Howard Baker Talk Abstract Submission

Suicide remains a critical public health concern, with other 720,000 deaths reported annually worldwide with rising rates of suicide ideation and behavior across populations. Despite extensive theoretical development, including ideation-to-action theories such as the Interpersonal Theory of Suicide, the percise behavioral and neural mechanisms underling the transition from suicidal thoughts to behavior remains unknown. One construct of interest is negative urgency, defined as an increased propensity to engage in an impulsive action as a result of intense negative affect. While negative urgency has been linked to various psychopathologies and elevated suicide risk, findings are inconsistent. Much of the existing literature relies on self-report measures, which may fail to accurately capture the momentary behavioral and neural correlates underlying negative urgency.

Therefore, the present study seeks to address this gap by addressing how negative affect modulates inhibitory control, a core executive function implicated in the ability to suppress maladaptive or impulsive actions. This study uses emotional response inhibition (ERI) as a behavioral proxy for negative urgency, as ERI appropriately captures the interaction between affective processing and cognitive control. Further, this study integrates neural measures through event-related potentials (ERPs), focusing on the P300 component as an index of inhibitory control engagement. By combining behavioral and neurophysiological methods, this work aims to provide a mechanistic understanding of how negative emotional states influence inhibitory control processes relevant to suicide risk.

To experimentally manipulate affective states, this study employed a within-subject mood induction procedure paradigm in conjunction with an emotional stop-signal task (ESST). Participants completed the ESST under both neutral and negative mood induction conditions,

allowing for comparisons of state-dependent changes in inhibitory control to be drawn. Particularly, the ESST required participants to categorize emotionally valenced images during go trials and inhibit their response on stop-signal trials triggered by an auditory cue. Importantly, this paradigm assesses late-stage action termination, with higher stopping latencies suggesting impaired inhibitory control. Key behavioral indices include stop signal reaction time (SSRT), Go reaction time, emotional classification accuracy, negativity bias, and miss rate. Neural indices focused on the P3 ERP component, measured at frontocentral electrode sites and time-locked to stimulus onset.

Importantly, the SSRT was calculated utilizing the integration method grounded from a horse-race model approach. This model supposes proper response execution (Go processes) and inhibition (Stop process) as independent processes racing toward completion. Therefore, successful inhibition occurs when the Stop process finishes before the Go process, whereas failed inhibition reflects the opposite outcome. This approach allows for the estimation of latent inhibitory processing speed, providing a behavioral marker of response inhibition efficiency.

The present study represents a pilot investigation conducted in a sample of healthy participants ($N = 6$), with analyses focusing on within-subject differences across mood conditions rather than between-group differences in suicide risk. Although the original study design aimed to compare individuals at elevated suicide risk with healthy controls, aims for the pilot investigation are focused on examining how experimentally induced mood states influence inhibitory control processes. This approach provides a foundational step toward understanding how affective states may disrupt cognitive control mechanisms implicated in negative urgency and suicide risk.

Behavioral results indicated the negative mood induction condition did not significantly impair inhibitory control performance as indexed by the negative SSRT. Contrary to hypotheses, the projected direction of effects showed greater inhibitory control for negative stimuli following the negative mood induction condition compared to neutral mood induction condition. This trend may reflect increased hypervigilance in negative mood conditions, hence greater attentional allocation to correctly responding in stop-trials. Similarly, no significant differences were observed in Go reaction time, emotional classification accuracy, miss rate, or negativity bias across conditions. Overall, these findings suggest that, at the behavioral level, participants were able to maintain stable performance despite changes in affective context.

In contrast to the behavioral findings, neural results revealed a significant modulation of inhibitory control processes by mood state. Specifically, P3 amplitude at frontocentral sites was significantly reduced during negative mood relative to neutral mood when time-locking epochs to negative images shown by the ESST. This reduction in P3 amplitude suggests diminished neural engagement of inhibitory control mechanisms under conditions of negative affect. Importantly, P3 latency did not differ between conditions, indicating that the timing of inhibitory processing remained intact even as the magnitude of neural activation was reduced.

This pattern of results reveals a potential dissociation between behavioral and neural indices of inhibitory control, highlighting the importance of incorporating neurophysiological measures when examining affect-related cognitive processes. While task performance appeared preserved, underlying neural mechanisms showed evidence of disruption, suggesting that behavioral measures alone may underestimate the impact of negative affect on cognitive control systems. This finding aligns with broader literature indicating that ERPs, particularly the P3

component, may provide a more sensitive index of inhibitory processing than behavioral outcomes alone.

From a theoretical perspective, these findings contribute to our understanding of negative urgency as a state-dependent construct. Rather than reflecting stable trait impulsivity, negative urgency emerges from interactions between affective states and cognitive control systems. Under conditions of negative mood, reduced neural engagement of inhibitory control processes may increase vulnerability to impulsive action, even in the absence of observable behavioral deficits. This has valuable implications for ideation-to-action models of suicide, suggesting that disruptions in neural control mechanisms may precede or underlie the transition from suicidal thoughts to behavior.

Despite these contributions, several limitations must be acknowledged. The pilot sample size was small, limiting statistical power and generalizability of findings. Furthermore, the absence of a clinical sample precludes direct conclusions regarding suicide risk populations. Future research should replicate these findings in larger, clinically diverse samples and incorporate more personalized mood induction techniques to strengthen affective manipulations.

In conclusion, the present study provides preliminary evidence that negative affect selectively disrupts neural mechanisms of inhibitory control without necessarily impairing behavior. These findings highlight the importance of integrating behavioral and neural approaches to capture the complexity of cognitive control processes under emotional distress. By identifying neural markers of disrupted inhibition, this work lays the groundwork for future research examining the role of negative urgency in suicide risk and highlights potential targets for intervention aimed at strengthening cognitive control under affective distress.