Electrophysiological Correlates of Suicide Risk in Selective Attention to Mortality-Related Stimuli

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SUBMISSION DETAILS

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Methodological Issue Being Addressed The methodological innovation of this study focuses on combining task-driven magnetoencephalography (MEG) with generative modeling approaches, such as dynamic causal modeling (DCM), to identify neurobiological markers of suicide risk. Unlike traditional studies relying on retrospective self-reports, this approach uses a task-driven paradigm (the suicide dot probe task) to objectively assess selective attention to death-related cues through neural measures. MEG provides millisecond-level temporal resolution to capture rapid attentional processes, while DCM allows examination of effective connectivity—how brain regions involved in sensory processing, attention, and emotional regulation interact during task performance. By integrating these advanced analytical approaches with a 2×2 factorial design and mixed-effects modeling, this study contributes a novel framework for characterizing the neurobiological mechanisms underlying suicidal behaviors.

Introduction Suicide is a leading cause of death. Previous suicide research has been limited by heterogeneity in individual suicide risk profiles as well as our incomplete understanding of the neurobiological mechanisms for suicide risk. Establishing a neurobiological marker of suicide risk is crucial because it can contribute to overcoming the limitations of traditional research methods in suicide research that heavily rely on retrospective self-reports. Recent research suggests that selective attention to emotional cues could be a putative biomarker of depression and suicide. However, the mechanistic understanding of how selective attention is processed in the brain remains limited.

Methods This study investigates the neural correlates of suicide risk using magnetoencephalographic (MEG) power. We examined whether suicide risk moderates the selective attention to death-related (i.e., mortality) cues in three groups (N=53): suicide risk (SR; n=17) with recent suicidal crisis, attempt history, or on-going suicidal ideation; clinical controls with depression but no suicide risk (CC; n=17); and healthy controls (HC; n=19). The 2x2 factorial design included death/life cues for mortality and congruent/incongruent cues for attention allocation. Participants focused on mortality-related (death) or non-mortality-related (life) cues versus neutral stimuli. Trials were congruent when attending to death/life cues relative to neutral cues, and incongruent when attending to neutral cues relative to death/life cues. The suicide dot probe task measured selective attention using behavioral (reaction times) and neural (MEG) data.

Statistical analysis used linear mixed-effects models (3dLMEr) in AFNI, controlling for age and biological sex. Effective connectivity was estimated using the Dynamic Causal Modeling (DCM) in the SPM12 pipeline.

Results No significant differences in reaction time were observed among the groups in any of the conditions (ps>.05). This suggests that selective attention to mortality cues may not be captured at the behavioral level. Compared to CC group, SR group exhibited decreased activity in the middle temporal gyrus (MTG) and entorhinal cortex (EC) in the beta band (15-29 Hz), especially in the death and incongruent conditions. Compared to CC group, SR group showed decreased beta and gamma (30-58 Hz) band activity in the incongruent condition, while CC group showed increased alpha (9-14 Hz) and beta band activity in the MTG within the congruent condition. Moreover, SR group revealed decreased activity in the superior parietal lobule (SPL) compared to the HC group in the alpha band (ps<.05) within the death and incongruent conditions, whereas CC group revealed decreased activity in the SPL compared to HC group in the alpha band (p<.05) within the congruent condition. These findings indicate that suicide risk can moderate the MEG power in brain regions involved in sensory information processing. Dynamic Causal Modeling revealed condition-specific connectivity differences in the SR group (posterior probabilities>.90): Death condition: reduced feedforward connectivity from visual cortex→EC→SPL→MTG, suggesting deficits in sensory updating and attention regulation. Life condition: reduced MTG→SPL connectivity, indicating impaired downregulation of attention during life-related processing. Congruent condition: MTG exerted constraining top-down influence on EC. Incongruent condition: reduced bottom-up signaling from EC to SPL. These findings suggest MTG plays a critical role in signal processing and integration during mortality-related attention orientation, with suicide risk moderating connectivity patterns across conditions.

Conclusion Our findings suggest that abnormalities in selective attention, due to inefficient signal updating as context updating continues in response to mortality cues, may serve as potential biomarkers for suicide risk. These deficits may not be apparent behaviorally, highlighting the need for neuroimaging research to characterize these impairments. These findings could enhance our understanding of the neurobiological underpinnings of suicide risk and inform the development of more effective prevention and intervention strategies.

Co-Authors

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Disclosures One or more authors report potential conflicts which are described in the program. Dr. Zarate is listed as a co-inventor on a patent for the use of ketamine in major depression and suicidal ideation; as a co-inventor on a patent for the use of (2R,6R)-hydroxynorketamine, (S)-dehydronorketamine, and other stereoisomeric dehydroxylated and hydroxylated metabolites of (R,S)-ketamine in the treatment of depression and neuropathic pain; and as a co-inventor on a patent application for the use of (2R,6R)-hydroxynorketamine and (2S,6S)-hydroxynorketamine in the treatment of depression, anxiety, anhedonia, suicidal ideation, and post-traumatic stress disorder. He has assigned his patent rights to the U.S. government but will share a percentage of any royalties that may be received by the government. Dr. Jobes receives grant support from the National Institute of Mental Health (NIMH), the National Institute of Alcohol Abuse and Alcoholism (NIAAA), the Patient-Centered Outcomes Research Institute (PCORI), and the Four Pines Fund. He also receives book royalties from Gilford Press. All other authors have no conflict of interest to disclose, financial or otherwise.