a framework for animal studies [23], while human studies were assessed for randomization/blinding, sample size, controls, product standardization, and completeness of reporting. In vitro/computational studies were assessed for the presence of controls, experimental validation, and proximity of outcomes to clinical effects. A composite score was not created because numerical scores can mask weaknesses in different domains.

2.5. Evidence Synthesis

The following figures are derived from the search and filtering results documented in this review. The calculations are consistent: 90 − 7 = 83. 83 − 54 = 29. 29 − 12 = 17. and 17 + 3 = 20.

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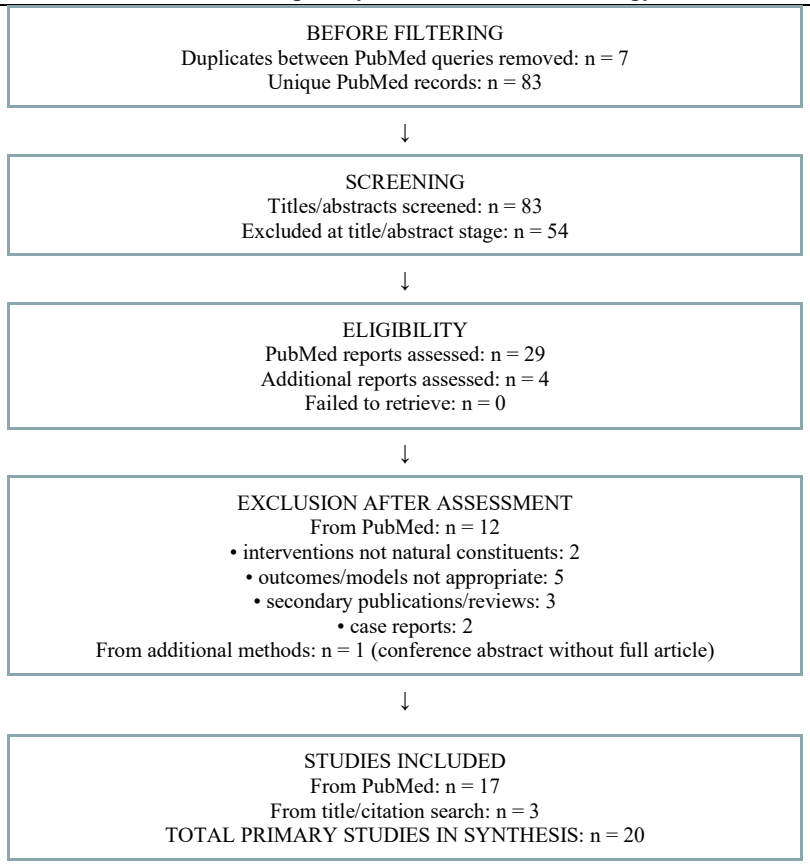

Figure 1. PRISMA 2020 flowchart populated with actual search data

## 3. Results and Discussion

### 3.1. Selection results and composition of evidence

A total of 20 primary studies were included: 7 studies in the analgesia/pain domain, 7 studies in the cognition or neurodegeneration domain, and 6 studies in the pharmacokinetics, drug interactions, or human tolerability domains. Based on design, there were 11 in vivo animal studies, 3 in vitro/computational studies, 5 experimental or human pharmacokinetic studies, and 1 human observational study.

Table 3. Distribution of included study designs

| Design | n | Significance of the Strength of Evidence |
|---|---|---|
| In vivo animals | 11 | Predominantly analgesic and cognitive evidence. Limited human translation. |
| In vitro/computational | 3 | Supports AChE mechanisms, network pharmacology, and CYP inhibition. Does not demonstrate clinical efficacy. |
| Experimental/human PK | 5 | Provides data on dosage, exposure, interactions, and short-term tolerability. Samples are generally small. |
| Observational humans | 1 | Reflects actual use for pain but is susceptible to selection and self-report bias. |

### 3.2. Evidence of analgesia and pain

Table 4. Evidence of analgesia and pain

| Study | Design | Intervention | Key Findings | Limitations |
|---|---|---|---|---|
| Foss et al., 2020 [1] | Rats, chemotherapy-induced neuropathy | Mitragynine | Reduced allodynia. Effects depend on α-adrenoceptor mechanisms. | Preclinical, no chronic/human safety testing. |
| Buckhalter et al., 2021 [2] | Male rats, analgesia and nerve oscillation test | Purified kratom alkaloid extract | Both doses produced analgesia and altered neural network oscillations. | Not a specific neuropathic pain model. Limited dosage/product. |

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|---|---|---|---|---|
| Farkas et al., 2022 [3] | Mice, chemotherapy-induced peripheral neuropathy | Mitragynine | Reduced allodynia with sex-related differences. Involves both adrenergic and opioid receptors. | Animal model and one etiology of neuropathy. |
| Ortiz et al., 2023 [4] | Mice, paclitaxel-induced allodynia | Mitragynine ± cannabidiol | Dose-dependent interactions; the combination may alter antiallodynia and behavioral effects. | Not directly translatable to human regimens. |
| Farkas et al., 2023 [5] | Mice, neuropathic pain and inflammation | Mitragynine | Cannabinoid mechanisms contribute to effects on neuropathic pain, but not inflammatory pain. | Preclinical, model specificity limits generalizability. |
| Mun et al., 2025 [6] | Humans, cross-sectional + ecological momentary assessment | Real-world kratom use | Users report kratom for pain self-management and acute pain changes after use. | Observational. Self-report, no randomization or comparator. |
| Indriani et al., 2025 [7] | Mice, hot-plate and withdrawal model | Crude extract/kratom alkaloids | Analgesia is mediated by the opioidergic and serotonin systems. Repeated exposure also produces withdrawal symptoms. | Tested acute pain, not neuropathic. Extract composition influences results. |

## 3.3. Cognitive and neurodegenerative evidence

Table 5. Cognitive and neurodegenerative evidence

| Studi | Design | Interventions | Key Findings | Limitations |
|---|---|---|---|---|
| Innok et al., 2021 [8] | In silico + in vitro AChE | Mitragynine and related alkaloids | Mitragynine interacts with AChE and exhibits in vitro inhibition (pIC50 3.57). | Preliminary potential, no animal/human Alzheimer's model. |
| Iman et al., 2021 [9] | Mice, 28-day chronic exposure | Mitragynine 1–25 mg/kg | High doses impair learning and memory. A CB1 antagonist partially reverses the deficits. | High i.p. dose, relevance to oral consumption uncertain. |
| Zul Aznal et al., 2022 [10] | Adolescent mice, behavior + metabolomics | Mitragynine/kratom decoction | Adolescent exposure alters cognitive behavior and brain metabolite profiles. | Male animals, no clinical correlation. |
| Suhaimi et al., 2023 [11] | Mice, follow-up into adulthood | Mitragynine/kratom decoction during adolescence | Deficits in object recognition and reference memory persist into adulthood, accompanied by metabolic changes. | Did not assess reversibility or human exposure threshold. |
| Yunusa et al., 2023 [12] | Mice, hippocampal electrophysiology | Mitragynine 1, 5, and ıg/kg for 14 days | Doses of 5–10 mg/kg suppress LTP and neuroplasticity-related proteins. 1 mg/kg shows no similar effects. | Surrogate outcome. Not a neurodegenerative disease model. |
| Yunusa et al., 2024 [13] | Mice, mitragynine withdrawal + epigenetics | Mitragynine, SAHA intervention | Withdrawal results in cognitive deficits and histone changes. An HDAC inhibitor partially reverses the effects. | Withdrawal focus. Not validated in humans. |
| Hossain et al., 2024 [14] | Network pharmacology, docking, and in vitro | M. speciosa extract/constitu | Identifying potential Alzheimer's targets and pathways and early experimental activity signals. | Hypothesis-generating evidence does not prove clinical benefit. |

## 3.4. Pharmacokinetics, drug interactions, and human tolerability

Table 6. Pharmacokinetics, drug interactions, and human tolerability

| Studi | Design/Model | Interventions | Key Findings | Limitations |
|---|---|---|---|---|
| Kamble et al., 2020 [15] | Human liver microsomes | Six kratom alkaloids | Mitragynine and corynantheidine inhibit CYP2D6, and some inhibition of CYP2C19/CYP3A was also detected. | In vitro, the magnitude of clinical interactions depends on actual exposure. |
| Tanna et al., 2022 [16] | Human PK. 6 healthy volunteers | 2 g characterized kratom product | Significant differences in PK between the alkaloids and the long terminal half-life of mitragynine were observed. | The sample size is very small and the single dose is low. |
| Tanna et al., 2023 [17] | Human interaction study. 12 volunteers | 2 g kratom tea + midazolam/dextromethorphan | The AUC and Cmax of midazolam were increased 1.39-fold and 1.50-fold, respectively. There was no significant effect on dextromethorphan. | The single dose is not representative of high-concentration extracts or chronic use. |

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|---|---|---|---|---|
| Huestis et al., 2024 [18] | Randomized dose-escalation. Single dose and 15-day intervals | 500–4000 mg encapsulated leaf powder | Mitragynine/7-OH exposure increased dose-dependently. Steady-state is approximately 7–9 days, and the half-life of mitragynine can be prolonged. | The product is standardized; the results do not automatically apply to all commercial products. |
| Mongar et al., 2024 [19] | Two-period study in healthy volunteers | Kratom tea + onazole | Itraconazole decreased 7-OH exposure and increased mitragynine Cmax, favoring 7-OH formation via CYP3A4. | The sample size is limited; only one potent CYP3A4 inhibitor is used. |
| Prevete et al., 2025 [20] | Phase 1, placebo-controlled, within-subjects. Total 15 volunteers | 5–40 mg Mitragynine | Doses up to 40 mg were tolerated short-term. 5 mg increased arousal/attention, while 40 mg increased amnesia/mild distress. | The single exposure and small sample size do not assess therapeutic efficacy or chronic safety. |

3.5. Neuropathic pain

Four studies directly using models of chemotherapy-induced neuropathy or neuropathic pain have shown a reduction in allodynia after mitragynine [1, 3–5]. The mechanism is not limited to μ-opioid receptors: blockade of α-adrenoceptors, opioid receptors, and cannabinoid pathways reduces the antiallodynia effect in certain models. These findings strengthen the possibility of multi-target pharmacology, but also suggest that responses may differ by neuropathy type, gender, and drug combination.

Human observational studies indicate that pain is a common motivation for use and that there are short-term changes in pain reported by users [6]. However, these designs cannot separate drug effects from expectations, regression to the mean, self-selection of doses, or the use of other substances. There are no randomized controlled trials evaluating kratom or mitragynine for neuropathic pain in patients. Therefore, the correct phrase is “analgesic potential supported by preclinical evidence,” not “proven therapy”.

3.6. Alzheimer's, Parkinson's, and cognition

Two studies support the Alzheimer's hypothesis through AChE inhibition, network pharmacology, molecular docking, and initial in vitro validation [8,14]. These outcomes are useful for target prioritization, but they do not assess effective blood-brain barrier penetration, effects on amyloid or tau in the organism, improvements in disease behavior, or clinical outcomes. This search found no primary studies from 2020–2025 demonstrating the effectiveness of kratom in Alzheimer's or Parkinson's patients.

Conversely, several animal studies have shown impairments in learning, memory, hippocampal LTP, and neuroplasticity proteins after high-dose exposure, adolescence, or withdrawal [9–13]. These findings do not prove that human consumption at all doses produces similar impairments, but they are a signal to consider when interpreting neuroprotective claims. Effects appear to be dose- and context-dependent. In a phase 1 human study, a single low dose signaled mild stimulation, while 40 mg increased amnesia and mild distress [20].

3.7. Pharmacokinetics and drug interactions

Human clinical studies have shown that mitragynine may have a long terminal half-life and reach steady-state after repeated use [16,18]. Because product compositions differ, a “gram leaf powder” dose is not equivalent to a dose of extract or pure mitragynine. Using product labels without alkaloid analysis may lead to erroneous exposure estimates.

In vitro data place CYP2D6 and CYP3A as important interaction pathways [15]. Low-dose clinical trials confirmed increased midazolam exposure consistent with intestinal CYP3A inhibition, while dextromethorphan did not significantly change it [17]. Studies of itraconazole have shown that CYP3A4 is also involved in the formation of 7-hydroxymitragynine [19]. Thus, CYP3A inhibitors or substrates may alter exposure to mitragynine and its active metabolite. The direction and clinical consequences are not always straightforward.

3.8. Security and translation implications

Acute tolerability in healthy volunteers does not equate to the safety of chronic use in patients with comorbidities, polypharmacy, or high-dose product use. Animal studies have shown withdrawal symptoms after repeated exposure [7,13], while available human studies primarily assess PK or single doses. Product standardization, alkaloid quantification, drug interaction monitoring, and dependence assessment should be mandatory components of future clinical trials.

Strengths of this review include a replicable search strategy, a log of the number of results, PMID-based deduplication, a list of included studies, and stated reasons for exclusion. Limitations include the use of a single primary database, screening by a single reviewer, the lack of protocol registration, and the lack of risk of bias assessment by two independent reviewers. Citation searches reduce, but do not eliminate, the possibility of missing studies. Therefore, the results should be viewed as a structured narrative review, not an estimate of effect equivalent to systematic reviews and meta-analyses.

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## 4. Conclusion

Evidence from 2020–2025 suggests that mitragynine has a relatively consistent antiallodynia effect in animal models of neuropathic pain, with contributions from adrenergic, opioid, and cannabinoid pathways. However, human evidence remains limited to real-world use reports, pharmacokinetic studies, drug interactions, and phase 1 clinical trials. No clinical trials have yet demonstrated efficacy for neuropathic pain. For Alzheimer's or Parkinson's, evidence of benefit remains at the computational and in vitro levels. Conversely, some animal studies have shown cognitive impairment and neuroplasticity with high or prolonged exposure. Therefore, claims of "neuroprotection" cannot be substantiated. Future studies need to use standardized products, randomized controlled designs, PK/PD monitoring, evaluation of CYP3A/CYP2D6 interactions, and follow-up on dependence and cognitive function.

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