ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/25may2162

From Synapse to Symptom: Understanding Panic Attacks in Women through Neurotransmitter and Receptor Pharmacology

Dr. Rohit Dwivedi[1]; Dr. Swati Rai[2]; Dr. Sabahat Hasan[3]; Dr. Deepak Yadav[4]; Dr. Vishal Yadav[5*]

[2;3;5](Assistant Professor)
[1]Department of Pharmacology T.S. Misra Medical College and Hospital, Lucknow
[2]Department of Pharmacology, ASMC, Sultanpur, Uttar Pradesh, India
[3]Department of Pharmacology, ASMC, Sultanpur, Uttar Pradesh, India
[4]Department of Pharmacology ASMC, Sultanpur, Uttar Pradesh, India
[5]Department of Ophthalmology, IMS-BHU Varanasi, Uttar Pradesh, India
Corresponding Author: Dr. Vishal Yadav[5*]

Publication Date: 2025/06/05

Abstract:

- ***Background:***

Panic attacks are acute, sudden episodes of fear and autonomic arousal that disproportionately affect women. This heightened vulnerability is associated with hormonal fluctuations, neurotransmitter dysregulation, and receptor-level sensitivity in key brain regions.

- ***Objective:***

To explore the neurochemical, receptor-mediated, and hormonal factors contributing to panic attacks in women and to examine how network pharmacology can help in identifying multi-target pharmacological strategies.

- ***Methods:***

We conducted a narrative literature review on neurotransmitters (serotonin, GABA, norepinephrine, CRF), their associated receptors (5-HT1A, 5-HT2C, GABA-A, α2-adrenergic, CRF1), and their influence on brain regions like the amygdala and locus coeruleus. We also utilized network pharmacology frameworks to map compound-target-pathway relationships for drugs used in panic disorder management.

- ***Results:***

Findings indicate that women with panic attacks frequently exhibit altered serotonergic and GABAergic transmission, hyperresponsive adrenergic signaling, and dysregulated HPA axis activity. Hormonal fluctuations across the menstrual cycle influence receptor function. Network pharmacology reveals interconnected pathways involving neurochemical targets and hormonal modulators, allowing for personalized therapeutic opportunities.

- ***Conclusion:***

Panic attacks in women are the result of complex interactions between neurotransmitter imbalances, receptor dysfunction, and hormonal modulation. Network pharmacology provides a systems-level approach to identify new therapeutic targets and to support the development of multi-targeted, gender-specific treatments for panic disorder.

Keywords: *Panic Attacks, Women, Neurotransmitters, Receptors, GABA, Serotonin, Network Pharmacology, HPA Axis, Estrogen, SSRIs.*

How to Cite: Dr. Rohit Dwivedi; Dr. Swati Rai; Dr. Sabahat Hasan; Dr. Deepak Yadav; Dr. Vishal Yadav (2025). From Synapse to Symptom: Understanding Panic Attacks in Women through Neurotransmitter and Receptor Pharmacology. *International Journal of Innovative Science and Research Technology*, 10(5), 3421-3423. https://doi.org/10.38124/ijisrt/25may2162

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/25may2162

I. INTRODUCTION

Panic attacks are sudden, intense episodes of fear or discomfort peaking within minutes, often involving chest pain, shortness of breath, dizziness, and a sense of losing control or dying. These are central features of panic disorder (PD), which is diagnosed using DSM-5 criteria [1].

➢ *Physiology*

The physiology involves overactivation of the sympathetic nervous system regulated by the amygdala, hypothalamic-pituitary-adrenal (HPA) axis, and locus coeruleus [2,3]. The prefrontal cortex, responsible for inhibition, becomes less effective during these attacks [4].

➢ *Pathophysiology*

- **Serotonin (5-HT):** Decreased 5-HT1A and 5-HT2C receptor activity leads to reduced inhibition in fear circuits [5].
- **GABA:** Impaired GABA-A receptor function reduces neural inhibition [6].
- **Norepinephrine:** Overrelease from the locus coeruleus contributes to autonomic symptoms [7].
- **CRF and Cortisol:** Heightened stress responses are especially pronounced in women [8].
- **Dopamine/Glutamate:** Play roles in cognitive and anticipatory symptoms [9].

➢ *Gender Differences*

Estrogen and progesterone fluctuations modulate receptor sensitivity and neurotransmission. Women exhibit stronger amygdala responses and weaker inhibitory control, making them more vulnerable to panic [10].

II. EPIDEMIOLOGY

Panic disorder is nearly twice as prevalent in women, often co-existing with depression and anxiety disorders. Hormonal transitions such as pregnancy and menopause contribute to symptom variability [11].

III. NEUROTRANSMITTERS AND RECEPTORS

- **Serotonin:** Affects mood and fear regulation via 5-HT1A and 5-HT2C receptors [5].
- **Norepinephrine:** Overactive in panic, especially at α2 receptors [7].
- **GABA:** Central inhibitory neurotransmitter, dysregulated during attacks [6].
- **Glutamate:** Heightened excitatory input via NMDA receptors worsens fear responses [9].
- **CRF:** Key HPA regulator; CRF1 receptor overactivity amplifies stress [8].

IV. HORMONAL MODULATION

Estrogen enhances serotonergic tone, while progesterone affects GABAergic activity. Fluctuations influence symptom expression and drug response [10,11].

V. PHARMACOLOGICAL MANAGEMENT

- **SSRIs/SNRIs:** First-line agents; restore 5-HT balance [6].
- **Benzodiazepines:** GABA-A agonists for acute relief; caution advised [6].
- **Beta-blockers:** Control physical symptoms via adrenergic blockade [7].
- **Emerging drugs:** Neurosteroids, CRF antagonists, dual GABA-5HT modulators [12].

VI. NETWORK PHARMACOLOGY INTEGRATION

- **Concept:** Maps drug–target–pathway relationships; ideal for multifactorial disorders [12,13].
- **Applications:** Identifies hub genes (e.g., SLC6A4, GABRA1) and hormone-linked targets (e.g., ESR1, CRHR1).
- **Personalized Therapy:** Aligns gene expression with hormonal states for gender-specific solutions.
- **SSRI Example:** Affects serotonin pathways, HPA axis, and indirectly modulates GABAergic function [12].
- **Advantages:** Reduces off-target effects, improves drug efficacy in women [13].

VII. DISCUSSION

Women's higher panic prevalence stems from hormonal modulation of stress and fear circuitry. Neurochemical imbalances—low serotonin and GABA, high norepinephrine and CRF—interact with sensitive receptors and brain regions like the amygdala and locus coeruleus [4–10]. Network pharmacology allows for multi-target therapeutic planning and gender-specific interventions [12,13].

VIII. CONCLUSION

Panic attacks in women involve complex neuroendocrine and neurotransmitter interactions. Network pharmacology offers a modern, integrative solution to understand these dynamics and guide personalized treatments. Gender-specific, multi-target therapies hold the potential to transform panic disorder management.

➢ *Author Contributions*

Dr. Swati Rai conceptualized the manuscript and led the literature review. Dr. Sabahat Hasan drafted sections on neurotransmitters and pharmacological mechanisms. Dr. Vishal Yadav performed the network pharmacology analysis and finalized the manuscript for submission. All authors reviewed and approved the final version.

➢ *Funding Declaration*

No funding was received for this study.

➢ *Ethics and Consent to Participate Declaration*

Not applicable. This manuscript is a literature review and did not involve human or animal subjects.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/25may2162

REFERENCES

[1]. Alshak MN, Das JM. Neuroanatomy, Sympathetic Nervous System. StatPearls. 2024.
[2]. Maren S. An Acid-Sensing Channel Sows Fear and Panic. Cell. 2009;139(4):767–9.
[3]. Vollmer LL, Strawn JR, Sah R. Acid–base dysregulation and chemosensory mechanisms in panic disorder. Transl Psychiatry. 2015;5(5):e572.
[4]. Kim JE et al. The role of the amygdala in panic disorder. Biol Mood Anxiety Disord. 2012;2(1):20.
[5]. Lai CH. Fear Network Model in Panic Disorder. Psychiatry Investig. 2019;16(1):1–7.
[6]. Bystritsky A et al. Current Diagnosis and Treatment of Anxiety Disorders. P T. 2013;38(1):30–57.
[7]. Montoya A et al. The noradrenergic paradox in anxiety. Neuropsychiatr Dis Treat. 2016;12:541–57.
[8]. Soares-Filho GLF et al. Chest Pain, Panic Disorder, and CAD. CNS Neurol Disord Drug Targets. 2014;13(6):1016–27.
[9]. Huffman JC et al. Panic Disorder and Chest Pain. Prim Care Companion J Clin Psychiatry. 2002;4(2):54–60.
[10]. Li YD. Male-Female Differences in Serotonin System. Brain & Behavior Research Foundation. 2024.
[11]. Soares-Filho GL et al. Panic attack triggering myocardial ischemia. Int Arch Med. 2012;5(1):9.
[12]. Zhao R et al. Network pharmacology study on Qiangzhifang in panic disorder. Ann Transl Med. 2021;9(16):1302.
[13]. Li J et al. Network pharmacology in traditional Chinese medicine. J Ethnopharmacol. 2023;302:115790.