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How Does Glycine/GABA Inhibition Exacerbate the Vicious Correlative Cycle of Sleep Paralysis and Bipolar Disorder Mania?

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Abstract

Biological functions related to sleep and emotional processing are integral to human brain function but often remain unexamined in the context of disorders such as Insomnia and Bipolar Disorder (BD). While genetic factors are recognized as key contributors to these conditions, the role of specific biochemical irregularities, particularly in neurotransmitter systems, has been less emphasized. This paper explores the critical involvement of inhibitory neurotransmitters—specifically GABA and glycine—in the pathophysiology of Sleep Paralysis (SP) and BD. Both neurotransmitters are essential for regulating brain activity, with glycine influencing sleep-wake cycles through glutamatergic modulation and GABA playing a crucial role in mood and sleep regulation. Dysregulation of these systems, including the irregular inhibition of GABA/pGABA and glycine, has been implicated in triggering or exacerbating symptoms of manic episodes in BD and increasing the severity of sleep paralysis in SP. Furthermore, the comorbidity of these conditions creates a vicious cycle, where the imbalance of neurotransmitters exacerbates both disorders. This paper discusses the biochemical underpinnings of this cycle and suggests that targeted psychopharmacological and psychodynamic interventions are necessary to break the cycle, offering potential preventative measures and therapeutic strategies for those suffering from these interconnected disorders.

Keywords

Bipolar Disorder, Sleep Paralysis, Neurobiology, GABA (Gamma-Aminobutyric Acid), Glycine, Neurotransmitter Dysregulation, Psychopharmacological Interventions, Molecular Psychiatry

1. Introduction

Biological functions, including sleep and emotional processing, have become incredibly second nature for the human brain today, leading to a lack of awareness of cause/sustainment. Conditions such as Insomnia and Bipolar Disorder have become so commonplace that the anatomical implications are often overlooked. Undoubtedly, genetic predisposition plays a significant role in the existence of both Bipolar Disorder and sleep abnormalities like Sleep Paralysis. However, specific biochemical irregularities contribute significantly to onset symptoms, prolonged/exaggerated episodes, and co-morbidity. Sleep and mood regulation processes are complex and involve numerous biochemical pathways such as the Krebs cycle, CNS activity, dopaminergic systems, and neuroplasticity. However, the role of inhibitory neurotransmitters—particularly GABA and glycine—emerges as a critical factor in both Sleep Paralysis (SP) and Bipolar Disorder (BD). These neurotransmitters are essential for modulating brain activity, and their dysregulation may contribute significantly to both conditions. For instance, glycine influences sleep-wake regulation by modulating glutamatergic systems, while GABA, the primary inhibitory neurotransmitter, directly impacts mood regulation and sleep quality. The imbalance of these neurotransmitters may act as a trigger or an exacerbating factor for the onset and severity of manic episodes in BD and sleep paralysis in SP. The sudden/irregular inhibition of GABA/pGABA and glycine levels in the brain are proven to aggravate (or even jumpstart) symptoms of a Bipolar Manic episode as well as a simultaneous period of worsened sleep paralysis/abnormalities. Since comorbidity of sleep and mood disorders is significantly common, each abnormality fuels another, and as levels of neurotransmitters stray further from equilibrium, the cycle continues. To avoid prolonged psychological torture and the continued vicious cycle, both psychopharmacological and psychodynamic solutions have been implemented as preventative measures.

2. Glycine/GABA: Mechanisms of Action

Glycine and GABA are inhibitory neurotransmitters that regulate mood, hormonal balance, and sleep processes. GABA, in particular, acts as the primary inhibitory neurotransmitter in the central nervous system, counterbalancing excitatory neurotransmitters like glutamate. Its dysfunction has been shown to directly contribute to symptoms of Bipolar Disorder (BD) by disrupting mood regulation and triggering manic episodes. Similarly, glycine is essential in regulating motor function and sleep-wake cycles. Its dysregulation, particularly in REM sleep, has been implicated in Sleep Paralysis (SP) episodes by impairing muscle atonia mechanisms during sleep. Both neurotransmitters have been shown to play a critical role in neuroplasticity, with imbalances potentially exacerbating the pathophysiology of both BD and SP. Often found in the spinal cord/plasma within the CNS, these neurotransmitters slow down/prevent neurons from firing, causing motor cortex and nerve functioning to decelerate, resulting in the sensation of relaxation in both the brain and muscle connectivity.

Additionally, GABA/Glycine "play a major role in controlling nerve cell hyperactivity associated with anxiety, stress and fear" (GABA/Glycine often remain consistent, experiencing rapid reuptake throughout the body's natural circadian rhythms, only increasing to an extent during times of cyclic sleep/REM), [1]. Yet, when an environmental stressor occurs, the CNS produces less glycine, while the brain inhibits less GABA. Neurobiologically speaking, if irregularities surrounding the accumulation of said neurotransmitters exist and the general production levels become skewed, many adverse effects can occur, ranging from emotional to physical.

3. Bipolar Disorder: Neurobiological Underpinnings

Bipolar Disorder is a mood disorder categorized under "manic/hypomanic episodes when the predominant mood is intensely happy or irritable, or depressive episodes, when…the ability to experience joy or pleasure disappears" [2]. Within a triggered manic episode, a person with bipolar disorder may indulge in impulsive behaviors, often involving the dopamine receptors in the brain (substance abuse, excessive spending, gambling, impulsivity, etc.). Additionally, hypersensation, ex, heightened exuberance, is experienced, often to a severe degree (grandiose delusions). For someone with Bipolar Disorder, especially one who's unmedicated, a simple environmental factor could spark an episode. These environmental stressors may include substance use/abuse, stress, sleep deprivation/abnormalities, diet, etc. In Bipolar Disorder (BD), the dysfunction of multiple neurotransmitter systems—including dopamine, serotonin, GABA, and glycine—can significantly alter mood regulation. For example, dopamine dysregulation, particularly in the striatum and prefrontal cortex, is implicated in the reward processing and impulsive behavior seen in manic episodes. Meanwhile, GABA's role in inhibiting excitatory neurotransmission in the cortex and hippocampus is critical for maintaining mood stability. Similarly, serotonin plays a modulating role, and serotonin dysregulation can contribute to the emotional dysregulation and depression often seen in BD. This complex interaction of neurotransmitters in multiple regions of the brain is compounded by genetic factors, with several polymorphisms associated with GABAergic and serotonergic systems being linked to a higher risk of BD and related sleep disorders [2].

4. Manic Episode Neurobiology: The Role of GABA/Glycine

After an environmental stressor occurs in a person's life (trauma, excessive stress, serious personal event, etc.), the neurotransmitter signals and synapse binding in the brain and CNS become dysfunctional. A decrease in the transmission of GABA may result in "high-frequency firing of local neurons in the brain," resulting in mood-related and CNS dysfunction issues and a tendency for mania in the brain. In a study conducted on patients with Bipolar and other mood disorders, GABA and neurotransmission levels were measured with the existence of a manic episode versus in people with a generally stabilized mood. Results found that neu-

rotransmitters "via interneuronal synapses in brain regions controlling mood, such as the striatum, globus pallidus, and cerebral cortex...demonstrated decreased activity of glutamic acid decarboxylase, an enzyme involved in GABA synthesis [subsequently causing GABA irregularities]," [3].

5. Sleep Paralysis: Neurobiological Underpinnings

REM Parasomnia, or Sleep Paralysis, falls under the general trajectory of Sleep Disorders, occurring when a person gains cognitive awareness "as the body enters or exits REM sleep…Sleep paralysis can last from several seconds to several minutes…longer [episodes]...may even provoke a panic response" [3]. The general sensation can be categorized as the mind being fully cognizant while the motor cortex is still in a state of muscle atonia, resulting in immobility and a sense of panic. A person experiencing sleep paralysis may open their eyes suddenly, mid-SCN cycle, and find that while trying to move their limbs, they are incapacitated. In around 75% of people who experience frequent SP episodes, sleep-related hallucinations may occur (Intruder, Vestibular-Motor, or chest pressure are common subtypes). Within these hallucinogenic subtypes, a person may experience "perception of a dangerous person or presence (Intruder), out-of-body sensations (Vestibular-Motor), or [an incited] feeling of suffocation (Chest Pressure)" [4].

6. Sleep Paralysis Neurobiology: Inhibitory Mechanisms and GABA/Glycine Dysregulation

Similar to a manic episode, if a person is experiencing environmental stressors (severe sleep deprivation after stress or emotional strain, a traumatic event, etc.), neurotransmission and cellular polarization are deregulated. Studies taken on inhibitory mechanisms surrounding sleep "indicate that both metabotropic GABA and ionotropic GABA/glycine receptor-mediated inhibition of skeletal motor neurons underlies REM sleep atonia" [5]. Cellular hyperpolarization is precipitated when receptors express inhibition of said neurotransmitters to a dysregulated degree. In the specific process of sleep paralysis, the coexistence of GABA and glycine in the metabotropic and ionotropic inhibition subtypes are essential to the occurrence of REM motor paralysis, "masseter REM atonia only [remaining] intact when…GABA/glycine receptors are simultaneously antagonized on trigeminal motor-neurons," [5]. When this dysfunction occurs, episodic sleep paralysis prevails.

7. Coexistence and Cyclic Effects

The same Monoaminergic regions that produce insomnia/promote wakefulness (high output of forebrain acetylcholine). Since episodic mania and sleep paralysis involve similar environmental stressors, the correlative implications are significant. Whether the environmental trauma happens to be generalized emotional overwhelm/episode or lack of sleep based on stress/overwork, adverse effects in both fields are proven comorbidly. In a bipolar manic episode, there is a reduced

desire to sleep, a person feeling as though they need little/no sleep to be able to function at a "normal" level. In an experiment done on the onset, emotionally based primary insomnia, "Average brain GABA levels were nearly 30% lower in patients with PI (0.18 +/− 0.06) compared to controls (0.25 +/− 0.11)" [6]. With more chemical imbalance, a person's manic episodic behavior typically is exacerbated. With a severe lack of sleep, which tends to be a primary environmental stressor, sleep paralysis can occur, further driving down levels of GABA/glycine in inhibitory processes. Sleep paralysis-induced neurotransmitter dysregulation may affect one's cognition. If someone develops a fear of sleeping, their sleep may decrease. With low levels of GABA/glycine attributing to a lack of tranquility, stress buildup and emotional strain will prevail, subsequently causing mood issues and furthering chemical imbalance.

This vicious cycle may continue, one factor worsening another, until a person is at a place of extreme psychological distress, often referred to as psychological terror. This psychological terror may create/increase suicidal ideations and even attempts recorded in 25% - 60% of bipolar patients experiencing episodic distress and severe levels of suicidality for those with generalized sleep disorders, specifically insomnia [7].

Although this specific experiment involved the comorbidity of Depression and Insomnia, due to the similarities in chemical imbalances of all generalized mood disorders, as well as any general sleep disorders, the same conclusions can be made. When testing "insomnia [which is a marker for increased risk of suicidal thinking]… Higher levels of insomnia and depression, as recorded with the HAM-D, corresponded to a significantly greater intensity of suicidal thinking ($p < 0.01$, $p < 0.001$, respectively)." 0.0005×33 specifically targets heightened rates of parasomnia in adolescents with pre-existing mental ailments (EX., BPD, BD, depression, etc.) [8]. Pretty consistently after the 33xr rate, there seems to be a sharp incline in parasomnia behavior for patients within the trial.

In terms of prognosis, the majority of patients who experience Bipolar Disorder coupled with an unstable sleep cycle (specifically the existence of Sleep Paralysis) "…show a markedly increased risk of premature death due to the increased risk of suicide and medical comorbidities, including cardiovascular, respiratory, and endocrine causes…Additionally, attempted suicides are more common…" [9].

8. Neuroplasticity and Long-Term Effects of Chronic Neurotransmitter Dysregulation

Prolonged dysregulation of neurotransmitters such as GABA and glycine has significant implications for both the structure and function of the brain. Over time, the brain's ability to maintain homeostasis—particularly in regions that govern emotional regulation, sleep, and cognitive function—becomes compromised. The concept of neuroplasticity refers to the brain's ability to reorganize and adapt by forming new neural connections in response to environmental stimuli, learning, or injury. However, when the brain is subjected to chronic stress, neurotransmit-

ter imbalances, and repeated episodes of manic or depressive states, this neuroplastic capacity can be impaired.

9. Impact of Chronic Stress on Neuroplasticity

Chronic stress, which is frequently associated with conditions like Bipolar Disorder and Sleep Paralysis (especially considering cortisol levels), has been shown to inhibit neuroplasticity, particularly in brain regions such as the hippocampus and prefrontal cortex. The hippocampus, a key area involved in memory and emotional regulation, is especially vulnerable to the neurotoxic effects of prolonged cortisol exposure, a hormone released during stress. Cortisol has been shown to reduce the proliferation of new neurons in the hippocampus, potentially impairing emotional regulation and cognitive functioning over time. Chronic Stress can even significantly increase the possibility of disease contraction; "heightened or prolonged glucocorticoid levels such as during chronic stress exposure are known to be predictive of, and contribute to, the development of various disease states [10].

In individuals with Bipolar Disorder, frequent mood fluctuations and episodes of mania and depression may cause a surge of neurotransmitter imbalances that negatively affect the brain's plasticity. For example, during manic episodes, the dysregulated dopaminergic and GABAergic systems may alter synaptic plasticity, impairing the brain's ability to "reset" and recover. This can contribute to the chronic, relapsing nature of Bipolar Disorder, where patients may become increasingly resistant to treatment as their brain circuits become less responsive to conventional therapeutic interventions.

Similarly, individuals suffering from Sleep Paralysis often experience disrupted sleep cycles, particularly in the REM phase. Chronic disruption of sleep—especially during REM sleep, which plays a crucial role in memory consolidation, emotional processing, and cognitive functioning—can lead to lasting changes in brain circuits. Sleep deprivation has been linked to "reduced gray matter volume (GMV) and density in several brain regions [11], including the anterior cingulate cortex (ACC), medial-orbital frontal cortex, hippocampus and insula," [12]. Over time, these changes can contribute to cognitive impairments, heightened emotional reactivity, and a further deepening of sleep-related disturbances.

10. Neurotransmitter Imbalance and Long-Term Cognitive Dysfunction

Long-term imbalances in inhibitory neurotransmitters like GABA and glycine can also have lasting effects on cognition. Both of these neurotransmitters play a critical role in regulating the balance between excitation and inhibition in neural networks. When their levels are chronically low, as is often the case in both Bipolar Disorder and Sleep Paralysis, excessive neural excitation can lead to cognitive difficulties, including memory impairments, attention deficits, and difficulty processing complex tasks.

In a study done on rats, it was "reported that dysregulation of GABAergic activity in the prefrontal cortex of aged rats negatively affected their working memory performance…," and in a human crossover trial, "individuals with [dysregulated] GABA levels in the prefrontal cortex show a greater reduction in working memory performance from lower loads than participants with [more stabilized] GABA levels," based on fMRI scan comparisons [13].

In the context of Bipolar Disorder, prolonged periods of mania can lead to an overactivation of certain brain regions, particularly those involved in reward processing (such as the striatum), which may disrupt cognitive function. The dysregulated activity of GABA and glycine receptors in these regions may impair normal executive functioning, making it more difficult for individuals to engage in logical decision-making or emotional regulation. In recent "electrophysiological and neuroimaging studies… (hypo)manic symptoms are associated with heightened reward-related activation in brain regions with high dopamine receptor density…evidence of abnormally elevated activity within the VS during reward anticipation, reward consumption, and to reward-predictive cues has been found in BP [especially within heightened manic periods] [13]. This can have long-term consequences on personal relationships, job performance, and overall quality of life.

In the case of Sleep Paralysis, recurrent episodes of sleep deprivation, particularly during REM sleep, can lead to the accumulation of cognitive deficits. Sleep plays an essential role in memory consolidation and the processing of emotions. When this process is repeatedly disrupted, individuals may experience difficulties in retaining information, regulating emotional responses, and coping with stress. With lack of sleep, it's proven that "an increased amygdala hyper limbic reaction occurs, resulting in stimuli with negative emotional connotations. This varying level of amygdala activity is linked to a loss of mPFC functional connectivity when sleep deprived, suggesting a decrease in prefrontal lobe inhibition signals," [14]. As a result, the brain may become "stuck" in a state of heightened alertness, making it more prone to anxiety, panic, and further disturbances in sleep. In a statistical analysis study conducted on a large group of sleep-deprived individuals' recorded moods, it was found that "sleep loss increased levels of negative mood states, $SE = 0.076$, $t(157) = 6.10$, $p < 0.001$, CI [0.30, 0.59]…[which can] help explain associations between sleep loss and the development of psychopathology, particularly depression… [episodically is a component of BD]" [15].

11. Plausible Solutions

With mood disorders generally involving a chemical imbalance in the brain, a multipath treatment approach is often recommended.

In terms of psychopharmacology, medication targeted for mood stabilization and anti-psychosis work is recommended. Typically, doctors prescribe benzodiazepines (eq., Alprazolam, Diazepam), a type of medication that works by "enhancing…GABA at the GABA-A receptor. This results in the sedative, hypnotic (sleep-inducing), anxiolytic (anti-anxiety), anticonvulsant, and muscle relaxant

properties for which the drugs are prescribed" [16]. Although this medication treatment targets mainly sleep properties, the increased need for GABA/glycine transmission in the emotional sense is immense. With this stability, the relaxant/sedative properties, in the short term, can be increasingly helpful for stabilizing a manic episode. In the long term, mood stabilizers (Lithium, Anticonvulsants, or antipsychotics) are used to keep moods stable and consistent. Mood stabilizing medication works to temper different synaptic connection irregularities within the brain, to balance both inhibitory and excitatory neurotransmitters. A popular example would be Valproic Acid, a mood stabilizer that targets Bipolar Disorder and emotional imbalance. VPA "exerts its anticonvulsant effects by increasing the availability of GABA in GABAergic synapses…expected to inhibit excessive firing of synapses" [12].

Similarly, antidepressants/SSRIs (ex. Olanzapine, fluoxetine) are recommended. This medication classification helps to enhance GABAergic and serotonergic signaling, which may play a role in rebalancing the inhibitory-excitatory processes in the brain, potentially restoring cognitive function and emotional regulation [8]. Over time, as these treatments promote neuroplasticity, individuals may experience fewer manic episodes, reduced sleep disturbances, and improved cognitive function, leading to a better overall prognosis.

With a stabilized mood, a person may also experience the average human desire for sleep, and with this extinction of insomnia, sleep paralysis episodes may significantly decrease/cease to exist. With the existence of chemical medication, a person's brain may be rewired and centered to help prevent suicidal ideations and increase stability.

In the psychodynamic lens, implementing psychological interventions cannot be denied. A clinical psychologist or psychotherapist utilizing psychodynamic or IP (Interpersonal) therapy can be incredibly effective on people experiencing sleep/mood disorders. Although psychotherapy may not be enough to mediate the effects of cognitive chemical imbalance, the ability of a patient to get to the root of the societal/environment "trigger" for episodic behavior can be extremely helpful in providing solace, adjunctive to medication-based treatment. Additionally, the "core objective of psychoeducation is to foster a clear rationale for individuals with bipolar disorder to seek, adhere to, and remain in treatment" [7]. Additionally, extenuating factors (cultural predispositions, socioeconomic status, familial strains, etc.) may limit the ability to access chemical treatment, and a therapeutic approach can be a helpful/available solution.

Though it is not currently fully neurologically backed, evidence is starting to show positive results on GABA/glycine levels when a person finds peace in psychodynamic therapy. If these psychotherapists utilize strategies to help rewire thought processes and look towards a more positive angle, a person may start to use these newfound ideals, which could contribute to increased levels of positive neurotransmission (to a more acute degree than actual chemical intervention, yet still partially effective) [8].

12. Conclusions

The intricate relationship between GABA/glycine inhibition and cognitive stability highlights the profound neurobiological underpinnings of mood and sleep disorders. The dysregulation of these inhibitory neurotransmitters, triggered by environmental stressors, plays a pivotal role in exacerbating the symptoms of both Bipolar Disorder and sleep abnormalities, including sleep paralysis. These disorders are deeply interconnected, with one fueling the other in a vicious cycle that can lead to severe emotional, cognitive, and physical distress. The interaction between GABA and glycine in these processes underscores a shared mechanism of dysfunction—whether it's the manic episode in Bipolar Disorder or the immobilizing experience of sleep paralysis—each condition intensifies the other, often resulting in a state of overwhelming psychological instability.

Despite the seemingly inescapable nature of this cycle, there is hope. Advances in psychopharmacology, particularly in medications that target GABAergic and glycinergic systems, have proven effective in restoring balance to the brain's neurotransmission pathways. Medications such as benzodiazepines, mood stabilizers like lithium, and anticonvulsants like valproic acid can provide both short-term relief and long-term stabilization by enhancing GABA's calming effects. This not only helps mitigate the symptoms of mania and sleep disturbances but also offers crucial protection against the risks of suicidal ideation and related co-morbidities. Additionally, psychotherapeutic interventions, especially those focused on addressing environmental stressors and reprogramming maladaptive thought patterns, can complement pharmacological treatment, offering individuals a more holistic approach to managing their conditions. Furthermore, a multipath approach may help create a more stable emotional environment, further supporting neuroplasticity and long-term brain health.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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