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Correspondence

Unraveling the Neurovisual Interconnection and Threat Processing in Posttraumatic Stress Disorder: New Perspectives and Future Directions

To the Editor:

The recent review by Harnett *et al.* (1) highlighted a critical yet underexplored dimension of posttraumatic stress disorder (PTSD): the intricate interplay between visual and threat-processing neurocircuitry. The recognition that PTSD implicates not only core threat circuits, including the prefrontal cortex, hippocampus, and amygdala, but also the ventral visual stream represents a paradigm shift in understanding the disorder. The reciprocal connections between these systems emphasize their joint role in encoding, processing, and retrieving threat-related memories, with sensorial inputs likely playing a pivotal role in shaping trauma-related responses. This raises essential questions, such as how the ventral visual stream uniquely contributes to the emotional salience of trauma-related stimuli and whether specific visual properties such as contrast, motion, or spatial frequency amplify threat memory formation. Addressing these questions could enhance our understanding of PTSD susceptibility and resilience. Furthermore, the bidirectional relationship between visual and threat circuits suggests novel therapeutic opportunities, including the use of neuromodulation techniques like transcranial magnetic stimulation targeting visual cortical regions to disrupt maladaptive threat memory consolidation. Clinical trials are warranted to assess the efficacy and safety of such interventions (2,3).

Structural changes in the brain, particularly gray matter atrophy in the primary visual cortex (V1) and other occipital regions, underscore the profound neurobiological impact of trauma. Individuals exposed to childhood trauma exhibit more pronounced reductions in cortical volume and thickness compared with those exposed to adult trauma, suggesting that childhood trauma disrupts the maturation of the ventral visual stream and amplifies susceptibility to PTSD through lifelong dysregulation of visual threat connectivity (4,5). Alterations in white matter integrity, such as decreased fractional anisotropy in the inferior longitudinal fasciculus, further highlight structural disconnections between visual and threat circuits. As the inferior longitudinal fasciculus links the ventral visual stream to the amygdala, disruptions here could impair rapid threat evaluation. Future research should explore whether targeted cognitive rehabilitation can restore inferior longitudinal fasciculus connectivity and improve outcomes in trauma-exposed individuals (6,7).

Functional abnormalities in the lingual and fusiform gyri, as noted in the review, provide further insights into PTSD pathophysiology. Hyperresponsivity to threat-related stimuli in these regions suggests heightened salience of trauma-related cues, though significant variability exists depending on trauma severity and stimulus characteristics. Some studies, for instance, report hypoactivation in response to emotional stimuli, indicating that visual reactivity may vary based on trauma history, symptomatology, or coping strategies (8,9). This underscores the need for task-specific functional imaging studies that delineate contexts in which visual regions become over- or underactive. Integrating eye tracking and psychophysiological measures into these studies could provide a comprehensive view of how visual attention biases modulate threat reactivity and symptom severity.

The differential impacts of childhood versus adult trauma on neurocircuitry are another critical avenue for understanding PTSD etiology. Childhood trauma appears to exert a greater influence on the development of visual circuits and their connectivity with threat-processing regions, aligning with the notion that early-life adversity disrupts sensorimotor-associative axis development, predisposing individuals to maladaptive threat perception and memory formation (10). In contrast, adult trauma primarily affects established threat circuits, leveraging preexisting visual-threat connections to exacerbate symptoms. These distinctions highlight the importance of age-specific interventions (11). For childhood trauma survivors, therapies might focus on rewiring disrupted visual circuits through sensory integration and exposure-based approaches (12,13). Conversely, for adult-onset PTSD, treatments could prioritize enhancing prefrontal cortex–mediated regulation of threat responses.

In light of these findings, we propose several intervention strategies. Neurostimulation techniques, such as transcranial magnetic stimulation or transcranial direct current stimulation, could be used to modulate visual cortical activity and reduce trauma-related hyperresponsivity (14). In addition to neurostimulation approaches, developmentally sensitive and less invasive interventions should be explored. For instance, visual attention bias modification training and virtual reality exposure therapy with controlled sensory parameters offer promising avenues for recalibrating visual-threat responsiveness (15,16). For children and adolescents, sensory integration therapies and mindfulness-based attention retraining may help restore disrupted sensorimotor-visual connectivity without the need for direct cortical stimulation (17). These methods warrant further empirical validation in both clinical and community settings.

Treatment designs should account for developmental sensitivity, with interventions tailored to the distinct neurobiological impacts of childhood versus adult trauma. In conclusion, the integration of visual processing into PTSD research opens new frontiers with profound implications for both basic science and clinical practice. By disentangling the complex relationships between visual and threat neurocircuitry, we can enhance our understanding of PTSD etiology and develop targeted interventions to improve outcomes for trauma survivors. We commend the authors for their groundbreaking work and encourage the field to pursue these promising directions further.

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