

Peer Review

Review of: "The Scarred Circuitry of Fear: A Computational–Clinical Synthesis of PTSD Neurobiology"

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This manuscript presents a sophisticated and well-written computational–clinical synthesis of PTSD neurobiology that fits very well within the scope of theory-driven and translational journals in computational psychiatry and affective neuroscience. The paper is conceptually ambitious, carefully argued, and generally restrained in its interpretation of empirical evidence. In particular, the emphasis on heterogeneity, moderation, and the limits of biomarker determinism is a notable strength. The proposed framing of PTSD as a disorder of threat inference and control, rather than a collection of isolated circuit abnormalities, represents a valuable and timely contribution.

From an editorial perspective, the manuscript's primary strength lies in its integrative formalization. The minimal multi-scale framework provides a coherent language for linking fear circuitry, stress physiology, learning dynamics, and clinical phenomena, and it generates testable predictions that could guide future empirical work. The extensive parameter-to-measurement mapping is especially helpful in clarifying what would count as supporting or falsifying evidence.

At the same time, several issues warrant attention before publication. First, the evidential status of the computational formalism would benefit from sharper delineation. While the model is presented as heuristic, parts of the exposition risk being read as stronger mechanistic claims than current data can adjudicate. Second, the number and interdependence of parameters raise concerns about identifiability and potential degeneracy, particularly given realistic limits of human multimodal datasets. Third, although attractor dynamics and control collapse are compelling explanatory metaphors, they remain inferential rather than directly demonstrated properties of human PTSD circuitry and should be framed accordingly. Finally, the translational implications—especially the mapping from parameters to

intervention strategies—would benefit from clearer specification of causal leverage versus state-dependent modulation.

Overall, this is a high-quality and journal-appropriate manuscript that makes a meaningful theoretical contribution. The concerns raised are primarily clarificatory rather than fundamental. With modest revisions that sharpen the methodological positioning of the formal model, clarify the limits of identifiability, and further calibrate causal claims, the paper would be well suited for publication and likely to have a lasting impact in the field.

Declarations

Potential competing interests: No potential competing interests to declare.