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Neural Dysconnectivity and Affective Disorders: Integrating Sleep and Emotional Regulation Perspectives

To the Editor: The recent article "Triple Network Model–Based Functional Dysconnectivity in Young People With Major Affective Disorders With or Without Current Suicidal Ideation" by Huang and colleagues[1] provides compelling evidence that alterations within the triple-network systems are closely associated with affective pathology and suicidal ideation in young individuals. This contribution is especially relevant given the rising prevalence and severity of mood disorders among adolescents.

An additional dimension that may further enrich the authors' neurobiological framework is the role of sleep disturbance and circadian dysregulation, both of which exert substantial influence on large-scale neural networks. Robust meta-analytic data have demonstrated convergent structural and functional alterations across multiple sleep disorders and sleep loss,[2] reinforcing the bidirectional relationship between wake-sleep disruption and network-level dysfunction. Longitudinal evidence indicated that sleep disturbances mediate the association between neural connectivity anomalies and deteriorating mental health in youth.[3] Likewise, insomnia, curtailed sleep duration, and irregular circadian timing, highly prevalent in adolescents, are consistently linked to emotional dysregulation, maladaptive salience processing, and vulnerability to suicidal ideation.[4-6]

Beyond neurobiological mechanisms, the clinical impact of wake-sleep disruption on adolescent health is profound. Chronic sleep restriction during this critical developmental period is associated not only with affective instability but also with increased impulsivity, heightened stress reactivity, cognitive inflexibility, impaired academic functioning, and worsening trajectories of anxiety and depression.[4] Adolescents experiencing recurrent sleep disturbances showed higher rates of self-harm behaviors, reduced resilience, and poorer adherence to psychiatric treatment. Integrating sleep and circadian metrics into connectivity models may offer a more complete understanding of the developmental pathways leading to suicidal thinking and behavior.[5]

Given the sensitivity of the adolescent brain to perturbations in sleep architecture and circadian timing, incorporating objective or subjective sleep measures, including polysomnography, actigraphy, rest–activity rhythms, or validated sleep questionnaires, could meaningfully enhance the explanatory power of triple-network models in youth with affective disorders. These multimodal approaches may help clarify whether dysconnectivity arises secondary to mood pathology, emerges as a parallel circadian-driven process, or reflects a synergistic interaction between both domains.

Expanding the authors' conceptual model to encompass sleep-related variables may improve early neurobiological risk stratification and guide progress of targeted, developmentally appropriate interventions for young individuals at elevated risk of severe affective dysregulation and suicidal behaviors.

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