Therapeutic Potential of *Bacopa monnieri* in Kindling-Induced Post-Ictal Depression: A Comprehensive Review

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ABSTRACT: Epilepsy, a neurological disorder characterized by recurrent seizures, is frequently associated with post-ictal depression, a significant mood disturbance occurring after seizures. This mood disorder severely impacts the quality of life and complicates the management of epilepsy. The kindling model, which induces chronic seizure susceptibility through repeated stimulation, is widely used to study epilepsy and its comorbidities, including post-ictal depression. *Bacopa monnieri*, a traditional Ayurvedic herb known for its neuroprotective, antioxidant, and mood-stabilizing properties, has shown potential in managing mood disorders associated with epilepsy. This review examines the therapeutic effects of *Bacopa monnieri* in kindling-induced post-ictal depression, exploring its mechanisms of action, including antioxidant activity, reduction of neuroinflammation, and neurotransmitter modulation. Preclinical studies indicate that *Bacopa monnieri* may alleviate depressive symptoms in epilepsy by countering oxidative stress, restoring neurotransmitter balance, and protecting neuronal integrity. Future directions, including the need for clinical trials and standardized dosing protocols, are discussed to evaluate *Bacopa monnieri* as an adjunct treatment for mood disturbances in epilepsy. This review highlights *Bacopa monnieri*'s potential to improve quality of life in patients suffering from post-ictal depression and provides a foundation for further research into its clinical applications.

KEYWORDS: Epilepsy, *Bacopa Monnieri*, Post-Ictal Depression, Neurological Disorder.

1. INTRODUCTION-

Epilepsy is a chronic neurological disorder affecting over 50 million people globally, marked by recurring, spontaneous seizures due to excessive and synchronous neuronal activity in the brain. Beyond seizures, epilepsy patients often suffer from various neuropsychiatric comorbidities, with depression being one of the most common and debilitating. [1] Depression related to epilepsy can manifest as either interictal depression, which occurs between seizures, or post-ictal depression, which follows a seizure and can persist for minutes to days. Post-ictal

depression contributes to poor quality of life, increased risk of suicide, and challenges in treatment for epilepsy patients. [2]

Post-ictal depression is particularly difficult to manage as it is influenced by complex neurochemical, structural, and functional changes in the brain due to repeated seizures. These changes include increased neuroinflammation, oxidative stress, neurotransmitter imbalances, and dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis. [3] Understanding these mechanisms is critical to developing effective treatments, yet many currently available options for epilepsy focus on seizure control and do not sufficiently address the emotional and behavioral complications that accompany the disorder. There is a growing need for safe, effective therapies that can simultaneously address both seizure activity and the psychological impact of epilepsy, particularly post-ictal depression. [4]

To investigate treatments for epilepsy-related mood disturbances, researchers use animal models like kindling, a widely accepted model for studying seizure susceptibility and chronic epilepsy. Kindling involves repeated sub-threshold electrical or chemical stimulation of brain regions to induce a progressive increase in seizure severity and frequency, eventually leading to spontaneous seizures. [5] The kindling model not only replicates the process of chronic epilepsy but also mirrors neurobiological and behavioral changes associated with post-ictal depression, making it an invaluable tool for preclinical research. [6]

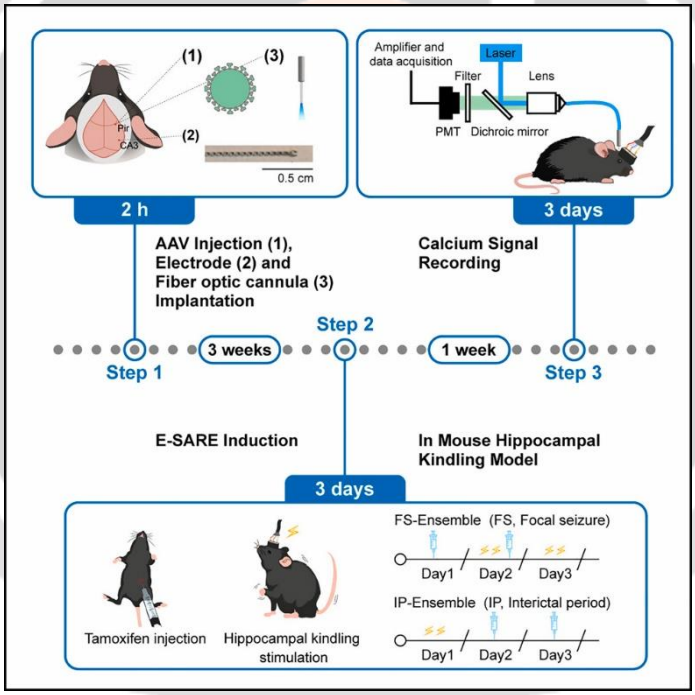


Fig 1. Kindling Model.

In recent years, there has been increased interest in natural compounds and herbal medicines as potential adjunct therapies for epilepsy and its psychiatric comorbidities. Bacopa monnieri (also known as Brahmi) is one such herb with a long history in Ayurvedic medicine, traditionally used for its cognitive-enhancing, anxiolytic, and neuroprotective effects. The primary active components of Bacopa monnieri are bacosides, which have been shown to possess antioxidant, anti-inflammatory, and neurotransmitter-modulating properties. Given these pharmacological effects, Bacopa monnieri has gained attention as a potential treatment for mood disorders and neurological conditions, including epilepsy. [7]

Preclinical studies suggest that Bacopa monnieri may have promising effects in reducing depressive symptoms associated with neurological disorders through multiple mechanisms. It reduces oxidative stress by scavenging free radicals and enhancing antioxidant enzyme activity, which is crucial in preventing neurodegeneration in epilepsy. [8]

Fig 2. Bacopa Monnieri.

Additionally, Bacopa monnieri modulates inflammatory pathways, potentially lowering levels of pro-inflammatory cytokines that contribute to seizure-induced neuroinflammation and mood disturbances. The herb also affects neurotransmitter systems involved in mood regulation, including serotonin, dopamine, and GABA, which may help counteract the neurotransmitter imbalances associated with post-ictal depression. [9]

This review aims to provide a comprehensive analysis of Bacopa monnieri's potential as a therapeutic agent for kindling-associated post-ictal depression. We examine the pathophysiology of post-ictal depression, the role of oxidative stress, neuroinflammation, and neurotransmitter dysregulation in epilepsy-induced mood disorders, and the pharmacological properties of Bacopa monnieri that may counteract these effects. By evaluating the preclinical evidence, this review seeks to determine whether Bacopa monnieri can be considered a viable adjunct treatment for mood disturbances in epilepsy and to identify future directions for research, including the potential for clinical application. This analysis aims to provide insights into a holistic approach to managing epilepsy and its comorbidities, addressing both seizure control and the often-overlooked psychological impacts of the disorder.

2. PATHOPHYSIOLOGY OF POST-ICTAL DEPRESSION

Post-ictal depression is a mood disturbance following a seizure, characterized by symptoms like low mood, fatigue, anxiety, and cognitive impairment. While the exact mechanisms of post-ictal depression is remain complex, several neurobiological processes are thought to contribute, including neurotransmitter imbalances, structural brain changes, altered neuroinflammatory responses, and disruptions in neural connectivity. [10] Below is an overview of the main factors involved in the pathophysiology of post-ictal depression.

1. Neurotransmitter Imbalances [11]

Seizures alter the brain’s neurotransmitter systems, leading to an imbalance that may contribute to depressive symptoms in the post-ictal period.

- GABAergic and Glutamatergic Systems: The GABAergic (inhibitory) and glutamatergic (excitatory) systems are heavily involved in seizure activity. Post-seizure, there is often a relative decrease in GABA levels and an increase in glutamate, leading to excitotoxicity and mood disturbances. This imbalance can contribute to the dysregulation of mood and cognition seen in post-ictal depression.

- Serotonin Deficiency: Serotonin, a key neurotransmitter in mood regulation, is often decreased following seizures. Low serotonin levels can result in depressive symptoms, fatigue, and anxiety, making it a significant factor in post-ictal depression.

- Dopaminergic Dysregulation: Dopamine levels fluctuate post-seizure, which may lead to impaired motivation, anhedonia, and other depressive symptoms. Dopaminergic dysregulation is especially relevant in limbic and reward-processing areas affected by seizure activity.

2. Neuroinflammatory Responses [12]

Seizures trigger an inflammatory response in the brain, which can contribute to post-ictal mood disturbances.

- Release of Pro-Inflammatory Cytokines: Seizure activity leads to the release of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6. These cytokines are associated with neuroinflammation,

which has been linked to mood disorders, including depression. Elevated cytokine levels in the post-ictal period may contribute to depressive symptoms.

- Activation of Microglia and Astrocytes: Following a seizure, microglial cells and astrocytes are activated, which can further enhance neuroinflammation. Activated glial cells release inflammatory mediators that may impair synaptic function and contribute to mood changes.
- Blood-Brain Barrier Disruption: Seizures can increase the permeability of the blood-brain barrier (BBB), allowing immune cells and other inflammatory molecules to enter the brain, exacerbating inflammation and potentially contributing to mood disturbances.

3. Hypothalamic-Pituitary-Adrenal (HPA) Axis Dysregulation [13]

The HPA axis, which regulates stress responses, often becomes dysregulated following seizures, affecting mood and emotional regulation.

- Elevated Cortisol Levels: Seizures can stimulate cortisol release from the adrenal glands, leading to elevated cortisol levels post-seizure. High cortisol is associated with stress and depression and may contribute to post-ictal depressive symptoms.
- Altered Corticotropin-Releasing Hormone (CRH): Seizure activity can affect CRH release from the hypothalamus, which influences the HPA axis. Changes in CRH levels may contribute to dysphoria and mood disruptions after a seizure.
- Long-Term HPA Axis Changes: Chronic seizures may lead to sustained changes in HPA axis function, contributing to recurrent post-ictal depression and increasing the risk of long-term mood disorders.

4. Neural Network Disruption and Connectivity Changes [14]

Seizures disrupt normal neural connectivity and synaptic function, leading to altered communication between brain regions involved in mood and cognition.

- Altered Limbic Circuitry: Seizures often affect the limbic system, which includes structures such as the amygdala, hippocampus, and prefrontal cortex that are involved in emotion regulation. Post-ictal disruptions in these areas may impair mood regulation, leading to depressive symptoms.
- Impaired Connectivity: Post-seizure, neural connectivity between regions involved in emotional processing and executive function (e.g., prefrontal cortex to limbic structures) is often disrupted. This impaired connectivity can contribute to difficulties in mood regulation and cognitive function in the post-ictal phase.
- Neuroplasticity and Synaptic Changes: Repeated seizures can cause long-term synaptic changes, including alterations in synaptic plasticity, which may influence mood and contribute to a heightened susceptibility to depression after each seizure.

5. Mitochondrial Dysfunction and Energy Deficits [15]

Seizures increase energy demands on neurons, potentially leading to mitochondrial dysfunction and contributing to post-ictal depression.

- Oxidative Stress: Seizures generate reactive oxygen species (ROS) that damage mitochondrial components, leading to oxidative stress and reduced ATP production. This energy deficit can impair cellular function, especially in high-energy-demand areas like the brain, contributing to fatigue and mood disturbances.
- Mitochondrial Dysfunction: Mitochondrial dysfunction may impair energy metabolism and increase susceptibility to oxidative damage, potentially leading to neuronal injury and contributing to mood disturbances in the post-ictal period.
- Lactic Acid Buildup: Due to the energy demands during a seizure, lactic acid accumulates as a byproduct of anaerobic metabolism. Elevated lactic acid levels can contribute to post-seizure fatigue and malaise.

6. Genetic and Epigenetic Factors [16]

Genetic predispositions and epigenetic modifications may also contribute to an individual's risk of developing post-ictal depression.

- Polymorphisms in Neurotransmitter Receptor Genes: Genetic variations in serotonin, dopamine, and GABA receptor genes may influence an individual's susceptibility to mood disturbances following seizures.
- Epigenetic Modifications: Seizure activity can lead to epigenetic changes, such as DNA methylation and histone modification, affecting gene expression related to mood regulation. These epigenetic modifications may contribute to the persistence of depressive symptoms in individuals with repeated seizures.

3. *BACOPA MONNIERI*: CHEMICAL STRUCTURE & BIOLOGICAL ACTIVITIES

Bacopa monnieri, commonly known as Brahmi, is a traditional medicinal herb widely used in Ayurvedic medicine, especially for its cognitive-enhancing and neuroprotective properties. It has a diverse phytochemical profile, including unique compounds that contribute to its therapeutic benefits, such as antioxidants, anti-inflammatory agents, & cognitive enhancers. [17]

Fig 3. Brahmi (Bacopa monnieri).

1. Chemical Structure of Bacopa monnieri Compounds [18,19]

The key active compounds in Bacopa monnieri are *bacosides*, which are a group of saponins and triterpenoid glycosides. Bacosides are categorized into bacoside A and bacoside B, each with distinct sub-structures contributing to the plant's therapeutic potential.

- Bacoside A: This is a mixture of multiple saponins, primarily including bacoside A3, bacopaside II, bacopaside X, and bacopasaponin C. These saponins contain glycoside-linked sugar moieties that are responsible for the neuroprotective effects of Bacopa.

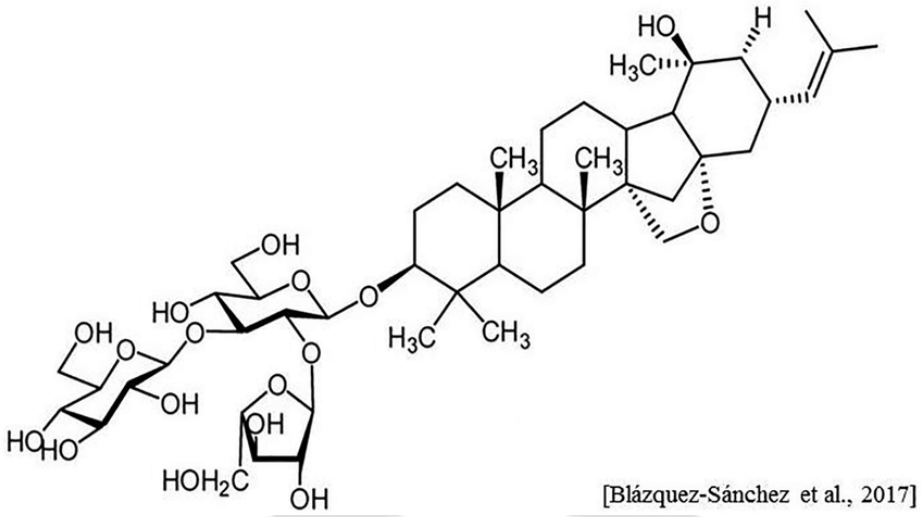
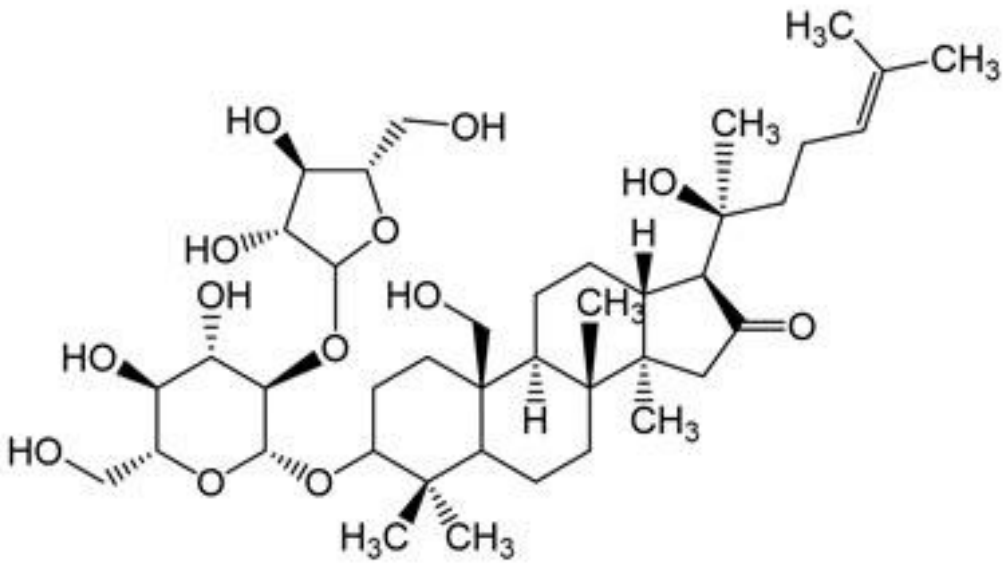


Fig 4. Bacoside A

- Bacoside B: While similar to bacoside A, bacoside B is believed to be a degradation product and shares a similar structure. It also contributes to the herb's antioxidant and neuroprotective properties.

Fig 5. Bacoside B



Other compounds in Bacopa monnieri include *bacopasides*, *alkaloids* (such as brahmine and herpestine), *flavonoids* (e.g., luteolin and apigenin), and *phytosterols* (such as stigmasterol and β-sitosterol).

2. Biological Activities of Bacopa monnieri [20,21,22]

Bacopa monnieri's compounds exhibit a range of biological activities, making it beneficial for neuroprotective, anti-inflammatory, antioxidant, and anxiolytic applications.

2.1 Neuroprotective and Cognitive-Enhancing Effects

Bacopa monnieri is best known for its cognitive-enhancing properties. These compounds improve memory, attention, and learning by multiple mechanisms:

- Cholinergic Modulation: Bacosides enhance the activity of acetylcholine, a neurotransmitter essential for memory and learning.
- Neurotransmitter Modulation: Studies suggest that Bacopa may modulate dopamine and serotonin systems, contributing to mood stabilization and cognitive function.
- Synaptic Plasticity: Bacopa has been shown to increase dendritic branching and synaptic plasticity, mechanisms critical for memory and cognitive flexibility.

2.2 Antioxidant Activity

Oxidative stress is a contributor to neuronal damage and aging. Bacopa monnieri helping to scavenge free radicals and protect against oxidative damage:

- Scavenging of Reactive Oxygen Species (ROS): Bacosides act as free radical scavengers, neutralizing ROS and reducing lipid peroxidation in neural membranes, thereby protecting cellular integrity.
- Induction of Endogenous Antioxidants: Bacopa upregulates endogenous antioxidant enzymes like superoxide dismutase (SOD), catalase, and glutathione peroxidase, enhancing the cell's intrinsic antioxidant defense.

2.3 Anti-Inflammatory Effects

Chronic inflammation is implicated in neurodegeneration and various psychiatric disorders. Bacopa monnieri exhibits anti-inflammatory properties by modulating inflammatory pathways:

- Inhibition of Pro-Inflammatory Cytokines: Bacosides inhibit the release of pro-inflammatory cytokines, such as TNF-α, IL-6, and IL-1β, reducing inflammation in neural tissues.
- NF-κB Pathway Inhibition: Bacopa monnieri suppresses the nuclear factor-kappa B (NF-κB) pathway, a key transcription factor involved in inflammatory responses, thus reducing inflammation-driven cellular damage.

2.4 Anxiolytic and Antidepressant Properties

Bacopa monnieri has shown promise as an adaptogen with anxiolytic and antidepressant effects. Its influence on neurotransmitter pathways is help to contribute to mood enhancement:

- Serotonergic and Dopaminergic Modulation: Bacopa influences the release and regulation of serotonin and dopamine, which play a role in mood regulation.
- Reduction of Corticosterone Levels: Bacopa monnieri has been observed to reduce corticosterone, a stress hormone, which could help counteract stress-induced depression and anxiety.

2.5 Neuroprotective Against Amyloid Beta and Neurotoxins

The neuroprotective effects of Bacopa monnieri extend to models of neurodegeneration, such as Alzheimer's disease:

- Inhibition of β-Amyloid Accumulation: Bacopa has been shown to inhibit β-amyloid aggregation, a hallmark of Alzheimer's disease, thereby protecting neural cells from amyloid toxicity.
- Protection Against Neurotoxicity: Bacopa mitigates neurotoxic damage caused by heavy metals and toxins, providing broad-spectrum neuroprotection.

2.6 Anticonvulsant and Anti-Kindling Activity

Bacopa monnieri has shown anticonvulsant properties, which make it beneficial in managing epilepsy and seizures:

- Reduction in Seizure Threshold: Bacopa increases the seizure threshold and reduces seizure frequency, likely through GABAergic modulation and antioxidant actions.
- Inhibition of Kindling Progression: Kindling, a process by which repeated seizures lead to increased seizure susceptibility and severity, is attenuated by Bacopa. This property may prevent progressive worsening of seizure conditions.

4. PHARMACOLOGICAL MECHANISMS OF *BACOPA MONNIERI* IN POST-ICTAL DEPRESSION (23,24,25)

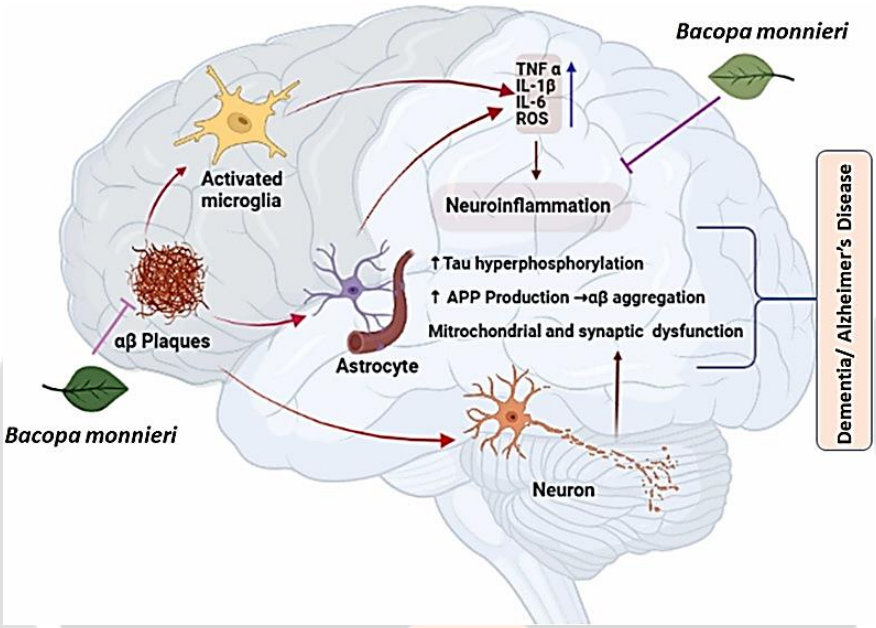


Fig 6. Bacopa Monnieri In Post-Ictal Depression

1. Modulation of Neurotransmitter Systems

Post-ictal depression is often linked to neurotransmitter imbalances, including deficiencies in serotonin, dopamine, and GABA activity. *Bacopa monnieri* contains active compounds, particularly bacosides, that help restore balance to these neurotransmitter systems, potentially alleviating mood disturbances in the post-ictal period.

- Serotonin Regulation: Bacosides in *Bacopa* enhance serotonin levels by inhibiting its reuptake and facilitating receptor activity, which can elevate mood and reduce anxiety. These actions on the serotonergic system may counteract the serotonin deficiency commonly observed in post-ictal depression.
- Dopaminergic Modulation: *Bacopa* also modulates dopamine levels, which are critical for motivation and emotional stability. Dopaminergic dysregulation is a known factor in post-ictal depression, and *Bacopa*'s ability to stabilize dopamine may help in managing depressive symptoms.
- GABAergic Support: *Bacopa monnieri* has been found to support GABA activity, helping to calm neural excitability post-seizure. By enhancing GABAergic inhibition, *Bacopa* can counterbalance excitotoxic effects from elevated glutamate levels during seizures, potentially preventing mood destabilization and neurotoxicity.

2. Anti-Inflammatory and Neuroprotective Effects

Seizures induce a neuroinflammatory response that contributes to cellular damage and mood disturbances. *Bacopa monnieri* is rich in antioxidants and has significant anti-inflammatory effects, which may mitigate the neuroinflammation associated with post-ictal states.

- Reduction of Pro-Inflammatory Cytokines: *Bacopa* inhibits the release of pro-inflammatory cytokines, such as TNF-α, IL-1β, and IL-6, which are elevated post-seizure and implicated in mood disturbances. By reducing these cytokines, *Bacopa* may help protect neurons from inflammation-induced damage and mood dysregulation.
- Microglial and Astrocyte Modulation: *Bacopa*'s anti-inflammatory properties also extend to modulating glial cell activity. Activated microglia and astrocytes contribute to neuroinflammation and neuronal damage after seizures. *Bacopa* inhibits overactivation of these glial cells, which may help preserve synaptic integrity and stabilize mood.
- Antioxidant Action: *Bacopa* enhances endogenous antioxidants like superoxide dismutase (SOD) and catalase, scavenging reactive oxygen species (ROS) generated during seizures. This antioxidant action protects neurons from oxidative stress, which is crucial in preventing mood disturbances associated with cellular damage.

3. HPA Axis Modulation and Stress Reduction

The hypothalamic-pituitary-adrenal (HPA) axis is often dysregulated following seizures, leading to elevated cortisol and stress responses that contribute to mood disturbances. *Bacopa monnieri*'s adaptogenic properties may help restore HPA axis balance.

- Cortisol Reduction: *Bacopa* has been shown to reduce cortisol levels, which can be elevated post-seizure and exacerbate depressive symptoms. By normalizing cortisol, *Bacopa* may relieve stress-related components of post-ictal depression.
- Regulation of Corticotropin-Releasing Hormone (CRH): CRH is a stress hormone released by the hypothalamus that can trigger cortisol release. *Bacopa* may attenuate CRH levels, helping to prevent prolonged stress responses that contribute to mood disturbances in the post-ictal phase.

4. Neuroplasticity and Synaptic Health

Repeated seizures can lead to structural changes in the brain, particularly in regions involved in mood and cognition. *Bacopa monnieri* has been found to promote neuroplasticity and synaptic health, which may counteract the deleterious structural changes associated with post-ictal depression.

- Promotion of Dendritic Growth and Synaptic Plasticity: *Bacopa* supports dendritic branching and increases synaptic plasticity, essential for learning, memory, and mood regulation. These properties may help in the recovery of neural circuits disrupted by seizures.
- Prevention of Synaptic Loss: Seizure activity can lead to synaptic loss and impair connectivity between mood-regulating brain regions, such as the prefrontal cortex and limbic structures. By promoting synaptic health, *Bacopa* may help preserve communication between these regions and mitigate depressive symptoms.

5. Mitochondrial Support and Energy Regulation

Mitochondrial dysfunction and energy deficits following seizures can impair cellular health, leading to fatigue and mood disturbances. *Bacopa monnieri* provides mitochondrial support, potentially alleviating these effects.

- Mitochondrial Protection: Bacosides protect mitochondria from oxidative damage, preserving their function and ensuring steady ATP production. This protection is essential for maintaining energy levels and preventing cellular damage, which can contribute to depressive symptoms.
- Reduction of Lactic Acid Accumulation: Seizures often lead to lactic acid buildup, which contributes to fatigue and malaise in the post-ictal period. *Bacopa* aids in reducing lactic acid and enhancing cellular recovery, potentially improving mood and energy levels post-seizure.

6. Epigenetic Modulation and Gene Expression

Repeated seizures can induce epigenetic changes affecting genes involved in mood regulation. *Bacopa monnieri* may influence these epigenetic pathways, helping to stabilize gene expression and prevent recurrent post-ictal depression.

- Histone Modification and DNA Methylation: *Bacopa* has been shown to affect histone acetylation and DNA methylation, two key mechanisms in gene expression. By modulating these processes, *Bacopa* may help prevent long-term changes in genes associated with mood regulation, reducing the risk of chronic depression.
- Regulation of Genes Associated with Neuroprotection: *Bacopa* upregulates genes involved in antioxidant defense, anti-inflammatory pathways, and synaptic plasticity, promoting resilience to seizure-induced mood disturbances.

5. FUTURE DIRECTIONS AND CLINICAL IMPLICATIONS

1. Clinical Trials for Human Validation:

Rigorous clinical trials are needed to verify BM's efficacy in patients with post-ictal depression. These studies should focus on dosing regimens, long-term safety, and effects on cognitive and mood parameters in epilepsy patients.

2. Exploring Mechanistic Effects in Humans:

Studies should investigate BM's impact on neurotransmitter levels, oxidative stress markers, and inflammatory profiles in human brains to elucidate its effects on post-ictal depression.

3. Combination Therapy Potential:

Research should evaluate BM as an adjunctive treatment with AEDs or antidepressants, potentially lowering the required doses of conventional drugs and reducing their side effects.

4. Dose Optimization and Standardization:

Future studies should focus on establishing optimal dosages of BM and standardizing active compound content in formulations for consistent clinical efficacy.

5. Exploration of Long-Term Neuroprotective Effects:

Investigating whether prolonged BM treatment can provide neuroprotection in chronic epilepsy cases may reveal preventive strategies against seizure progression & post-ictal mood disorders.

6. Personalized Medicine Approaches:

Research into genetic factors and biomarkers that predict responsiveness to BM could lead to more personalized treatment approaches.

7. Evaluation of Cognitive and Quality-of-Life Improvements:

Studies should examine BM's effects on cognitive function and quality of life in epilepsy patients to determine whether it can address both mood and cognitive impairments related to kindling-induced seizures.

Clinical Implications

If validated in clinical settings, *Bacopa monnieri* could become a valuable addition to the treatment options for post-ictal depression, offering a natural and relatively safe alternative to traditional antidepressants. As a complementary therapy, *Bacopa* may help address the unique mental health needs of individuals with epilepsy, who often face limitations with current medications due to side effects or drug interactions. Given its

neuroprotective properties, *Bacopa monnieri* could also serve as a preventative therapy for reducing long-term brain damage associated with seizures, which may lead to better mental health outcomes over time.

By establishing *Bacopa monnieri*'s place in the clinical management of post-ictal depression, healthcare providers may offer patients an integrative approach that combines standard treatments with evidence-based herbal therapies. This integrative model could foster a more holistic management plan for individuals with epilepsy, emphasizing both neurological health and mental well-being.

6. CONCLUSION-

This review underscores the potential of *Bacopa monnieri* as a natural therapy for managing post-ictal depression associated with kindling-induced seizures. *Bacopa monnieri*, a traditional herb widely recognized for its brain-boosting and stress-relieving properties, offers multiple benefits that could make it valuable in the treatment of post-ictal mood disturbances, which often follow repeated seizure activity. Preclinical studies show that *Bacopa monnieri* can positively influence neurotransmitter systems, such as serotonin and dopamine, which are crucial for mood regulation and often disrupted by seizures. By boosting levels of these "feel-good" neurotransmitters, *Bacopa* may help reduce depressive symptoms after seizures. Additionally, *Bacopa* exhibits significant anti-inflammatory and antioxidant properties. Seizures cause inflammation and oxidative stress in the brain, which contribute to cellular damage and can worsen mood disturbances. *Bacopa* counteracts these harmful effects, helping protect brain cells and reduce the negative impact of repeated seizures on mood. While the preclinical findings are promising, it is essential to validate these effects in human studies. Clinical trials will be necessary to confirm *Bacopa monnieri's* effectiveness in managing post-ictal depression, to identify safe and effective dosages, and to monitor any possible side effects. If these studies are successful, *Bacopa monnieri* could be a valuable addition to the treatment options for people with epilepsy, offering a natural, complementary approach to improving mental health and quality of life in individuals experiencing post-seizure depression.

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