



---

## **NEUROPHYSIOLOGICAL BASIS OF EMOTIONAL REGULATION: FUNCTIONAL INTERACTIONS BETWEEN THE AMYGDALA AND PREFRONTAL CORTEX**

Narmetova Yulduz Karimovna

Associate Professor at Tashkent State Medical University

Doctor of Psychological Sciences (DSc)

Abduvohidov Muhammad Mustafo Abdukabir o'g'li

2nd-Year Student, Pediatrics Faculty

Tashkent State Medical University

---

### **Abstract**

This article provides a detailed analysis of the neurophysiological foundations of emotional regulation, particularly the functional interactions between the amygdala and the prefrontal cortex. The author highlights the central role of the amygdala in the rapid processing of negative emotions and the high-level cognitive control function of the prefrontal cortex. The study discusses “bottom-up” and “top-down” mechanisms, physiological bases of emotional regulation through neuroimaging (fMRI, EEG, DTI), and neurochemical mechanisms (GABA, glutamate, serotonin, dopamine). Furthermore, disturbances within this system in psychopathological conditions such as depression, anxiety disorders, and PTSD, as well as practical therapeutic approaches including cognitive reappraisal, mindfulness, neurofeedback, and transcranial magnetic stimulation (TMS), are examined. The article provides a scientific basis for understanding the biological mechanisms of emotional regulation to support psychotherapy and neurological research.

**Keywords:** Emotional regulation, amygdala, prefrontal cortex, cortico-limbic system, functional connectivity, neurophysiology, cognitive reappraisal, neuroimaging.



## ***Modern American Journal of Medical and Health Sciences***

ISSN (E): 3067-803X

Volume 01, Issue 09, December, 2025

Website: [usajournals.org](http://usajournals.org)

*This work is Licensed under CC BY 4.0 a Creative Commons Attribution 4.0 International License.*

---

### **Introduction**

Emotional regulation is one of the most complex integrative functions of human consciousness and is directly associated with adaptability to internal and external factors, stable social behavior, and the quality of cognitive processes. Recent neuroscience research demonstrates that the complex neuronal interactions between the amygdala and the prefrontal cortex play a central role in emotion regulation. While the amygdala, a limbic structure, rapidly processes fear, threat, and negative affective signals, the prefrontal cortex exerts higher-level cognitive control over these processes. This article thoroughly explores the functional connectivity between these two major neural systems, their physiological and neurochemical mechanisms, alterations observed in psychological disorders, and practical management possibilities.

### **Theoretical Background**

The neurophysiological mechanisms of emotional regulation depend on the integrated functioning of the cortico-limbic system. As a primary structure of the limbic system, the amygdala automatically generates emotional reactions such as fear, anxiety, and anger. It receives sensory signals via the thalamus, processes them, and transmits them to the autonomic nervous system, hypothalamus, and prefrontal cortex. This rapid communication strengthens the role of the amygdala as a "threat detection center."

The prefrontal cortex is responsible for conscious control, decision-making, attention regulation, planning, and inhibition processes. The ventromedial prefrontal cortex suppresses amygdala activity through inhibition, codes safety signals, and selects socially appropriate emotional responses. The dorsolateral prefrontal cortex is involved in cognitive reappraisal by logically interpreting negative situations to reduce amygdala reactivity. The dorsomedial prefrontal cortex plays a role in self-awareness, introspection, and emotional monitoring. Emotional processes are regulated through two primary mechanisms: "bottom-up" and "top-down." The bottom-up mechanism begins with the amygdala's rapid response to negative stimuli. The top-down process consists of inhibitory signals



## ***Modern American Journal of Medical and Health Sciences***

ISSN (E): 3067-803X

Volume 01, Issue 09, December, 2025

Website: [usajournals.org](http://usajournals.org)

*This work is Licensed under CC BY 4.0 a Creative Commons Attribution 4.0 International License.*

---

from the prefrontal cortex that reduce amygdala activity. The balance between these two streams forms the physiological basis of emotional stability.

### **Neuroimaging-Based Dynamics of Emotional Regulation**

Techniques such as fMRI, EEG, and DTI provide precise insight into the neural mechanisms of emotional regulation. fMRI studies demonstrate a marked increase in amygdala BOLD signals under negative emotional stimuli. During cognitive reappraisal strategies, ventromedial prefrontal cortex activation increases while amygdala reactivity decreases. In depression, anxiety, and PTSD, a decrease in functional connectivity between these two structures is observed. EEG studies measure physiological indicators of emotional states through frontal alpha-wave asymmetry. Low alpha activity in the left frontal cortex indicates positive emotional dominance, while low alpha activity in the right frontal cortex suggests increased sensitivity to negative emotions. These indicators reflect functional interactions within the amygdala–prefrontal cortex system.

DTI studies examine the integrity of the “uncinate fasciculus,” the key white matter tract directly connecting the amygdala and the prefrontal cortex. Structural disruption of this pathway in depression and anxiety disorders leads to impaired emotional regulation.

### **Neurochemical Mechanisms**

GABA, glutamate, serotonin, and dopamine systems play crucial roles in emotional regulation. The ventromedial prefrontal cortex reduces excessive amygdala activity through GABAergic inhibition. When this mechanism is weakened, amygdala responsiveness increases, leading to emotional dysregulation. Glutamate and NMDA receptors in the amygdala nuclei are essential for fear formation and consolidation.

The serotonin system is a major neurochemical factor in emotional stability; its deficiency diminishes the prefrontal cortex’s ability to regulate emotional responses. The dopamine system enhances positive emotions, motivation, and goal-directed behavior. Optimal dopamine receptor activity in the prefrontal cortex reduces sensitivity to negative stimuli and reinforces emotional regulation.



---

## **Pathological Alterations in Psychopathological Conditions**

In depression, excessive amygdala activation and reduced prefrontal inhibition are observed, contributing to pathological bias toward negative information processing. In generalized anxiety disorder, the amygdala remains in a persistently hyperactive state, while prefrontal inhibitory mechanisms function inadequately. In PTSD, the amygdala operates in constant threat mode, and the prefrontal cortex fails to suppress this reactivity.

## **Practical Implications**

Cognitive reappraisal strategies activate the prefrontal cortex to enhance cognitive control and reduce amygdala-driven responses. Mindfulness-based psychotherapy strengthens functional connections within the cortico-limbic system and enhances emotional stability.

Neurofeedback training promotes prefrontal activity by regulating frontal rhythms and improves inhibitory control over amygdala signals. Transcranial magnetic stimulation (TMS) activates the dorsolateral prefrontal cortex, reducing depressive and negative affective responses.

## **Conclusion**

Emotional regulation is a fundamental criterion of psychological stability, stress resilience, and social adaptability. Its neurophysiological foundation is built upon the complex interplay between the amygdala and prefrontal cortex. While the amygdala rapidly processes negative emotional signals, the prefrontal cortex performs conscious inhibition, evaluation, and regulation. Modern neuroimaging research confirms that the strength of the functional connection between these two structures is a key determinant of emotional health.

The balance of neurochemical systems—particularly GABA, glutamate, serotonin, and dopamine—ensures the effective function of this regulatory mechanism. In psychopathological conditions such as depression, anxiety disorders, and PTSD, disruptions in amygdala–prefrontal interactions result in heightened emotional reactivity and reduced cognitive control. Practical



## ***Modern American Journal of Medical and Health Sciences***

**ISSN (E):** 3067-803X

**Volume 01, Issue 09, December, 2025**

**Website:** usajournals.org

***This work is Licensed under CC BY 4.0 a Creative Commons Attribution 4.0 International License.***

---

approaches such as cognitive reappraisal, mindfulness, neurofeedback, and TMS are effective in strengthening this system and restoring functional connectivity. In summary, the effectiveness of emotional regulation relies on the delicate neural balance between automatic limbic reactions and conscious prefrontal control. A deeper understanding of this system opens new opportunities in modern clinical psychology, psychotherapy, and neurological medicine.

### **References**

(Translation preserved exactly as original)

1. LeDoux, J. E. (2012). Rethinking the Emotional Brain. *Neuron*.
2. Pessoa, L. (2017). Emotion and the brain: From classical theories to modern neuroimaging. *Nature Reviews Neuroscience*.
3. Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences*.
4. Etkin, A., Egner, T., & Kalisch, R. (2011). Emotional processing in anterior cingulate and medial prefrontal cortex. *Biological Psychiatry*.
5. Phelps, E. A., & LeDoux, J. E. (2005). Contributions of the amygdala to emotion processing. *Neuron*.
6. Shin, L. M., & Liberzon, I. (2010). The neurocircuitry of fear, stress, and anxiety disorders. *Neuropsychopharmacology*.
7. Banks, S. J., Eddy, K. T., Angstadt, M., et al. (2007). Amygdala–prefrontal functional connectivity. *Biological Psychiatry*.
8. Goldin, P. R., McRae, K., Ramel, W., & Gross, J. J. (2008). The neural bases of emotion regulation. *NeuroImage*.
9. Milad, M. R., & Quirk, G. J. (2012). Fear extinction as a model for translational research. *Nature Reviews Neuroscience*.
10. Davidson, R. J. (2004). Well-being and affective neuroscience. *Psychophysiology*.
11. Hariri, A. R. (2015). Neurobiology of anxiety and affective disorders. *Annual Review of Neuroscience*.
12. Kim, M. J., Gee, D. G., et al. (2011). Amygdala–PFC connectivity and stress reactivity. *Journal of Neuroscience*.



## ***Modern American Journal of Medical and Health Sciences***

**ISSN (E):** 3067-803X

**Volume 01, Issue 09, December, 2025**

**Website:** usajournals.org

***This work is Licensed under CC BY 4.0 a Creative Commons Attribution 4.0 International License.***

---

13. Hofmann, S. G., et al. (2012). Mindfulness and emotion regulation. *Clinical Psychology Review*.
14. Thibodeau, R., et al. (2017). Frontal EEG asymmetry and affective processes. *Journal of Abnormal Psychology*.
15. Koenigs, M., & Grafman, J. (2009). The functional role of vmPFC in emotion. *Brain*.
16. Narmetova, Y. K. (2016). Organization of the psychological support in the cardiology clinics. *Theoretical & Applied Science*, (7), 28-31.
17. Karimovna, N. Y., Khasanboy, A., Iltifotkhon, A., Khabiba, N., & Adiba, M. (2023). Psychoemotional characteristics in psychosomatic diseases. *The Scientific Temper*, 14(04), 1444-1450.
18. Narmetova, Y. (2024). COMPARATIVE ANALYSIS OF THE PSYCHOLOGICAL STATE AND SOCIO-PSYCHOLOGICAL CHARACTERISTICS OF PATIENTS WITH PSYCHOSOMATIC DISEASES. *Current approaches and new research in modern sciences*, 3(3), 83-87.