The Role of the Default Mode in Anxiety Disorders El Papel del Modo Predeterminado en los Trastornos de Ansiedad O Papel do Modo Padrão nos Transtornos de Ansiedade

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Abstract
The default mode network (DMN) is a large brain network that is most active when we are at rest or thinking about ourselves. It plays a key role in shaping our sense of self through memory, understanding others, and imagining the future. Recent research shows that problems with the DMN has a major role in anxiety disorders. People with anxiety often show a mix of DMN underconnectivity during rest, which disrupts their internal narrative, and overactivity when facing threats or focusing on tasks. These issues are made worse by poor coordination with the brain's salience network and executive control networks, creating an imbalance that leads to symptoms like constant rumination, negative memory bias, and unhelpful self-focus. We look at these patterns in generalized anxiety disorder, social anxiety disorder, and show how a disrupted self-narrative and increased self-other monitoring contribute to symptoms. We also discuss how treatments such as cognitive-behavioral therapy, mindfulness, and new medications can help rebalance the DMN and reduce anxiety. While current research has some limitations, focusing on the DMN may help guide the development of better treatments for anxiety disorders.

Keywords: Default Mode Network, Anxiety Disorders, Self-Referential Thinking, Neuroimaging, Rumination

1. Introduction
Anxiety disorders are among the most common and disabling mental health problems in the world, with symptoms that affect thinking, emotions, and the body. Initially, neuroscience research focused on fear responses and the regions involved with threat processing. However, more recent studies show that the DMN also plays an important role in anxiety.

The DMN was discovered just over twenty years ago and is a large brain network that becomes more active when we are at rest or in a state of introspection. It is the neural basis for constructing the self, including thoughts about oneself, personal memories, plans in the future, and mentalization processes [1]. When the DMN is dysregulated, it can cause psychological distress. Research shows that, in cases of pathological anxiety, the DMN has low connectivity at rest, becomes hyperactive in responses to threats, and has difficulty communicating with other cognitive control and salience networks [1,2].

Dysregulation of the DMN appears in the main symptoms of anxiety, such as constant rumination, excessive worry, a tendency to recall negative experiences, and a distorted view of oneself and others [3]. Understanding the role of the DMN in anxiety is important for explaining the mechanisms of this suffering and creating more effective treatments. Moreover, DMN dysfunction has specific characteristics in disorders such as Generalized Anxiety Disorder, Social Phobia, which highlights new therapeutic possibilities focused on balancing this central network of human experience.

2. Anxiety Disorders
Anxiety disorders are a group of psychological conditions characterized by excessive fear, persistent worry, or hyperreactivity to perceived threats, significantly affecting social, occupational, or academic functioning. According to the most recent global data, anxiety disorders are among the most common mental health conditions, affecting nearly 301 million people worldwide, with a particularly high prevalence among adolescents and young adults [4]. Anxiety disorders can be categorized into different types. First, the most common type is generalized anxiety disorder, which is characterized by difficult-to-control worry, accompanied by muscle tension, irritability, and sleep disturbances [5]. Panic disorders are also a part of anxiety disorders, they involve recurrent and unexpected panic

attacks, accompanied by intense physiological symptoms such as palpitations or shortness of breath. Other forms of anxiety, such a social anxiety, are characterized by a marked fear of situations involving interaction or performance; they are particularly prevalent among young people [6]. Recent studies highlight the major functional impact of these disorders and the effectiveness of cognitive-behavioral approaches and digital interventions in their management [7,8].

3. The Default Mode Network (DMN): The Basis of the 'Self'

The DMN is a large-scale brain network formed by interconnected regions, such as the medial prefrontal cortex (mPFC), the posterior cingulate cortex (PCC)/precuneus, the angular gyrus (AG) in the inferior parietal lobe, and structures of the medial temporal lobe (MTL), including the hippocampus [1,10,11].

This network becomes more active during rest or when a person is engaged in internal mental processes, such as self-reflection, retrieval of autobiographical memories, simulation of future events, and daydreaming [1,12]. This activity is essential for creating a continuous internal narrative, integrating past experiences, current self-perception, and projection into the future, which supports a coherent sense of identity (self).

The DMN is not a single structure. Indeed, it has specialized functional subsystems, which are mPFC and medial temporal/hippocampal subsystem. The mPFC is involved in self-referential evaluation, construction of autobiographical context, and emotional regulation, while the medial temporal/hippocampal subsystem is more related to the retrieval of detailed episodic memories and the sensory and emotional richness of remembered experiences [1,13].

The dynamic interaction between these subsystems integrates information from memory, meaning, and emotion, forming the neural basis of our continuous personal narrative. This flow of internal thought defines who we are, how we relate to others, and how we project our future [1].

4. DMN Dysfunction in Anxiety: A Neurophysiological Paradox

When anxiety becomes pathological, the DMN appears to have a contradictory dysfunction, either highly activated, leading to rumination, or hypoactive in certain subnetworks, which explains the difficulty in regulating oneself and maintaining a calm state when necessary. According to Menon (2023), the DMN, which is responsible for internal thoughts, the Salience Network (SN), which is responsible for threat detection, and finally the Frontoparietal Executive Control Network (FPN), which is responsible for executive control, must all be in balance to function properly [1].

Researchers have shown that people with pathological anxiety, have a desynchronized DMN at rest because the key areas that comprise it, such as the medial prefrontal cortex (mPFC) and the posterior cingulate cortex (PCC), are less connected to each other [10]. As a result, the DMN no longer functions smoothly, disrupting emotions and internal narrative, and increasing rumination [1].

Paradoxically, in response to threat stimuli or during tasks, the DMN often exhibits hyperactivity and fails to suppress its activity adaptively [14]. according to Bryant (2019) and Mishra and Varma (2023), this network allows us to perceive dual behavior at rest and during action, which is called a dual pattern [15,16]. However, in this network there is a disturbance in adaptive flexibility, meaning that it does not adapt correctly in different situations. In other words, during rest there is weak connectivity in the network and hyperactivity in the network when performing the task. This inflexibility occurs partly due to dysfunction in the Salience Network, whose center in the anterior insula acts as an important neural "switch" to suppress the DMN and activate the FPN for external demands [1]. When this mechanism fails, the DMN inadequately interferes with goal-directed cognition.

DMN dysfunction can be observed in other contexts, for example in certain clinical conditions such as generalized anxiety disorders (GAD) and social phobia. In these conditions, there may be a dysfunction of the network because the person experiences intense anxiety and rumination. This dominance is due to threatening scenarios created in the brain.

The DMN plays an essential role in theory of mind, i.e., knowing what others are thinking and feeling [3]. However, in social anxiety disorder (SAD), the DMN is overactive, meaning that the fear of being judged by others is heightened and distorted. This creates hypervigilance and a fear of being judged negatively by others.

In brain systems, these symptoms are maintained by dysfunctional interactions between the DMN, the Salience Network (SN), and the Central Executive Network (CEN) [2]. In anxious individuals, the DMN, CEN, and Salience Network are disrupted, leading to an imbalance. The fact that the DMN is activated in certain situations leads to rumination, a cycle of negative thoughts and therefore the other networks are less effective at regulation and controlling the situation [17]. Finally, this explains the difficulty in adopting effective strategies and being able to have an easy life [18].

5. Therapeutic Implications: Rebalancing the Network

Dysregulation of the Default Mode Network (DMN) in pathological anxiety shows up in core clinical symptoms through two main pathways, which are autobiographical memory and mentalization. When it comes to autobiographical memory, the content recalled tends to become selectively negative or threatening. This shift is not simply a recall error; it reflects an active process within the DMN that strengthens a personal narrative of vulnerability and danger. As negative memories become more present in self-perception, the hyperactive DMN amplifies hypervigilance and constant doubt about one's own abilities. In addition, the DMN plays a central role in mentalization, the capacity to understand the mental states of others [3].

In SAD, its hyperactivity makes this function a threat mechanism. As a result, the simulation of others judgments becomes exaggerated and distorted, increasing self-monitoring

and the fear of negative evaluation. This process produces an internal split, in which the person becomes highly focused on their own social performance ("self as actor") while, at the same time, viewing it critically from an external point of view ("self as observer"). Together, these processes strongly intensify social discomfort (Ruth et al., 2020).

In the brain, these symptoms are maintained by dysfunctional interactions between the DMN, the Salience Network (SN), and the Central Executive Network (CEN) [19,20]. In anxiety disorders, this mechanism does not operate as it should. The SN struggles to suppress DMN in situations that require external attention, which allows the DMN to remain active and introduce self-referential and threatening thoughts. At the same time, the CEN, which is responsible for cognitive control, has less capacity to inhibit this inappropriate DMN activity [17]. This leads to an imbalance between the three networks, trapping the person in rigid cycles of negative thinking and reducing the cognitive flexibility needed for healthy adaptation.

Understanding this cerebral mechanism, offers a framework that allows to develop more precise and better underlying process-centered interventions. The cognitive behavioral therapy (CBT) acts in a direct way on the content of the altered internal narrative», generated by an overactive DMN. By modifying negative automatic thinking, the CBT facilitates the cognitive reevaluation which is accompanied by a functional decoupling between mPFC and the PCC. This neurofunctional phenomenon represents the reduction of the influence of self-centered and negative thought patterns, directly countering the ruminative focus of the DMN [21].

Complementarily, mindfulness practices and Acceptance and Commitment Therapy (ACT) do not aim to change the content of thoughts, but rather to alter the individual's relationship with them. By strengthening present-moment attention and promoting non-judgmental awareness of internal states, these approaches reduce identification with anxious narratives. From a neuroscientific perspective, this is reflected in reduced DMN activation and increased engagement of the salience network and the CEN, there by supporting the brain "switching" that allows detachment from maladaptive internal mental states [21].

Beyond psychological approaches, new neurofeedback techniques aim to directly normalize DMN connectivity patterns, providing targeted brain training to restore a healthy balance. Additionally, innovative pharmacological interventions show potential to rapidly restore the dynamics of brain networks. Early evidence suggests that selective serotonin reuptake inhibitors (SSRIs), particularly psychedelic substances and MDMA-assisted therapy, may facilitate functional desegregation and rebalancing between the DMN, the salience network (SN), and the CEN. These compounds are thought to promote a true neuroplastic "reset," enhancing flexibility and communication between networks that, in pathological anxiety, had become rigid and dysregulated.

6. Methodological Considerations and Future Directions

It is important to acknowledge the limits that characterize this line of research. A large part of the existing work relies on cross-sectional designs, which makes it difficult to establish causality: we still cannot determine whether DMN hypoconnectivity is a vulnerability factor that precedes anxiety, or if it develops because of long-standing anxiety [22]. In addition, the clinical samples used across studies are often highly heterogeneous, and imaging protocols vary considerably from one laboratory to another, creating confounding factors that affect the robustness and generalizability of the findings [23]. Longitudinal studies are beginning to clarify some of these issues (for example, by showing that childhood trauma may influence DMN maturation) but they remain limited by small samples and by the difficulty of adequately controlling for psychiatric comorbidities or broader socio-environmental factors [23,24].

Despite these limitations, current research confirms the central importance of DMN dysregulation in the neurobiology of anxiety. Its dual and seemingly contradictory pattern, marked by connectivity at rest but reactivity to threat stimuli, together with its problematic interactions with the Salience Network (SN) and the Central Executive Network (CEN), now forms a coherent neurofunctional framework. This model explains important clinical symptoms, including persistent rumination, negative memory biases and cognitive rigidity.

Therefore, future research that addresses these current limitations, using large-scale longitudinal studies and rigorous variable control, is essential Additionally, developing interventions that aim to restore more balanced DMN connectivity and dynamics, whether through psychotherapy, medication, or neurofeedback, appears to be one of the most promising approaches for improving treatment outcomes in anxiety disorders.

7. Conclusion

In conclusion, recent research has shown that the Default Mode Network (DMN) plays a central role in the neurobiology of anxiety disorders. Dysfunction of the DMN results in resting-state hypoconnectivity and hyperreactivity to threat signals, as well as maladaptive interactions with the Salience Network (SN) and the Central Executive Network (CEN). According to Lanius et al. (2020), this pattern is observed across multiple anxiety disorders [19].

These disruptions help explain common symptoms such as rumination, biased memory recall, and cognitive rigidity. Moreover, when the DMN is excessively influenced by threat-related content, it fragments the internal narrative that underpins one's sense of self, leading to disturbances in self-perception and identity.

These insights have practical implications. Targeting the DMN in treatment allows for the development of interventions designed to restore its balance and dynamics, including mindfulness, cognitive-behavioral therapy (CBT), and psychedelic-assisted psychotherapies. By restoring cognitive flexibility, individuals may disrupt cycles of negative thinking and reassert mastery over their inner dialogue.

Looking forward, the diversity of samples underscores the need for longitudinal and methodologically rigorous research to better understand causal mechanisms and optimize treatment strategies. Ultimately, understanding the DMN clarifies the neural bases of anxiety and provides a foundation for developing more individualized and efficacious interventions focused on the human experiences.

References

1. Menon, V. (2023). 20 years of the default mode network: A review and synthesis. *Neuron, 111*(16), 2469-2487.
2. Yuan, M., Liu, B., Yang, B., Dang, W., Xie, H., Lui, S., ... & Zhang, W. (2023). Dysfunction of default mode network characterizes generalized anxiety disorder relative to social anxiety disorder and post-traumatic stress disorder. *Journal of affective disorders, 334,* 35-42.
3. Dixon, M. L., Moodie, C. A., Goldin, P. R., Farb, N., Heimberg, R. G., Zhang, J., & Gross, J. J. (2022). Frontoparietal and default mode network contributions to self-referential processing in social anxiety disorder. *Cognitive, Affective, & Behavioral Neuroscience, 22*(1), 187-198.
4. Bie, F., Yan, X., Xing, J., Wang, L., Xu, Y., Wang, G., ... & Rao, Z. (2024). Rising global burden of anxiety disorders among adolescents and young adults: trends, risk factors, and the impact of socioeconomic disparities and COVID-19 from 1990 to 2021. *Frontiers in Psychiatry, 15*, 1489427.
5. Papola, D., Miguel, C., Mazzaglia, M., Franco, P., Tedeschi, F., Romero, S. A., ... & Barbui, C. (2024). Psychotherapies for generalized anxiety disorder in adults: a systematic review and network meta-analysis of randomized clinical trials. *JAMA psychiatry, 81*(3), 250-259.
6. Walder, N., Frey, A., Berger, T., & Schmidt, S. J. (2025). Digital Mental Health Interventions for the Prevention and Treatment of Social Anxiety Disorder in Children, Adolescents, and Young Adults: Systematic Review and Meta-Analysis of Randomized Controlled Trials. *Journal of Medical Internet Research, 27,* e67067.
7. Kolaas, K., Berman, A. H., Hedman-Lagerlöf, E., Lindsäter, E., Hybelius, J., & Axelsson, E. (2024). Internet-delivered transdiagnostic psychological treatments for individuals with depression, anxiety or both: a systematic review with meta-analysis of randomised controlled trials. *BMJ open, 14*(4), e075796.
8. Fatouros, P., Tsirmpas, C., Andrikopoulos, D., Kaplow, S., Kontoangelos, K., & Papageorgiou, C. (2025). Randomized controlled study of a digital data driven intervention for depressive and generalized anxiety symptoms. *npj Digital Medicine, 8*(1), 113.
9. Azarias, F. R., Almeida, G. H. D. R., de Melo, L. F., Rici, R. E. G., & Maria, D. A. (2025). The Journey of the Default Mode Network: Development, Function, and Impact on Mental Health. *Biology, 14*(4), 395.
10. Graner, J., Oakes, T. R., French, L. M., & Riedy, G. (2013). Functional MRI in the investigation of blast-related traumatic brain injury. *Frontiers in neurology, 4,* 16.
11. Xiong, H., Guo, R. J., & Shi, H. W. (2020). Altered default mode network and salience network functional connectivity in patients with generalized anxiety disorders: An ICA-based resting-state fMRI study. *Evidence-Based Complementary and Alternative Medicine, 2020*(1), 4048916.
12. Tashjian, S. M., Goldenberg, D., Monti, M. M., & Galván, A. (2018). Sleep quality and adolescent default mode network connectivity. *Social cognitive and affective neuroscience, 13*(3), 290-299.
13. Kolesar, T. A., Bilevicius, E., Wilson, A. D., & Kornelsen, J. (2019). Systematic review and meta-analyses of neural structural and functional differences in generalized anxiety disorder and healthy controls using magnetic resonance imaging. *NeuroImage: Clinical, 24*, 102016.
14. Kim, G. W., Yoon, W., & Jeong, G. W. (2018). Whole-brain volume alteration and its correlation with anxiety severity in patients with obsessive-compulsive disorder and generalized anxiety disorder. *Clinical imaging, 50,* 164-170.
15. Bryant, R. A. (2019). Post-traumatic stress disorder: a state-of-the-art review of evidence and challenges. *World psychiatry, 18*(3), 259-269.
16. Mishra, A. K., Varma, A. R., & Varma, A. (2023). A comprehensive review of the generalized anxiety disorder. *Cureus, 15*(9).
17. Zidda, F., Andoh, J., Pohlack, S., Winkelmann, T., Dinu-Biringer, R., Cavalli, J., ... & Flor, H. (2018). Default mode network connectivity of fear-and anxiety-related cue and context conditioning. *Neuroimage, 165*, 190-199.
18. Lanius, R. A., Terpou, B. A., & McKinnon, M. C. (2020). The sense of self in the aftermath of trauma: Lessons from the default mode network in posttraumatic stress disorder. *European journal of psychotraumatology, 11*(1), 1807703.
19. Chou, T., Deckersbach, T., Dougherty, D. D., & Hooley, J. M. (2023). The default mode network and rumination in individuals at risk for depression. *Social cognitive and affective neuroscience, 18*(1), nsad032.
20. Li, R., Shen, F., Sun, X., Zou, T., Li, L., Wang, X., ... & Chen, H. (2023). Dissociable salience and default mode network modulation in generalized anxiety disorder: a connectome-wide association study. *Cerebral Cortex, 33*(10), 6354-6365.
21. Nogueira, S., Nascimento, C. M., & Krupskyi, O. P. (2025). The evolution of cognitive and behavioural therapies across three generations–a state of the art.
22. Acuña, A., Morales, S., Uriarte-Gaspari, L., Aguirre, N., Brandani, A., Huart, N., ... & Gradin, V. B. (2025). Increased default mode network activation in depression and social anxiety during upward social comparison. *Social cognitive and affective neuroscience, 20*(1), nsaf012.
23. Hao, X., Ma, M., Meng, F., Liang, H., Liang, C., Liu, X., ... & Ming, D. (2025). Diminished attention network activity and heightened salience-default mode transitions in generalized anxiety disorder: Evidence from resting-state EEG microstate analysis. *Journal of Affective Disorders, 373,* 227-236.
24. Ho, S. I., Lin, I. M., Hsieh, J. C., & Yen, C. F. (2024). EEG coherences of the default mode network among patients comorbid with major depressive disorder and anxiety symptoms. *Journal of Affective Disorders, 361*, 728-738.
25. Shen, Z., Yang, X., She, T., Zhao, G., Dou, Z., Luo, Y., ... & Yu, S. (2024). Deficits in brain default mode network connectivity mediate the relationship between poor sleep quality and anxiety severity. *Sleep, 47*(3), zsad296.

26. Wu, Y., Li, X., Ji, X., Ren, W., Zhu, Y., Chen, Z., & Du, X. (2025). Trends in the epidemiology of anxiety disorders from 1990 to 2021: A global, regional, and national analysis with a focus on the sociodemographic index. *Journal of Affective Disorders, 373,* 166-174.