

What Is the Generalized Anxiety Disorder?

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Abstract: Generalized Anxiety Disorder (GAD) is characterized by persistent, excessive worry about everyday situations, lasting for at least six months. Individuals with GAD often experience both psychological and physical symptoms, including restlessness, muscle tension, and sleep disturbances. Freud's early idea of "anxious expectation" is still a key part of understanding the disorder, along with the more modern concepts of "free-floating anxiety" and panic attacks. Research into the brain shows that Generalized Anxiety Disorder (GAD) is tied to issues with certain neurotransmitter systems, like serotonin, norepinephrine, and GABA. These imbalances play a big role in mood changes and anxiety symptoms. To create better treatments, it's important to dig deeper into how genetic, environmental, and neurochemical factors all work together to cause GAD.

Keywords: Generalized Anxiety Disorder, abnormalities in neurotransmitter systems

1. Introduction

Generalized Anxiety Disorder (GAD) is a common yet challenging mental health condition that causes excessive, uncontrollable worry about everyday situations. Unlike short-term situational anxiety, which is tied to specific stressors, GAD is a long-lasting issue that affects many areas of life, from work and relationships to physical health. People with GAD often experience physical symptoms like restlessness, muscle tension, and trouble sleeping, which can make daily life even harder and significantly lower their quality of life.

The origins of Generalized Anxiety Disorder (GAD) can be linked to early psychoanalytic theories, particularly Freud's idea of "anxious expectation," which describes a state of ongoing apprehension without an identifiable cause. In modern times, GAD is understood through both psychological and neurobiological perspectives. Studies indicate that imbalances in key neurotransmitters—such as serotonin, norepinephrine, and GABA—are central to the development and persistence of anxiety symptoms. These chemical imbalances disrupt the brain's ability to regulate mood, process emotions, and manage cognitive functions, ultimately contributing to the chronic and pervasive anxiety that defines GAD.

This paper will examine the key features of GAD, its symptoms, and the neurobiological factors that contribute to its development. Understanding the intricate relationship between these factors is crucial for improving treatment strategies and enhancing the quality of life for those affected by the disorder.

2. Generalized Anxiety Disorder

2.1. Defining the Generalized Anxiety Disorder

Generalized anxiety disorder is a condition of excessive worry about everyday issues and situations. Worry occurs more often than not for at least 6 months and is excessive. People with GAD often experience persistent anxiety and tension, even when there is no apparent reason for concern. The individual finds it difficult to control the worry [2]. (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)) Anxious expectation represented the “nuclear” symptom of anxiety neurosis and involved nervousness, apprehension, and free-floating anxiety [1].(Freud S 1957) These anxious thoughts are frequent, sudden, sudden entry into consciousness, and may be related to thoughts of death. For example, (1) a woman can not answer the phone of her husband outside, she will think that her husband is dead. (2) When a woman goes to someone's house and sees the neighbor's dog, she automatically thinks that something has happened to her own dog. Anxiety neurosis, according to Freud, consists of four major clinical syndromes: general irritability, chronic apprehension/anxious expectation, anxiety attacks, and secondary phobic avoidance[1].(Freud S 1957) Anxiety neurosis frequently occurs in conjunction with other neuroses, which called “mixed neurosis.” Anxiety symptoms frequently occur in combination with those of neurasthenia, hysteria, and obsessions [3]. The concept of “neurosis” as a major organizing principle in structuring the anxiety disorders[4][5].The dominant American psychiatric theory is that all psychopathology is caused by psychological conflict, namely anxiety. Psychosis is thought to be the result of excessive anxiety leading to self-breakdown and degeneration, while neurosis is the result of partially successful defense against anxiety leading to symptom formation[8].

2.2. Symptoms of Generalized Anxiety Disorder

Generalized anxiety disorder (GAD) is characterized by excessive worry that is difficult to control accompanied by physical symptoms including restlessness, being easily fatigued, difficulty concentrating, irritability, muscle tension, or sleep disturbance[8]. Freud described anxiety symptoms as primarily somatic symptoms that may occur either in a “free-floating anxiety” or “apprehension” state or in a “sudden anxiety attack[1].” The symptoms most likely to occur in both anxiety states and anxiety attacks as trepidation, arrhythmia, dyspnea, sweating, nausea, heavy feeling in the stomach, tremor, increased urination, increased appetite, diarrhea, vertigo-giddiness, paresthesias, pavor nocturnus, increased sensitivity to pain, a decrease of sexual interest, low self-esteem, and marked affective mobility[7]. In individuals experiencing the "free-floating anxiety" or "apprehension" state of generalized anxiety disorder (GAD), several symptoms like Persistent, uncontrollable, and excessive worries about all aspects of life, such as work, health, family, or finances. Often feel tense or uneasy or have difficulty relaxing. Persistent feeling of fatigue or tiredness, even without significant exercise. Unable to concentrate because of worry. Physical symptoms, such as muscle tension or soreness, often have no clear physical cause. Increased irritability or feeling easily agitated. Trouble falling asleep, staying asleep, or feeling rested after sleep is a common struggle for individuals with generalized anxiety disorder (GAD). Physical symptoms like trembling, sweating, nausea, or stomach discomfort are also frequent and can significantly impact daily life and overall well-being. Another key aspect of GAD is the occurrence of sudden anxiety episodes, often referred to as panic attacks[7]. These attacks involve a sudden wave of intense fear or discomfort that typically reaches its peak within minutes. During a panic attack, people might experience symptoms such as a racing or pounding heartbeat, excessive sweating, shaking, difficulty breathing or feeling smothered, a choking sensation, chest pain or tightness, nausea or stomach upset, dizziness, lightheadedness, chills or hot flashes, tingling or numbness, and a sense of detachment or unreality. Many also feel an

overwhelming fear of losing control, going crazy, or even dying. It's important to recognize that panic attacks can happen without warning, even in situations that aren't threatening. These episodes can be incredibly overwhelming and may cause individuals to avoid certain places or situations out of fear of triggering another attack[8].

2.3. What happens in the process when someone develops Generalized Anxiety Disorder

Research suggests that Generalized Anxiety Disorder (GAD) is linked to irregularities in neurotransmitter systems, particularly serotonin, norepinephrine, and gamma-aminobutyric acid (GABA)[9]. Serotonin, a key neurotransmitter, plays a major role in regulating mood, emotions, and anxiety[10]. It influences various brain functions like emotional processing, cognition, and sleep. Studies have shown that people with GAD often have reduced serotonin activity or availability in certain areas of the brain, such as the amygdala and prefrontal cortex[11]. This serotonin imbalance may occur due to problems with how serotonin is produced, released, or reabsorbed in the brain. For instance, lower activity of tryptophan hydroxylase (TPH), the enzyme responsible for producing serotonin, could lead to decreased serotonin levels.

In individuals with GAD, reduced TPH activity could result from various factors, including genetic predisposition, environmental stress, or other neurobiological issues. Additionally, serotonin transporter proteins, which are responsible for reabsorbing serotonin from the synaptic cleft, might not function properly. This can prolong serotonin's action and further disrupt its balance. Another contributing factor is changes in serotonin receptors, such as the 5-HT1A receptor subtype. Alterations in how these receptors function or their sensitivity and density can impact the brain's ability to regulate anxiety.

Serotonin imbalances in GAD may also stem from disruptions in how serotonin interacts with other neurotransmitter systems, like norepinephrine and GABA. When these interactions are dysregulated, the balance between excitatory and inhibitory signals in the brain can be thrown off, which can ultimately lead to heightened anxiety symptoms.

3. Conclusion

Generalized Anxiety Disorder (GAD) is a challenging and complex condition defined by excessive worry and physical symptoms that interfere with daily life. It involves persistent and uncontrollable anxiety that goes beyond specific stressors and lasts for at least six months. While Freud's early idea of "anxious expectation" still helps explain the core features of GAD, modern perspectives have expanded on this by incorporating concepts like "free-floating anxiety" and panic attacks. Today, both psychological and neurobiological factors are recognized as key contributors to the development of GAD, building on Freud's foundational ideas.

On a neurobiological level, GAD is linked to imbalances in major neurotransmitter systems, particularly serotonin, norepinephrine, and gamma-aminobutyric acid (GABA). Serotonin dysfunction, in particular, plays a major role by disrupting mood regulation and emotional processing. These disruptions, combined with irregularities in other neurotransmitter systems, can disturb the balance between excitatory and inhibitory signals in the brain, further fueling anxiety symptoms. Although more research is needed to fully unravel the biological mechanisms behind GAD, current findings suggest that a combination of genetic predispositions, environmental factors, and neurochemical imbalances all play a part.

GAD is a highly complex disorder that impacts emotional well-being and physical health, emphasizing the importance of treatment approaches that address both psychological and biological aspects. By better understanding the underlying mechanisms of GAD, future interventions can more

effectively target neurotransmitter imbalances and help reduce the overwhelming symptoms that define this disorder.

References

- [1] Freud S. *Collected Papers, Vol.1*. London, England: Hogarth Press; 1956:76-106
- [2] American Psychiatric Association. *Diagnostic and statistical manual of mental disorders (DSM-5)*. 5th ed. Arlington, VA. 2013 pages 222-225
- [3] Karl Rickels, M.D., and Moira A. Rynn, M.D., *J Clin Psychiatry* 2001;62 (suppl 11); Page 5
- [4] American Psychiatric Association Mental Health Service. *Diagnostic and Statistical Manual of Mental Disorders*. Washington, DC: American Psychiatric Association Mental Health Service; 1952
- [5] American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, Second Edition*. Washington, DC: American Psychiatric Association; 1968
- [6] Klein DF. Anxiety reconceptualized. In: Klein DF, Rabkin J, eds. *Anxiety: New Research and Changing Concepts*. New York, NY: Raven Press; 1981:235, 243–245
- [7] Freud F, "Inhibitions, Symptoms and Anxiety" (1926)
- [8] American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. Arlington, VA: American Psychiatric Publishing. Pages 222-225.
- [9] American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. Arlington, VA: American Psychiatric Publishing. P208-210.
- [10] Bandelow, B., & Michaelis, S. (2015). Epidemiology of anxiety disorders in the 21st century. *Dialogues in Clinical Neuroscience*, 17(3), 327–335.
- [11] Nutt, D. J. (2002). The role of dopamine and norepinephrine in depression and antidepressant treatment. *The Journal of Clinical Psychiatry*, 63(Suppl 6), 12–19.
- [12] Baldwin, D., Rudge, S., & Persson, R. (2002). Quetiapine is no better than placebo in generalized anxiety disorder: A randomized controlled trial. *International Clinical Psychopharmacology*, 17(2), 77–82.
- [13]