

### Neuroscience and Emotion: How are Internal States Generated and Shaped? Ina Malkouari

## Abstract

Anxiety is a familiar feeling of stress and despair in the face of fear. Typically, anxiety affects a great portion of the population. Considering this, it becomes critical to understand the reasoning behind such immense fear. The amygdala within the brain contributes a majority of the signals that will trigger feelings of anxiety. Other structures, such as the prefrontal cortex, will also contribute to the neurological anatomy of this disorder. Inside these structures, neurons fire signals to the body to protect against danger using specific neurotransmitters (serotonin, norepinephrine, acetylcholine, etc.). However, anxiety can be sporadic, needing treatment from some of the more severe disorders as medicine continues to evolve and new therapies for these disorders, outside of pharmacology, are implemented.

### Introduction

Anxiety, the constant feeling of immense worrying and or fear due to strenuous environment or personal circumstances, has become a prevalent issue among individuals over the past couple of years. It has become one of the most common mental illnesses in the United States, affecting about 19.1% of the population ages 18 and above every single year (ADAA, 2022). For most anxiety disorders, the feeling of anxiety is persistent and can worsen over time, leading to increased difficulty in daily tasks involving jobs, social interactions, and personal relationships (NIMH, n.d.). Therefore, the importance of discovering the correlation between various neurological instances and environmental circumstances becomes important with the association of anxiety as a unit.

#### 6 Basic Emotions & Anxiety

A pivotal theory for understanding how anxiety is showcased involves the six basic emotions proposed by Paul Ekman. After discovering that many investigators also cultivated evidence about the six basic emotions, Paul Ekman disclosed the "Big 6 Basic Emotions" as disgust, fear, sadness, surprise, happiness, and anger. Fifteen years following this, after reviewing subsequent information, the emotions were considered universal due to overwhelming evidence (Ekman, 1992). Considering these universal emotions, anxiety can be best coupled with the emotion of fear. Anxiety itself has been depicted to involve an immense amount of fear and feelings of hopelessness at times (NIMH, n.d.). Fear, as commonly experienced with anxiety disorders, is typically described as being scared or afraid of an object or situation and can be heightened in the presence of imminent danger or possible harm to the subject being affected. Although this emotion is distinctively described as a "negative" emotion, it is instead the reason for survival as it allows for the mental processing of potential danger (Ekman, n.d.). Hand in hand with the feeling of danger is our fight-or-flight tendencies. It is often coupled with anger and fear, as commonly found in anxiety disorders across the board. Sigmund Freud referred to this tendency as a way to frequently respond to fear or experience generalized anxiety as neuroticism (Dong et al., 2022).

#### Anatomy

One of the more critical anatomical parts in the brain relating to anxiety is the amygdala, located in the temporal lobe. The amygdala is a paired structure, meaning there is one for each



side of the brain —although it is not commonly referred to as multiple structures— and contains glial cells and neurons to relay chemical and electrical signals. This structure is responsible for expressing fear and aggression, having a role necessary for retrieving emotional and other fear-related memories of specific stimuli. The amygdala's central nucleus (CeA) is interconnected with cortical regions, including the limbic cortex. (Martin et al., 2009). These connections allow for supplementary information to be collected from the hippocampus, thalamus, and hypothalamus.

Other sectors of the brain that contribute to anxiety are the medial prefrontal cortex (mPFC) and posterior cingulate cortex (PCC). Both of these structures will receive critical information from the insula, which will process visceral information; the amygdala, which is responsible for coding the relevance of a stimulus; and the medial temporal lobe, which is involved with our memory (Saarimäki et al., 2016). All these structures, alongside the thalamus and hypothalamus, comprise the medial side of the default mode network (DMN), which is responsible for self-referencing (Saarimäki et al., 2016). The image below depicts the anatomical structures in the brain for better visual representation of the sectors of the brain mentioned previously.



### **Neurotransmitters and Receptors**

Neurotransmitters are chemical messengers responsible for sending signals to the rest of the body from neurons to target cells in response to varying stimuli (Cleveland Clinic, 2021). One of the most common neurotransmitters found in the instance of anxiety is Gamma-aminobutyric acid (GABA). GABA is an inhibitory neurotransmitter typically found in the central nervous system (CNS) and balances neuronal excitation and inhibition. Aside from balancing neuronal activity, GABA is the center for regulating anxiety —benzodiazepines and other anxiety-reducing drugs target this particular neurotransmitters such as serotonin, opioid peptides, endocannabinoids, and neuropeptide Y are also involved with this sensation (Nuss, 2015).

Focusing on GABA, receptors of this neurotransmitter substantially raise chloride conductance in cell membranes. Therefore, causing neurons not to be able to reach their action potential —rapid changes in voltage across the membrane—meaning a stage of phasic inhibition of the neuron. Moreover, low concentrations of GABA in extracellular spaces can constantly "turn on" extrasynaptic GABAA receptors to be stuck in a "tonic" inhibitory state (Nuss, 2015). This factor often makes the neuron no longer sensitive to excitatory stimuli.

Relating to anxiety, extensive activity found in the emotion-processing sections of the brain (amygdala, insula, or insular cortex) in anxiety disorder patients could be a result of lower inhibitory signaling from GABA or even excitatory neurotransmission by the neurotransmitter glutamate (Martin et al., 2009).

# **Anxiety Disorders and Treatment**

Immense fear and feelings of potential helplessness characterize anxiety disorders. Disorders in the category of anxiety include Obsessive Compulsive Disorder (OCD), Generalized Anxiety Disorder (GAD), and Post-Traumatic Stress Disorder (PTSD). All of which involve some form of extreme worrying or consistent feeling of being trapped and in a constant cycle of not being able to relax (National Institute of Mental Health). When looking at the big picture, GAD affects just about 6.8 million adults in the United States (AADA, 2022), thus creating a need for understanding the disorder and others just like it.

Nurture versus nature sparks a great ordeal concerning mental health disorders, especially anxiety; nature (genetics) can be seen as a possible predisposition to the disorder based on heredity. Genes such as 5-HTTLPR, which is a serotonin transporter gene, have been linked to an increased risk of developing an anxiety disorder due to its fluctuating serotonin levels in the brain (Beevers et al., 2009). The Brain-derived Neurotrophic Factor (BDNF), essential for brain plasticity and resilience, has variants linked to increased anxiety vulnerability.

Nurture, however, focuses on environmental factors that may contribute to particular circumstances. For anxiety disorders, this can be placed in many instances. One of the greater circumstances begins in childhood and can span to adulthood. Parenting style, for one, is most prominent in childhood and adolescence; overly protective and restrictive or even abusive parenting has been shown to increase anxiety in children significantly. This also includes trim to no autonomy over one's life growing up, thus inducing anxiety (Budinger et al., 2013). Other instances, including physical and emotional abuse, can increase the chances of developing Post-Traumatic Stress Disorder (PTSD) based on the circumstances.

Regardless of nature or nurture, anxiety has become very manageable with a variety of treatment options. From a therapy standpoint, Cognitive Behavioral Therapy (CBT) is a psychotherapy that has been proven helpful for those with anxiety disorders. This type of treatment focuses on different ways of thinking and reacting to help patients feel less fearful or anxious in triggering situations (NIMH Anxiety Disorders, n.d.). A branch of CBT is called Exposure Therapy; this type of therapy focuses on facing the fears that trigger one's anxiety and helps the patient engage with the fears in a safe environment, thus lowering the anxiety surrounding the situation (NIMH Anxiety Disorders, n.d.). However, from a pharmacological approach, beta-blockers—usually used to treat high blood pressure— have been shown to lessen the physical symptoms of extreme anxiety, including shaking, fast heartbeat, and blushing. These are found to have the best results when used for short periods. Therefore, making it a short-term solution in comparison to the therapies (NIMH Anxiety Disorders, n.d.).

## Conclusion

The development of anxiety and its associated disorders are webbed among a variety of circumstances that involve the structures of the brain, genetic components, as well as environmental factors. In the brain, the amygdala is the center of fear and anxiety-related emotions, which is ultimately coupled with the neurotransmitters found in the region. GABA is the main culprit for the signals of helplessness and extreme worry being signaled and

transported throughout the body. Also, considering the influence of one's environment is crucial for understanding anxiety as a part of fear (Big 6 Emotions) and mental disorders that affect millions of people yearly.

## References

- [1] (2022, March 14). *Neurotransmitters*. Cleveland Clinic. <u>https://my.clevelandclinic.org/health/articles/22513-neurotransmitters</u>
- [2] (2023, April 11). *Amygdala*. Cleveland Clinic. <u>https://my.clevelandclinic.org/health/body/24894-amygdala</u>
- [3] (2022, October 28). Anxiety Disorders Facts & Statistics. Anxiety & Depression Association of America. <u>https://adaa.org/understanding-anxiety/facts-statistics</u>
- [4] (n.d.). Any Anxiety Disorder. National Institute of Mental Health. <u>https://www.nimh.nih.gov/health/statistics/any-anxiety-disorder</u>
- [5] Beevers, C. G., Wells, T. T., Ellis, A. J., & McGeary, J. E. (2009). Association of the serotonin transporter gene promoter region (5-HTTLPR) polymorphism with biased attention for emotional stimuli. Journal of abnormal psychology, 118(3), 670–681. <u>https://doi.org/</u> 10.1037/a0016198
- [6] Crosby Budinger, M., Drazdowski, T. K., & Ginsburg, G. S. (2013). Anxiety-promoting parenting behaviors: a comparison of anxious parents with and without social anxiety disorder. Child psychiatry and human development, 44(3), 412–418. <u>https://doi.org/10.1007/s10578-012-0335-9</u>
- [7] Dong, J., Xiao, T., Xu, Q., Liang, F., Gu, S., Wang, F., & Huang, J. H. (2022). Anxious personality traits: Perspectives from basic emotions and neurotransmitters. *Brain sciences*, *12*(9), 1141.
- [8] Ekman, P. (1992). Are there basic emotions? Retrieved from https://www.paulekman.com/wp-content/uploads/2013/07/Are-There-Basic-Emotions1.pdf
- [9] Ekman, P. (n.d.). What is fear? Paul Ekman Group. Retrieved from https://www.paulekman.com/universal-emotions/what-is-fear/
- [10] Martin, E. I., Ressler, K. J., Binder, E., & Nemeroff, C. B. (2009). The neurobiology of anxiety disorders: brain imaging, genetics, and psychoneuroendocrinology. *The Psychiatric Clinics of North America*, 32(3), 549–575. <u>https://doi.org/10.1016/j.psc.2009.05.004</u>
- [11] National Institute of Mental Health. (n.d.). Anxiety disorders. National Institute of Mental Health. Retrieved from <u>https://www.nimh.nih.gov/health/topics/anxiety-disorders</u>



- [12] Ninan, P. T. (1999). The functional anatomy, neurochemistry, and pharmacology of anxiety. *Journal of Clinical Psychiatry*, pp. *60*, 12–17.
- [13] Nuss, P. (2015). Anxiety disorders and GABA neurotransmission: A disturbance of modulation. Neuropsychiatric Disease and Treatment, pp. 11, 165–175. doi: 10.2147/NDT.S58841. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4303399/
- [14] Saarimäki, H., Gotsopoulos, A., Jääskeläinen, I. P., Lampinen, J., Vuilleumier, P., Hari, R., ... & Nummenmaa, L. (2016). Discrete neural signatures of basic emotions. *Cerebral cortex*, 26(6), 2563-2573.