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REVIEW ARTICLE

Herbal Adaptogens versus Benzodiazepines: A Pharmacological Review in Anxiety Management

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Abstract

Anxiety disorders constitute a major global mental health burden, prompting the need for effective and safer therapeutic strategies. Benzodiazepines (BZDs), long considered a first-line pharmacological treatment, offer rapid symptomatic relief but are limited by risks of dependence, cognitive impairment, and withdrawal. In contrast, herbal adaptogens, including *Withania somnifera*, *Rhodiola rosea*, *Panax ginseng*, and *Bacopa monnieri*, have emerged as promising natural alternatives with multifactorial mechanisms, such as modulation of the hypothalamic-pituitary-adrenal axis, neurotransmitter regulation, and antioxidant effects. This review provides a comparative pharmacological analysis of BZDs and herbal adaptogens in anxiety management, evaluating differences in mechanisms of action, clinical efficacy, safety profiles, risk of dependence, and cognitive-emotional outcomes. While BZDs remain indispensable for acute anxiety episodes, adaptogens demonstrate sustainable anxiolytic benefits with superior tolerability and negligible addiction potential. However, limitations in present research, including variability in herbal preparation and a paucity of high-quality clinical trials, warrant cautious interpretation. Future integrative approaches combining conventional and herbal therapies, supported by standardized, large-scale studies, may offer optimized and holistic management options for anxiety disorders.

Key words: Anxiety management, benzodiazepines, herbal adaptogens, pharmacological comparison, *Withania somnifera*

INTRODUCTION

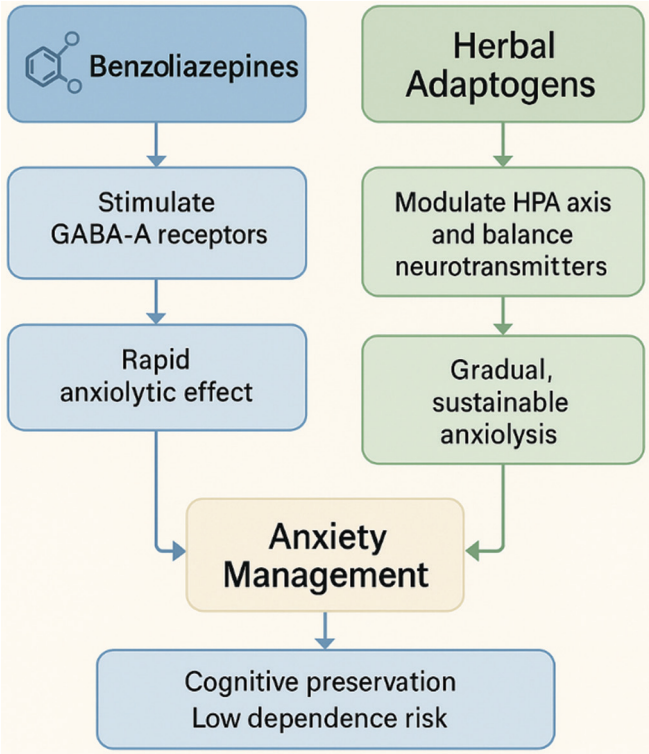
Anxiety disorders are one of the most common types of mental illnesses globally, as 301 million were estimated to be suffering from them by the World Health Organization (2019).[1] Anxiety is characterized by excessive fear, apprehension, and somatic symptoms and greatly affects social, occupational, and physical functioning. The existing pharmacological treatments essentially consist of synthetic anxiolytic agents from which benzodiazepines (BZDs) stand as a blockbuster due to their rapid onset of action and proved efficacy.[2] Nevertheless, the long-term use of BZDs is associated with several adverse effects, such as tolerance, dependence, cognitive dysfunction, and withdrawal symptoms, and alternative therapeutic strategies are to be explored.[3] Natural products, particularly herbal adaptogens, have been receiving more and more attention in recent years as possible interventions to deal with anxiety.[4] Herbal therapeutics, such as adaptogens are known to enhance the resilience of the body to stress through modulation of key homeostatic mechanisms, including the hypothalamic-pituitary-adrenal (HPA) axis and neurochemical signal pathways. There are several known examples, such as *Withania somnifera* (Ashwagandha), *Rhodiola rosea*, and *Panax ginseng,* which have shown anxiolytic activity in pre-clinical and clinical studies with a favorable safety profile.[5] Although there is increasing evidence of the use of adaptogens, a full comparison with conventional BZD therapy

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GRAPHICAL ABSTRACT



has not been conducted. It is essential for future therapeutic approaches to have a pharmacologic review to describe similarities and differences in the mechanisms of action, efficacy, safety, and clinical outcomes.[6] As a consequence, the aim of the present review is to critically appraise and compare the pharmacokinetic as well as pharmacodynamic profiles of BZDs and herbal adaptogens in regard to their respective applications regarding anxiety treatment, and shed light on the positive and negative aspects of such treatment as well as the potential of integrating both types into clinical practice.[7]

PATHOPHYSIOLOGY OF ANXIETY

Neurochemical basis

At the molecular level, anxiety is characterized by an imbalance in key neurotransmitters. Gamma-aminobutyric acid (GABA), the principal inhibitory neurotransmitter in the central nervous system (CNS), plays a critical role in reducing neuronal excitability. A deficiency in GABAergic signaling has been strongly associated with heightened anxiety states.[8] In addition, serotonergic (5-HT) and dopaminergic pathways contribute significantly to emotional regulation, and dysregulation in these systems can exacerbate anxiety symptoms. The noradrenergic system, through excessive release of norepinephrine, also heightens arousal and vigilance, further contributing to the anxiety response.[9]

Role of the HPA axis

The location of the HPA axis is the hypothalamus and pituitary, which respond to stressful situations by secreting corticotropin-releasing hormone (adrenocorticotropic hormone, and cortisol. Chronic hyperactivation of the HPA axis provokes disease and permanent anxiety, and due to persistent elevation of cortisol levels, impaired negative feedback mechanisms.[10] The HPA axis has been found to be dysregulated in both generalized anxiety disorder (GAD) and post-traumatic stress disorder.[11]

Neurotransmitters involved: GABA, serotonin, and dopamine

The anxiolytic effect of many pharmacological agents is mediated through modulation of GABAergic and serotonergic systems. BZDs enhance GABA type A (GABA-A) receptor-mediated chloride influx, promoting neuronal hyperpolarization and anxiolysis.[12] Selective serotonin reuptake inhibitors also demonstrate anxiolytic properties by increasing synaptic serotonin availability. Dopaminergic signaling, although less directly targeted in present anxiolytic therapies, influences motivational and reward aspects of anxiety-related behaviors.[13] The major neurotransmitters implicated in anxiety pathophysiology and their clinical relevance are summarized in Table 1.

BZDs IN ANXIETY MANAGEMENT

BZDs have been pivotal in the pharmacological treatment of anxiety disorders since their discovery in the mid-20th century. Due to their rapid anxiolytic action and relatively favorable safety profile compared to earlier sedatives, BZDs have been widely adopted in both acute and chronic anxiety management. However, concerns regarding their long-term safety, dependence potential, and cognitive side effects have led to critical reevaluation of their clinical role.[10]

Mechanism of action

GABA-A is a ligand-gated chloride channel in the CNS, and BZDs modulate the activity at this receptor. It binds to an allosteric site at the receptor complex that is between the α and γ subunits of the receptor. It increases the binding of the receptor to GABA, and its consequent affinity increases the frequency of chloride ion channel opening events.[9] During action, the resulting hyperpolarization of neuronal membranes decreases neuronal excitability in brain regions known to be involved in the generation of fear and anxiety response, i.e., the amygdala and hippocampus. An overall outcome of anxiolytic, sedative, muscle relaxant, and anticonvulsant action is realized.[11]

Pharmacokinetics and pharmacodynamics

The pharmacokinetic properties of BZDs vary widely among different agents, influencing their onset and duration of action. Lipophilicity, plasma protein binding, hepatic metabolism, and the presence of active metabolites determine their clinical profiles. Short-acting BZDs, such as alprazolam and lorazepam, are often preferred for acute anxiety episodes, while long-acting agents, such as diazepam and clonazepam are suited for managing GADs or preventing withdrawal symptoms.[5] Most BZDs are metabolized hepatically via cytochrome P450 enzymes, predominantly CYP3A4, and undergo subsequent conjugation to inactive glucuronide metabolites for renal excretion. Pharmacodynamically, the intensity of their effect is determined by receptor affinity, intrinsic efficacy, and their ability to cross the blood–brain barrier rapidly.[6]

Clinical applications and efficacy

BZDs are clinically employed in various anxiety-related disorders, including GAD, panic disorder, social anxiety disorder, and situational anxiety. In GAD, they alleviate both psychic and somatic symptoms, providing rapid symptomatic relief. In panic disorder, their immediate onset aids in aborting acute panic attacks and reducing anticipatory anxiety.[8] In social anxiety disorder, agents, such as clonazepam have shown efficacy in improving social performance and reducing fear of scrutiny, BZDs are used adjunctively during the initial phase of antidepressant therapy to counteract activation symptoms. They are also indicated in managing anxiety secondary to medical conditions, such as in patients with myocardial infarction or respiratory illnesses where anxiety exacerbates clinical outcomes.[12] Numerous clinical trials and meta-analyses have demonstrated that BZDs provide superior short-term symptomatic relief compared to placebo, although long-term benefits remain limited due to tolerance development.[13] The pharmacological characteristics and clinical applications of commonly used BZDs in anxiety management are summarized in Table 2.

Adverse effects and limitations

The therapeutic use of BZDs is tempered by a spectrum of adverse effects. Commonly reported side effects include sedation, psychomotor slowing, impaired coordination, memory deficits, and cognitive dysfunction. These effects are particularly problematic in elderly populations, increasing the risk of falls and fractures.[21] BZDs may also contribute to emotional blunting, anhedonia, and a reduction in attentional capacities. Moreover, with continued use, the efficacy of

Table 1: Major neurotransmitters and their role

Neurotransmitter	Role in anxiety	Clinical relevance	Reference
Gamma-aminobutyric acid	Inhibitory neurotransmitter; reduces neuronal excitability	Targeted by benzodiazepines to enhance inhibitory effects	[14]
Serotonin (5-HT)	Regulates mood, emotion, and sleep	SSRIs used to increase synaptic serotonin in anxiety disorders	[15]
Norepinephrine	Mediates arousal, vigilance, and stress responses	Overactivity linked to hypervigilance and panic symptoms	[5]
Dopamine	Involved in motivation, reward, and emotional regulation	Dysregulation may influence anxiety-related anhedonia and stress	[16]
Cortisol (via HPA axis)	Stress hormone; regulates metabolism and immune response	Chronic elevation linked to persistent anxiety and depression	[4]

HPA: Hypothalamic-pituitary-adrenal, SSRIs: Selective serotonin reuptake inhibitors

Table 2: Pharmacological and clinical profile of commonly used benzodiazepines in anxiety management

Drug	Duration of action	Primary indications	Adverse effects	References
Diazepam	Long-acting	GAD, situational anxiety, alcohol withdrawal	Sedation, cognitive impairment, accumulation with prolonged use	[14]
Alprazolam	Short-acting	Panic disorder, acute anxiety episodes	Dependence, severe withdrawal symptoms	[17]
Lorazepam	Intermediate-acting	Acute anxiety, procedural anxiety	Sedation, amnesia, respiratory depression (at high doses)	[18]
Clonazepam	Long-acting	Panic disorder, social anxiety disorder	Drowsiness, psychomotor impairment	[19]
Midazolam	Short-acting (IV use)	Procedural sedation, acute agitation	Respiratory depression (especially IV route)	[20]

GAD: Generalized anxiety disorder

BZDs may diminish due to pharmacological tolerance, necessitating escalating doses to achieve the same anxiolytic effect. This phenomenon complicates long-term management strategies and highlights the importance of limiting BZD prescriptions to short-term or intermittent use.[22]

Risk of dependence and withdrawal

One of the most significant limitations of BZD therapy is the potential for dependence and withdrawal. Physical dependence can develop after several weeks of continuous use, and abrupt discontinuation may precipitate withdrawal symptoms, including rebound anxiety, insomnia, irritability, tremors, palpitations, and, in severe cases, seizures and psychosis. Withdrawal severity is influenced by the half-life of the BZD, the dose, the duration of use, and individual susceptibility factors. Short-acting agents, such as alprazolam, are associated with more intense withdrawal syndromes compared to longer-acting agents, such as diazepam. Therefore, tapering protocols involving gradual dose reduction over weeks to months are recommended to minimize withdrawal risks. Clinical guidelines advocate for cautious prescribing practices, patient education regarding the risks of long-term use, and consideration of alternative therapies where appropriate.[23]

HERBAL ADAPTOGENS IN ANXIETY MANAGEMENT

Concept of adaptogens

Adaptogens are defined as natural substances that increase the body's non-specific resistance to stress and exert a normalizing effect irrespective of the direction of pathological changes. First introduced by Soviet scientist Nikolai Lazarev in the 1940s, the concept of adaptogens encompasses three core properties: They are non-specific in their action, exert a normalizing effect on physiology, and are innocuous to normal body functions. Unlike BZDs, adaptogens do not induce sedation or significant cognitive impairment, making them attractive candidates for long-term use in anxiety management.[24]

Mechanisms of action

The mechanisms by which adaptogens exert anxiolytic effects are multifactorial. Major proposed mechanisms include:

- Modulation of the HPA axis: Adaptogens help normalize cortisol secretion patterns, mitigating the detrimental effects of chronic stress
- Neurotransmitter regulation: Adaptogens influence serotonergic, dopaminergic, and GABAergic signaling pathways, promoting emotional stability
- Antioxidant and anti-inflammatory effects: Many adaptogens reduce oxidative stress and neuroinflammation, factors implicated in anxiety pathophysiology
- Mitochondrial protection and energy homeostasis: Some adaptogens enhance cellular energy production and resilience to metabolic stress.[24]

Prominent adaptogens and their pharmacological profiles

W. somnifera (ashwagandha)

W. somnifera, commonly known as Ashwagandha, is a traditional Ayurvedic herb renowned for its adaptogenic properties. Its active constituents, including withanolides, sitoindosides, and alkaloids, have been shown to modulate the HPA axis, reduce serum cortisol levels, and enhance GABAergic signaling. Pre-clinical studies indicate that Ashwagandha extracts exert anxiolytic effects comparable to lorazepam, without associated sedative side effects. Clinical trials have demonstrated significant reductions in Hamilton anxiety rating scale scores with Ashwagandha supplementation over placebo, with a favorable safety profile.[25]

R. rosea

R. rosea, also known as Arctic root, has been traditionally used in Eastern European and Asian medical systems for enhancing resilience to stress. The key bioactive compounds, rosavins and salidroside, modulate stress-induced changes in monoamine neurotransmitter levels and improve serotonin activity. *Rhodiola* enhances mental performance under stress and reduces symptoms of generalized anxiety and fatigue. Clinical studies suggest that standardized *Rhodiola* extracts produce significant improvements in stress-induced anxiety symptoms, with minimal adverse effects.[26]

P. ginseng

P. ginseng, a central herb in traditional Chinese medicine, contains ginsenosides, which exhibit a wide range of neuroprotective, anti-inflammatory, and adaptogenic activities. Ginsenosides influence the HPA axis, upregulate neurotrophic factors, such as brain-derived neurotrophic factor, and enhance serotonergic transmission. Experimental models suggest anxiolytic-like effects, and some clinical data support the use of *P. ginseng* in improving mood and cognitive functions under stress. However, variability in preparation and dosing necessitates further standardized clinical trials.[27]

Bacopa monnieri

B. monnieri, known as Brahmi in Ayurveda, is rich in bacosides, which exhibit antioxidant, neuroprotective, and neurotransmitter-modulating properties. It has been shown to enhance synaptic communication, promote GABAergic activity, and protect against stress-induced neuronal damage. Clinical studies indicate that *Bacopa* supplementation improves memory, reduces anxiety, and alleviates symptoms of cognitive fatigue. Adverse effects are generally mild, including occasional gastrointestinal discomfort.[28]

Clinical evidence and safety profiles

Clinical investigations into adaptogens have generally demonstrated favorable outcomes for anxiety management, although study methodologies vary widely. Randomized controlled trials (RCTs) on *W. somnifera* have shown up to 44% reductions in anxiety scores compared to baseline, with minimal adverse events reported. *R. rosea* supplementation has been associated with improvements in stress-induced anxiety within 2–4 weeks of treatment.[29] Safety profiles of adaptogens are notably superior to BZDs, with a low incidence of adverse effects and no evidence of dependence or withdrawal phenomena. Nevertheless, challenges, such as variability in herbal preparation, differences in phytochemical composition, and limited long-term data necessitate caution. Standardization of extracts, rigorous quality control, and larger-scale clinical trials are essential for validating adaptogens as mainstream therapeutic options in anxiety management.[30]

COMPARATIVE PHARMACOLOGICAL ANALYSIS

The pharmacological profiles of BZDs and herbal adaptogens reveal significant differences in their mechanisms of action, clinical efficacy, safety profiles, potential for dependence, and impact on cognitive-emotional functioning. A comparative analysis is essential to understand their relative advantages and limitations in anxiety management.[31]

Mechanistic differences

BZDs act primarily by enhancing GABAergic neurotransmission through positive allosteric modulation of GABA-A receptors. This results in rapid, profound neuronal inhibition, accounting for their immediate anxiolytic and sedative effects. However, this non-specific CNS suppression can lead to cognitive and psychomotor impairments; adaptogens exert a more modulatory and restorative effect on stress response systems.[32] They normalize HPA axis activity, regulate neurotransmitter balance (serotonin, dopamine, GABA), reduce oxidative stress, and promote neuroplasticity. Adaptogens do not cause generalized CNS depression, thus preserving cognitive and psychomotor functions while mitigating anxiety.[33] The mechanistic pathways of BZDs and herbal adaptogens in anxiety management are illustrated in Figure 1.

Clinical efficacy comparison

BZDs exhibit superior efficacy in the short-term reduction of acute anxiety symptoms, providing rapid relief within hours of administration. Their role is particularly critical in severe panic attacks, procedural anxiety, and emergency psychiatric settings. Adaptogens, although slower in onset (requiring days to weeks), offer gradual yet sustainable anxiolytic benefits. Clinical studies suggest that standardized extracts of *W. somnifera* and *R. rosea* produce clinically meaningful reductions in anxiety scores, comparable to first-line pharmacotherapies over extended periods. However, head-to-head trials comparing adaptogens directly with BZDs remain limited.[34]

Safety and tolerability

BZDs are associated with a broad range of adverse effects, including sedation, cognitive dulling, psychomotor impairment, and paradoxical reactions, such as increased irritability. Their use in elderly populations carries significant

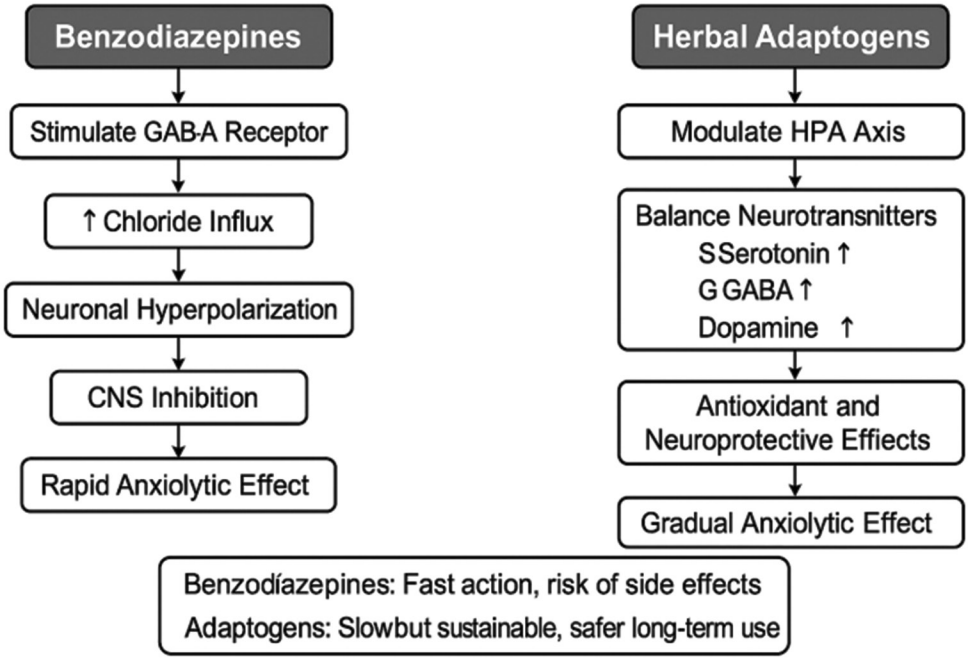


Figure 1: Mechanistic overview of benzodiazepines and herbal adaptogens in anxiety management

Table 3: Comparative pharmacological profile of benzodiazepines and herbal adaptogens in anxiety management[13,14]

Feature	Benzodiazepines	Herbal adaptogens
Mechanism of action	Positive allosteric modulation of GABA-A receptors	Modulation of the HPA axis, neurotransmitter balancing, antioxidative action
Onset of action	Rapid (within hours)	Gradual (over days to weeks)
Clinical efficacy	Highly effective in acute anxiety	Effective for chronic and stress-induced anxiety
Risk of dependence	High	Negligible
Cognitive effects	Impairment of memory, attention, psychomotor skills	Cognitive preservation or enhancement
Tolerability	Moderate (sedation, psychomotor impairment, withdrawal)	High (mild adverse effects, such as gastrointestinal discomfort)
Withdrawal symptoms	Common and sometimes severe	No withdrawal symptoms reported

HPA: Hypothalamic pituitary adrenal, GABA-A: Gamma-aminobutyric acid type A

risks, including falls and fractures. Adaptogens generally exhibit excellent tolerability with minimal side effects. Mild gastrointestinal disturbances are the most commonly reported adverse events. Importantly, adaptogens do not impair cognitive performance or psychomotor coordination, and they have not been associated with serious adverse events in clinical trials when used at recommended dosages.[35]

Cognitive and emotional outcomes

BZD therapy is associated with impairments in memory consolidation, attention, executive function, and emotional blunting. Long-term use may exacerbate cognitive decline, particularly in older adults. Adaptogens, conversely, may enhance cognitive and emotional resilience. *B. monnieri* has demonstrated pro-cognitive effects in both healthy and cognitively impaired individuals. *R. rosea* and *P. ginseng* have shown benefits in improving mood, reducing mental fatigue, and enhancing cognitive performance under stress. Thus, from a cognitive and emotional outcome perspective, adaptogens not only avoid the impairments associated with BZDs but may actively promote neurocognitive health.[36] A comparative summary of the pharmacological and clinical profiles of BZDs and herbal adaptogens is presented in Table 3.

LIMITATIONS IN PRESENT RESEARCH

Despite the promising findings regarding both BZDs and herbal adaptogens in anxiety management, several limitations persist in the present body of research. For BZDs, most clinical studies focus on short-term outcomes, with relatively few addressing the long-term cognitive, emotional, and physiological consequences of chronic use. There is also limited high-quality, head-to-head comparative research between BZDs and emerging alternative therapies, making it difficult to evaluate relative benefits comprehensively. In the context of herbal adaptogens, while pre-clinical and small-scale clinical trials have demonstrated encouraging anxiolytic effects, methodological inconsistencies, such as variability in extract standardization, dosing regimens, and outcome measures, reduce the generalizability of findings.[5] Furthermore, the majority of adaptogen studies suffer from small sample sizes, short follow-up periods, and a lack of rigorous placebo-controlled designs. Regulatory frameworks for herbal products also vary globally, leading to concerns regarding the quality, purity, and reproducibility of adaptogen formulations used in research. Therefore, future studies must prioritize standardized, large-scale RCTs, direct comparative analyses with conventional therapies, and long-term safety evaluations to establish definitive clinical guidelines for the use of herbal adaptogens in anxiety management.[6]

CONCLUSION

Anxiety disorders remain a major global health concern, necessitating effective and safe therapeutic strategies. BZDs continue to offer rapid symptomatic relief and remain invaluable for acute anxiety management; however, their long-term use is constrained by significant risks of dependence, cognitive impairment, and withdrawal symptoms. In contrast, herbal adaptogens present a promising natural alternative, with mechanisms that modulate the stress response, enhance neurotransmitter balance, and promote neuroprotection without inducing generalized CNS depression. Clinical evidence, although still evolving, suggests that adaptogens, such as *W. somnifera*, *R. rosea*, *P. ginseng*, and *B. monnieri* provide sustainable anxiolytic benefits with superior safety profiles. Nevertheless, substantial gaps in high-quality, standardized research remain, particularly in the context of large-scale RCTs and direct comparisons with established pharmacotherapies. A balanced, evidence-based approach that incorporates both conventional and complementary treatments, guided by individualized patient needs and risk profiles, is essential for optimizing anxiety management in clinical practice. Future research should aim to integrate adaptogens more robustly into evidence-based psychiatric

care, paving the way for safer and more holistic therapeutic options.

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