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Editorial Letter

Heart-Brain Interactions in Depression: Insights from HRV and Neurocognitive Correlates

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Dear Editor,

We read with genuine interest the article by Sharma et al., titled "Analysis of HAM-D Scores on Cognitive Functions and Heart Rate Variability in Patients with Major Depressive Disorder," published in the Nigerian Medical Journal. The authors address a highly relevant and timely topic, exploring how depression severity, as measured by the Hamilton Depression Rating Scale, relates to both cognitive performance and heart rate variability in individuals with Major Depressive Disorder (MDD). The study's multidisciplinary approach, which brings together psychometric, physiological, and electrophysiological measures, is particularly commendable and adds meaningful value to the literature on the systemic effects of depression.[1]

The results indicating a strong inverse relationship between HAM-D scores and cognitive test outcomes (MMSE, MoCA, P300 latency) are consistent with what we increasingly observe in clinical practice that depression often extends beyond affective symptoms, influencing cognition and even autonomic regulation.[2,3] Incorporating P300 as an objective neurophysiological marker is a thoughtful addition that lends further credibility to the findings. It is refreshing to see studies that go beyond questionnaires and incorporate measurable biological signals. That said, while the study provides valuable insights, there are a few areas where future research might benefit from refinement. The practical cross-sectional design, for instance, naturally limits the ability to understand how these associations unfold over time. As cognitive deficits and autonomic dysfunction in depression may evolve dynamically and even persist post-remission, a longitudinal design could better capture these trajectories.[4] The HRV findings are intriguing but somewhat limited by the borderline statistical significance of RMSSD and the non-significance of HF. While the direction of effect is in line with previous work, a more detailed exploration of potential confounders such as medication use, sleep disturbances, and circadian rhythms would help clarify the physiological underpinnings.[5] Furthermore, autonomic dysregulation has also been associated with psychological constructs such as alexithymia, particularly in the context of blood pressure variability in essential hypertension.[6] Such findings point to a broader psychosomatic interplay that may also be relevant in depressive disorders.

The study population, restricted to relatively young and medically healthy individuals, provides a focused sample but limits generalizability. Many patients with MDD, particularly in clinical settings, present with comorbid conditions such as diabetes, hypertension, or chronic kidney disease, all of which can independently affect both cognitive function and autonomic regulation [6,7]. Including a broader clinical population or conducting subgroup analyses could strengthen the ecological validity of future studies. Regarding cognitive testing, while MMSE and MoCA provide a general overview, breaking down which

specific domains (e.g., attention, executive function, memory) were most affected would offer more actionable insights. Depression-associated cognitive dysfunction is rarely uniform and often disproportionately affects certain areas, like processing speed and executive control.[8]

Despite these limitations, Sharma and colleagues make a strong case for the importance of viewing depression as more than a mood disorder. Their work reinforces the need for integrative treatment models that consider not only affective symptoms but also the cognitive and physiological changes that often accompany MDD.[9]This thoughtful and well-structured study will likely stimulate further investigation. We commend the authors for their contributions and look forward to future research in this important area.

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