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Neurocognitive mechanisms of emotion-related impulsivity: The role of arousal

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Abstract

Prior research suggests that a traitlike tendency to experience impulsivity during states of high emotion is robustly associated with many forms of psychopathology. Several studies tie emotion-related impulsivity to response inhibition deficits, but these studies have not focused on the role of emotion or arousal within subjects. The present study tested whether arousal, measured by pupil dilation, amplifies deficits in response inhibition for those high in emotion-related impulsivity. Participants ($N = 85$) completed a measure of emotion-related impulsivity, underwent a positive mood induction procedure that reduced heterogeneity in mood states, and completed a response inhibition task. Pupil dilation was used to index arousal during the response inhibition task. Generalized linear mixed effect modeling yielded the hypothesized interaction between arousal (pupil dilation) and emotion-related impulsivity in predicting response inhibition performance at the trial level. Emotion-related impulsivity relates to more difficulties with response inhibition during moments of high arousal.

Keywords

cognitive control; emotion; impulsivity; pupillometry; response inhibition

1 | INTRODUCTION

Emotion-related impulsivity is defined as the tendency to react impulsively when experiencing heightened emotional states (Whiteside & Lynam, 2001). For example, a person high in emotion-related impulsivity might express anger by abruptly acting aggressively rather than letting the anger dissipate. Emotion-related impulsivity has been shown to be statistically distinct from other forms of impulsivity (Berg, Latzman, Bliwise, & Lilienfeld, 2015; Whiteside, Lynam, Miller, & Reynolds, 2005).

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Emotion-related impulsivity predicts many different poor psychosocial outcomes, including psychopathology and problematic behaviors (Berg et al, 2015; Johnson, Carver, & Joormann, 2013; Muhtadie, Johnson, Carver, Gotlib, & Ketter, 2014). In a meta-analysis of over 40,000 individuals, emotion-related impulsivity was compared with other aspects of impulsivity and emerged as the best predictor of every clinical diagnosis and symptom group, including depression, anxiety, eating disorders, borderline personality traits, suicidality, and nonsuicidal self-injury (Berg et al., 2015). In clinical populations, emotion-related impulsivity predicts lower quality of life, higher rates of comorbidity, self-injury, suicidal action, aggression, and poor social well- being (Auerbach, Stewart, & Johnson, 2017; Muhtadie et al., 2014; Victor, Johnson, & Gotlib, 2011). Emotion-related impulsivity appears to be a transdiagnostic vulnerability factor for both internalizing and externalizing disorders (Carver, Johnson, & Timpano, 2017; Johnson, Carver, & Joormann, 2013). A growing body of longitudinal research supports the predictive power of this form of impulsivity in the onset of problems with substance abuse (Kaiser, Bonsu, Charnigo, Milich, & Lynam, 2016), eating disorders (Pearson, Combs, Zapolski, & Smith, 2012), and nonsuicidal self-injury (Riley, Combs, Jordan, & Smith, 2015).

To fully understand these effects, it is desirable to isolate neurocognitive mechanisms underlying emotion-related impulsivity. There is some evidence that persons with high levels of emotion-related impulsivity are not simply more emotionally reactive; instead, most evidence suggests that comparable emotional arousal levels differentially trigger impulsive responses among persons with high emotion-related impulsivity (Cyders & Coskunpinar, 2010; Johnson, Tharp, Peckham, Sanchez, & Carver, 2016). The evidence suggests that this form of impulsivity is not explained by emotionality alone.

Difficulties controlling impulses during states of heightened emotion represent a form of deficit in response inhibition (Bechara & Van der Linden, 2005; Carver, Johnson, & Joormann, 2008). Response inhibition is a specific facet of cognitive control that corresponds to the ability to override a planned or initiated action or to suppress an automatic or prepotent response tendency (Aichert et al., 2012). Poor response inhibition has been documented across many forms of psychopathology (Bari & Robbins, 2013; Wright, Lipszyc, Dupuis, Thayapararajah, & Schachar, 2014). It is possible that an inability to override unwanted automatic emotional impulses reflects a neurocognitive deficit in response inhibition. Indeed, findings of a recent meta-analysis indicated that high emotion-related impulsivity significantly relates to poor response inhibition (Johnson et al., 2016). Despite the positive findings, caution is warranted in that effect sizes have been fairly small and have emerged only among samples with higher impulsivity levels. Nonetheless, recent findings dovetail with these behavioral effects in identifying a differential profile of activation of the frontoparietal network during a response inhibition task for those with high emotion-related impulsivity (Chester et al., 2016; Wilbertz et al., 2014). While there is some evidence suggesting a relation between emotion-related impulsivity and response inhibition, further research is needed to understand the mechanisms driving this link.

We argue that one reason for the mixed findings is the limited attention paid to the role of heightened emotion in driving response inhibition decay. To date, only three studies in this area have tested response inhibition after mood inductions (Dekker & Johnson, 2018; Gunn

& Finn, 2015; Johnson et al., 2016). Unfortunately, these studies were constrained by limited success of mood inductions. Additional research is warranted to test the hypothesis that individuals high in emotion-related impulsivity experience emotion-induced decays in response inhibition. Because behaviors associated with emotion-related impulsivity only emerge in the context of heightened emotion states, we hypothesize that emotion-related impulsivity will be correlated with a greater within-subject decline in response inhibition during heightened emotion states.

We further hypothesize that the feature of emotion leading to impulsive behaviors is arousal, rather than valence. While early models of emotion-related impulsivity characterized a factor defined as negative urgency, the tendency to act impulsively in response to negative emotion (Whiteside & Lynam, 2001; Whiteside et al., 2005), later work identified a separable factor of positive urgency, the tendency to act impulsively in response to positive emotion (Cyders & Smith, 2007). More recently, factor analytic evidence suggests that these two factors have a common underlying core (Carver, Johnson, Joormann, Kim, & Nam, 2011; Sperry, Lynam, & Kwapil, 2018). Because tendencies to respond impulsively to heightened emotion occur across both positive and negative emotions (Cyders et al., 2007), heightened arousal, rather than valence, may be the trigger. We are unaware of data, however, that measure whether arousal induces deficits in response inhibition among persons exhibiting high levels of emotion-related impulsivity.

This study aimed to directly examine whether emotion- related impulsivity corresponds to individual differences in the effects of arousal on response inhibition. Pupil dilation provides a marker of rapid changes in arousal (Beatty & Lucero-Wagoner, 2000; Meese, 2011) and has been shown to correspond to emotional evocation (Snowden et al., 2016). The pupil serves as a window into the autonomic nervous system: dilation reflects sympathetic activity and corresponds to other peripheral measures of arousal including cardiac acceleration and skin conductance (Beatty & Lucero-Wagoner, 2000). Task-evoked pupil dilation corresponds to fluctuations in activity in the locus coeruleus, the area of the brain stem responsible for synthesis of norepinephrine (Joshi, Li, Kalwani, & Gold, 2016). Norepinephrine functions to mobilize the brain and body for action (e.g., fight or flight response) and is elevated during periods of stress (Tank & Wong, 2015). Pupils dilate and constrict rapidly in response to stimuli and, correspondingly, reflect rapid changes in norepinephrine activity during cognitive tasks.

The goal of the present study was to examine whether dynamic fluctuations in arousal would relate to response inhibition for those with higher levels of emotion-related impulsivity. Participants underwent a well-validated positive mood induction procedure to reduce heterogeneity in mood states. Dynamic fluctuations in arousal were measured by pupil dilation on each trial of a response inhibition task. First, we hypothesized that higher emotion-related impulsivity scores would relate to poorer overall response inhibition performance in general. More importantly, we further hypothesized that higher emotion-related impulsivity would interact with higher arousal, measured by pupil dilation, to predict trial-level deficits in response inhibition performance.

2 | METHOD

2.1 | Participants and design

Participants ($N = 92$) were undergraduates (53% female, age $M = 20.67$, $SD = 3.30$) who completed written informed consent before engaging in study procedures. Participants' reported ethnicities were 56.1% European/ Caucasian, 15.3% Asian, 9.6% Hispanic/Latino, and 19.2% other. They reported a mean grade point average of 3.44 ($SD = 0.39$) on a 4-point scale, and their mean socioeconomic scale as 4.76 ($SD = 1.55$) on the 10-point MacArthur scale (Adler & Stewart, 2007). Participants received partial course credit in psychology classes for taking part in the study.

All procedures were conducted during an individual session at the laboratory. Data were gathered as part of a larger study including multiple behavioral and self-report measures not discussed here, administered in random order (see Johnson et al., 2016). The antisaccade task directly followed one of four positive mood inductions. Our goal was not to consider the role of positive valence; rather, the design of the study has the advantage of reducing heterogeneity in affective state across participants.

2.1.1 | Three-factor impulsivity measure—Emotion-relevant impulsivity was measured by a self-report measure derived from a factor analysis of several scales measuring the heterogeneous construct of impulsivity and self-control (Carver et al., 2011). The three-factor analytically derived subscales include two measures of impulsive reactions to emotions and a third factor reflecting impulsiveness without an emotional antecedent. One factor, referred to as pervasive influence of feelings, corresponds to overly broad influences of (mostly negative) emotion on cognition (e.g., "My feelings greatly affect how I see the world") and motivation ("I can't get myself going"). Another factor, referred to as feelings trigger action, corresponds to speech or action in response to positive or negative (or unspecified) emotions (e.g., "When I feel a desire, I act on it immediately," and "When I feel bad, I will often do things I later regret in order to make myself feel better now"). A third factor, labeled as lack of follow-through, refers to impulsiveness interfering with completion of intended actions (e.g., "I am easily distracted by stray thoughts"), but with no mention of emotion as a precipitator. The emotion-related factors have been found to be related to depression, anxiety, manic tendencies, externalizing syndromes, and suicidality (Johnson, Carver, & Joormann, 2013; Johnson, Carver, Mulé, & Joormann, 2013), as well as genetic polymorphisms related to serotonin and to early adversity (Carver et. al., 2011). Lack of follow-through was included to test discriminant validity of emotion-related impulsivity compared to nonemotion-related impulsivity. Each subscale had high interitem reliability (feelings trigger action: $\alpha = 0.94$; pervasive influence of feelings: $\alpha = 0.80$; lack of follow-through: $\alpha = 0.89$); the three factors were all significantly correlated (feelings trigger action and pervasive influence of feelings: $r = 0.61$; feelings trigger action and lack of follow-through: $r = 0.30$; pervasive influence of feelings and lack of follow-through: $r = 0.43$, all ps < 0.01).

2.1.2 | Mood inductions—Participants were randomly assigned to take part in one of four positive mood induction procedures. Each task has been shown to induce positive mood (Cyders, Coskunpinar, & Lehman, 2012; Pronin, 2013).

Facial symmetry feedback: At the start of the session, participants were informed that research has shown a direct link between attractiveness and facial symmetry. A photo of their face was then taken to be analyzed by a special program. Later in the session, participants were provided their fake printed results from the "facial symmetry software." Results always stated that the participant has above-average facial symmetry and that 91% of people will find them attractive.

Modified Iowa gambling task: In this task, participants were presented with four decks of cards on the computer. They were told that there are "good decks" and "bad decks" and that the card they draw each trial from one of the decks would result in reward or penalty. Unlike the original Iowa gambling task (Bechara, Damasio, Damasio, & Anderson, 1994), all four decks were good decks, so that participants would win anywhere between $25 to $1,250 over the course of 100 trials. On about 25% of trials, though, one deck had cards with values of $0 to −$350. Participants were informed at the start of the task that one person would be chosen from the total participant pool that semester to receive a payout of 10% of their winnings.

Shape tracing task: During this task, participants were asked to trace over a set of three shapes (figures from Glass & Singer, 1972) without lifting their pencil from the page or retracing any parts. They were told that they would be allowed to attempt each shape only once, that the cognitive puzzles have been shown to highly relate to IQ, and that "only about 5% of individuals complete all three of these figures correctly" within the 5- min time period. In actuality, the majority of people complete this task relatively quickly, with a mean completion time of 1.73 min and standard deviation of 0.85.

Thought speed task: In this task (Pronin, 2013), participants were told to read sentences aloud without pausing or skipping. Using Microsoft PowerPoint, they were presented with 60 statements. Each statement appears one letter at a time in Arial 44-point font at a speed of 40 ms per letter until it is presented in its entirety. There is 320 ms between the conclusion of one statement and the start of the next. The first six statements are neutral, and the following 54 become increasingly positive as the task continues (e.g., from "I do feel pretty good today, through" to "Wow, I feel great!"). The task has been validated as inducing elevated mood and related characteristics such as self-confidence, energy, creativity, and risk-taking (Pronin, 2013).

2.1.3 | Mood rating—Mood was measured using a High Arousal Positive (HAP) affect scale comprised of three items (active, amused, enthusiastic) reported on a 5-point Likert scale, with 1 = *very slight or not at all* to 5 = *extremely*. To validate mood induction effects, the HAP scale was administered after mood induction and task completion.

2.1.4 | Antisaccade task—The antisaccade task (Kane, Bleckley, Conway, & Engle, 2001) is a well-validated measure of response inhibition (Miyake & Friedman, 2012). Each

trial begins with a fixation in the center of the screen (randomly varying between 200–1,800 ms), followed by a blank screen (10 ms), and then a distractor on either the left or right side of the screen at a visual angle of 8.68 degrees (250 ms). Afterward, a randomly selected target (*B*, *P*, or *R*) is presented on either the left or right side of the screen (100 ms), immediately followed by the letter *H* as a mask (50 ms), and then the number *8*. Participants are asked to indicate which letter they saw before the mask appeared, and they are given up to 10 s to respond by button press. Each trial is followed by a 400-ms intertrial interval.

The task includes prosaccade and antisaccade trials. In prosaccade trials, the distractor and target letter appear on the same side of the screen. During antisaccade trials, the distractor and target letter appear on opposite sides of the screen. Thus, an accurate response on an antisaccade trial requires inhibiting the reflexive response of looking toward the cue and, instead, voluntarily looking to the opposite side of the screen for the letter. Participants pressed keys to indicate whether the stimulus presented was a *B*, *P*, or *R*. Participants completed 10 practice trials to learn which keys to press, 10 prosaccade trials, 10 antisaccade practice trials, and then 40 antisaccade trials. Antisaccade performance was scored as correct key press (1) or incorrect key press (0). Average antisaccade accuracy was calculated as the mean score across the block of 40 antisaccade trials, with higher scores reflecting greater accuracy. Because trials occur rapidly, reaction time showed little variability. Rather, as with previous work, we focus on accuracy on antisaccade trials, controlling for prosaccade (Roberts, Hager, & Heron, 1994).

2.2 | Apparatus and data cleaning

Pupillometry was captured during the response inhibition task using a Tobii T-120 infrared eye tracker (Tobii Technologies, Dandyred, Sweden), alongside a secondary computer used to display stimuli. The response inhibition task was programmed with E-Prime Professional, Version 2.0, linked with the eye tracker using E-Prime Extensions for Tobii (Psychology Software Tools, Pittsburgh, PA). Eye tracking was recorded at 120 Hz, yielding one sample per 8.3 ms. Stimuli were displayed on a 17-in. computer monitor with 1,280 × 1,024 screen resolution. A nine-point calibration was conducted before administering the response inhibition task. The camera simultaneously recorded pupil diameter for the left and right eye. Data were cleaned using a local fit procedure described by Johnson, Miller Singley, Peckham, Johnson, and Bunge (2014). This procedure involves the use of an automated regression model to calculate the average pupil diameter across both eyes at each data point (8.3 ms), using data for one eye when data for both were not available. We aggregated across data points to calculate the mean pupil diameter over each of the 40 response inhibition trials. Data were removed if outside the range of five standard errors above or below the locally defined, weighted mean, and participants were excluded if their cleaned pupil data had less than 50% valid data.

2.3 | Data analysis plan

Preliminary analyses examined variable distributions and considered potential confounds. To provide basic descriptive information before considering multivariate models, correlations were computed between the three impulsivity scores and the response inhibition and pupil dilation scores per individual aggregated across trials. Linear and curvilinear effects were

considered throughout analyses. To test the primary hypothesis, we used generalized linear mixed effect modeling (Hox, 2002) and the lme4 package for R (Bates, Mächler, Bolker, & Walker, 2015). We used generalized linear mixed effect modeling to determine the main fixed effects and interaction of emotion-related impulsivity and pupil dilation on trial-by-trial performance on the response inhibition task.

Pupillometric research considering the predictive effect of pupil dilation on trial-level behavioral performance generally uses a time-lagged approach, in which dilation on one trial is used to predict performance on the subsequent trial (Chiew & Braver, 2013). This time-lagged approach enables the assessment of dynamic temporal processes (Ram & Gerstorf, 2009). We used this procedure to assess how arousal affects performance. Our main hypothesis was that arousal (pupil dilation) on one trial would interfere with performance on the subsequent trial for participants with higher emotion-related impulsivity.

3 | RESULTS

All analyses were completed using R and were two-tailed with alpha = 0.05. Seven participants were excluded from analyses: two for sleeping fewer than 5 hr the night before, one who did not meet 75% of attention checks interspersed throughout the questionnaire battery (i.e., “Please circle ‘a lot’ for this item”), one for not successfully obtaining 50% accuracy on prosaccade trials of the impulsivity task, and three because they had limited English skills (e.g., difficulties with comprehending instructions coupled with a small number of years speaking English). After excluding these participants, 85 participants were included in analyses.

Basic correlations among key variables are shown in Table 1. As shown, impulsivity did not significantly relate to pupil dilation, suggesting that those with high impulsivity were not prone to more or less arousal.

3.1 | Effectiveness of mood induction

A prior published report validated the successful mood induction of 63 of the present study participants by showing mood inductions led to greater skin conductance levels (SCL) and higher high-arousal positive affect ratings (see Johnson et al., 2016). The four mood inductions did not differ significantly in their effects on subjective or physiological arousal with all Fs < 1.65, ps > 0.11. Scores on the emotion-related impulsivity measure did not predict the degree of change in affect ratings or SCL, controlling for baseline, partial rs < 0.10, ps > 0.50. The degree of change in affect or SCL in response to the mood induction, controlling for baseline, did not significantly relate to antisaccade performance (all Fs < 0.96, ps > 0.33). As hoped, participants generally endorsed a mildly positive mood state after the mood induction, M = 2.51 out of 5, SD = 0.88.

3.2 | Preliminary analyses:

Potential confounds—Response inhibition and pupil diameter were not related to caffeine, alcohol, nicotine use, medication use, or hunger ratings. Response inhibition was not significantly related to age, $t(83) = 0.63$, $p = 0.95$, or gender, $t(83) = -1.58$, $p = 0.12$. Although pupil diameter has been shown in other research to be related to age and gender,

neither age, $t(74) = -0.28$, $p = 0.78$, nor gender, $t(74) = 0.47$, $p = 0.64$, was associated with pupil dilation in our sample.

3.3 | Main analyses

Our main hypothesis was that emotion-related (but not none-motion-related) impulsivity would interact with arousal as reflected in pupil dilation on a given trial of response inhibition to predict performance on the next trial. To investigate this, we used three generalized linear mixed effect models to test the three impulsivity factors separately (feelings trigger action, pervasive influence of feelings, lack of follow-through). In each model, we entered prosaccade performance, lagged pupil dilation (previous trial pupil diameter), the impulsivity factor, and the interaction of pupil dilation during the prior trial with the impulsivity factor. For each model, we included the random intercept so that trial-level data adjust for the mean pupil dilation per individual. Models with random intercepts, but not random slopes, account for individual differences in average pupil diameter.

Modeling a random intercept does not, however, account for individual differences in systematic shifts across the block of trials. To consider this variable, we also conducted these generalized linear mixed effect models with both random slopes and intercepts. The random slope models did not obtain better fit, suggesting that there was not significant between-subjects variability in change across the block, and these models are not presented here.

For the model including feelings trigger action and shown in Figure 1, there was a significant interaction of Feelings Trigger Action ×Pupil dilation on response inhibition accuracy ($\beta = -0.30$, $SE = 0.04$, $z = -2.05$, $p < 0.05$); there was no main effect for either feelings trigger action ($\beta = 0.11$, $SE = 0.99$, $z = 0.98$, $p = 0.32$) or pupil dilation ($\beta = 0.11$, $SE = 1.15$, $z = 0.14$, $p = 0.25$). These findings indicate that those higher in feelings trigger action show decays in response inhibition performance on trials in which they experience higher arousal, as measured by pupil dilation.

For the model including pervasive influence of feelings shown in Figure 2, there was a significant interaction of pervasive influence of feelings and pupil dilation on response inhibition accuracy ($\beta = -0.22$, $SE = 0.09$, $z = -2.35$, $p < 0.05$); there was no main effect for either pervasive influence of feelings ($\beta = 0.03$, $SE = 0.09$, $z = 0.35$, $p = 0.71$) or pupil dilation ($\beta = 0.16$, $SE = 0.10$, $z = 1.62$, $p = 0.10$). Findings indicate that those lower in pervasive influence of feelings show improvements in response inhibition performance on trials in which they experience higher arousal, as measured by pupil dilation.

For the model including lack of follow-through, the interaction between lack of follow-through and pupil dilation was not significant in relation to response inhibition accuracy ($\beta = 0.05$, $SE = 0.10$, $z = 0.44$, $p = 0.65$), nor was the main effect significant for either lack of follow-through ($\beta = 0.18$, $SE = 0.11$, $z = 1.70$, $p = 0.08$) or pupil dilation ($\beta = 0.08$, $SE = 0.10$, $z = 0.83$, $p = 0.41$).

4 | DISCUSSION

Because self-reported emotion-related impulsivity is related to psychopathology and poor psychological outcomes, it is important to understand the processes underlying that impulsivity. Expanding on prior evidence of an association between emotion-related impulsivity and poor response inhibition (Cyders & Coskunpinar, 2012), the present study assessed within-subject trial-level response inhibition performance. We tested the interaction of emotion-related impulsivity with dynamic shifts in trial-level arousal, as measured by pupil dilation, on subsequent response inhibition. We did not find associations between emotion-related impulsivity and overall performance on the response inhibition task (i.e., independent of arousal). However, as predicted, we found a significant interaction between emotion-related impulsivity and arousal, as measured by changes in pupil dilation, in predicting trial- level accuracy on the response inhibition task.

The obtained results support our primary hypothesis that emotion-related impulsivity would interact with arousal to predict trial-level performance on the response inhibition task. The two emotion-relevant impulsivity factors, feelings trigger action and pervasive influence of feelings, but not the general impulsivity factor, lack of follow-through, interacted with pupil diameter during the prior trial to predict accuracy on the subsequent trial. The combination of higher emotion- related impulsivity and higher arousal was associated with decline in response inhibition on the next trial. These findings suggest higher arousal causes differential effects on response inhibition, such that those higher in emotion-related impulsivity show slightly reduced accuracy while those lower in emotion-related impulsivity show improved accuracy. It is important to acknowledge that these effects appear to be driven by improved response inhibition ability for those with lower levels of emotion-related impulsivity. These findings are consistent with recent meta-analytic evidence suggesting arousal improves behavioral response inhibition in nonclinical populations (Shields, Sazma, & Yonelinas, 2016). In other words, although it is normative for arousal to enhance response inhibition, arousal does not assist those with higher levels of emotion-related impulsivity. This could help to explain their loss of control during high arousal states. Of import, these findings also demonstrate discriminant validity, given that the model with the impulsivity factor lack of follow-through did not show this effect. These models suggest arousal induces differentiated effects for response inhibition that may serve as a mechanism for emotion-related impulsivity, but not for nonemotion-related impulsivity.

It is important to acknowledge that the only effect we observed for response inhibition with impulsivity was within the context of the more carefully controlled within-subject analyses of dynamic shifts in arousal. The lack of relationship between emotion-related impulsivity and overall response inhibition performance corresponds with a growing literature suggesting that this relationship may only emerge consistently in those with clinically elevated levels of emotion-related impulsivity. For example, in a meta-analysis, the relationship between emotion-related impulsivity and response inhibition was more pronounced in clinical samples (Cyders & Coskunpinar, 2011; Johnson et. al, 2016). Prior work in a larger sample of undergraduates found a curvilinear, rather than linear, relationship between emotion-related impulsivity and response inhibition ability (Johnson et al., 2016). Although we considered curvilinear effects in our sample, our undergraduate sample may

not have experienced sufficiently high levels of emotion-related impulsivity to detect these effects. An alternative explanation could be that our sample did not exhibit sufficiently high levels of arousal overall to detect a relationship between aggregated antisaccade accuracy and emotion-related impulsivity. Our mood induction procedures were meant to foster modest increases in positive affect, which may not involve sufficient arousal to drive an overall decay in response inhibition ability for those with high levels of emotion-related impulsivity.

This study is not without its limitations. First, only a subset of our sample had manipulation checks to validate successful mood induction. Therefore, we cannot verify whether all participants experienced a positive mood state. Second, it is possible that pupil dilation is impacted by cognitive effort (Steinhauer & Hakerem, 1992). Although our results are consistent with the interpretation that pupil dilation is driven by arousal, future research should consider additional physiological indicators of arousal to investigate this alternative explanation. Third, in the current study, we employed a positive mood induction and did not test the effects of negative mood inductions. We believe these results would replicate across positive and negative mood inductions because (a) emotion-related impulsivity emerges across positive and negative emotions, and (b) pupillometry is a sensitive measure of arousal that responds to both positive and negative emotions (Babiker, Faye, & Malik, 2013). Nonetheless, there is a need for research that tests the generalizability of this effect across valence. Finally, in addition to determining whether these effects generalize across emotion states, it is important to determine whether the effects generalize across response inhibition tasks or other measures of executive function.

While this study is limited by including only a positive mood induction, we believe that these results support the model that those high in emotion-related impulsivity experience high arousal-induced changes in cognitive control. The emergence of these effects in the context of positive affect strengthen the claim that arousal may serve a primary function in eliciting impulsive responses to emotion. Items on the pervasive influence of feelings subscale tend to reflect impulsivity in response to primarily negative emotions, and yet we observed effects of arousal with this subscale. This supports the model that arousal rather than valence may be the precipitator of changes in cognitive control leading to impulsive thought and behaviors.

This study is one of the best tests to date assessing a dominant model to explain how emotion-related impulsivity emerges. Our results suggest that emotion-related impulsivity is related to arousal-induced decays in response inhibition. This study is one of the first to consider fluctuations of arousal within persons and response inhibition performance for those high in emotion-related impulsivity. These findings offer important clinical implications and future directions. Specifically, the central role of arousal in eliciting decays in response inhibition suggests that interventions targeting high arousal states or targeting cognitive control while in a high arousal state may be the most effective at treating emotion-related impulsivity. Future research is needed to better understand the mechanisms of emotion-related impulsivity and to determine the clinical utility of interventions aimed at improving these mechanisms.

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REFERENCES

Adler NE, & Stewart JE (2007). Reaching for a healthier life: Facts on socioeconomic status and health in the U.S. San Francisco, CA: John D. & Catherine T. Macarthur Foundation Research Network on Socioeconomic Status and Health.

Aichert DS, Wöstmann NM, Costa A, Macare C, Wenig JR, Möller H-J, … Ettinger U. (2012). Associations between trait impulsivity and prepotent response inhibition. Journal of Clinical and Experimental Neuropsychology, 34(10), 1–17. 10.1080/13803395.2012.706261 [PubMed: 22034987]

Auerbach RP, Stewart JG, & Johnson SL (2017). Impulsivity and suicidality in adolescent inpatients. Journal of Abnormal Child Psychology, 45, 91–103. 10.1007/s10802-016-0146-8 [PubMed: 27025937]

Babiker A, Faye I, & Malik A (2013). Pupillary behavior in positive and negative emotions Paper Presented at the International Conference on Signal and Image Processing Applications, Banff, AB, Canada 10.1109/ICSIPA.2013.6708037

Bari A, & Robbins TW (2013). Inhibition and impulsivity: Behavioral and neural basis of response control. Progress in Neurobiology, 108, 44–79. 10.1016/j.pneurobio.2013.06.005 [PubMed: 23856628]

Bates D, Mächler M, Bolker B, & Walker S (2015). Fitting linear mixed-effects models using lme4. Journal of Statistical Software, 67(1), 1–48. 10.18637/jss.v067.i01

Beatty J, & Lucero-Wagoner B (2000). The pupillary system In Handbook of psychophysiology (2nd ed, pp. 142–162). New York, NY: Cambridge University Press.

Bechara A, Damasio AR, Damasio H, & Anderson SW (1994). Insensitivity to future consequences following damage to human prefrontal cortex. Cognition, 50(1–3), 7–15. 10.1016/0010-0277(94)90018-3 [PubMed: 8039375]

Bechara A, & Van der Linden M (2005). Decision-making and impulse control after frontal lobe injuries. Current Opinion in Neurology, 18(6), 734–739. 10.1097/01.wco.0000194141.56429.3c [PubMed: 16280687]

Berg JM, Latzman RD, Bliwise NG, & Lilienfeld SO (2015). Parsing the heterogeneity of impulsivity: A meta-analytic review of the behavioral implications of the UPPS for psychopathology. Psychological Assessment, 27(4), 1129–1146. 10.1037/pas0000111 [PubMed: 25822833]

Carver CS, Johnson SL, & Joormann J (2008). Serotonergic function, two-mode models of self-regulation, and vulnerability to depression: What depression has in common with impulsive aggression. Psychological Bulletin, 134(6), 912–943. 10.1037/a0013740 [PubMed: 18954161]

Carver CS, Johnson SL, Joormann J, Kim Y, & Nam JY (2011). Serotonin transporter polymorphism interacts with childhood adversity to predict aspects of impulsivity. Psychological Science, 22(5), 589–595. 10.1177/0956797611404085 [PubMed: 21460340]

Carver CS, Johnson SL, & Timpano KR (2017). Toward a functional view of the p factor in psychopathology. Clinical Psychological Science, 5, 880–889. 10.1177/2167702617710037 [PubMed: 29057170]

Chester DS, Lynam DR, Milich R, Powell DK, Andersen AH, & De Wall CN (2016). How do negative emotions impair self-control? A neural model of negative urgency. NeuroImage, 132, 43–50. 10.1016/j.neuroimage.2016.02.024 [PubMed: 26892861]

Chiew KS, & Braver TS (2013). Temporal dynamics of motivation-cognitive control interactions revealed by high-resolution pupillometry. Frontiers in Psychology, 4 10.3389/fpsyg.2013.00015

Cyders MA, & Coskunpinar A (2010). Is urgency emotionality? Separating urgent behaviors from effects of emotional experiences. Personality and Individual Differences, 48(7), 839–844. 10.1016/j.paid.2010.02.009 [PubMed: 20514352]

Cyders MA, & Coskunpinar A (2011). Measurement of constructs using self-report and behavioral lab tasks: Is there overlap in nomothetic span and construct representation for impulsivity? Clinical Psychology Review, 31(6), 965–982. 10.1016/j.cpr.2011.06.001 [PubMed: 21733491]

Cyders MA, & Coskunpinar A (2012). The relationship between self-report and lab task conceptualizations of impulsivity. Journal of Research in Personality, 46(1), 121–124. 10.1016/j.jrp.2011.11.005

Cyders MA, Coskunpinar A, & Lehman ZA (2012). Difficulties and advancements in the assessment and induction of emotion- based impulsivity: Development of the three-task procedure In Cyders MA (Ed.), Psychology of impulsivity. Hauppauge, NY: Nova Science Publishers.

Cyders MA, & Smith GT (2007). Mood-based rash action and its components: Positive and negative urgency. Personality and Individual Differences, 43(4), 839–850. 10.1016/j.paid.2007.02.008

Cyders MA, Smith GT, Spillane NS, Fischer S, Annus AM, & Peterson C (2007). Integration of impulsivity and positive mood to predict risky behavior: Development and validation of a measure of positive urgency. Psychological Assessment, 19(1), 107–118. 10.1037/1040-3590.19.1.107 [PubMed: 17371126]

Dekker MR, & Johnson SL (2018). Major depressive disorder and emotion-related impulsivity: Are both related to cognitive inhibition? Cognitive Therapy and Research, 42(4), 298–407. 10.1007/s10608-017-9885-2

Glass DC, & Singer JE (1972). Behavioral aftereffects of unpredictable and uncontrollable aversive events: Although subjects were able to adapt to loud noise and other stressors in laboratory experiments, they clearly demonstrated adverse aftereffects. American Scientist, 60(4), 457–465. [PubMed: 5043170]

Gunn RL, & Finn PR (2015). Applying a dual process model of self-regulation: The association between executive working memory capacity, negative urgency, and negative mood induction on pre-potent response inhibition. Personality and Individual Differences, 75, 210–215. 10.1016/j.paid.2014.11.033 [PubMed: 25530648]

Hox J (2002). Multilevel analysis: Techniques and applications. Mahwah, NJ: Lawrence Erlbaum Associates.

Johnson SL, Carver CS, & Joormann J (2013). Impulsive responses to emotion as a transdiagnostic vulnerability to internalizing and externalizing symptoms. Journal of Affective Disorders, 150(3), 872–878. 10.1016/j.jad.2013.05.004 [PubMed: 23726781]

Johnson SL, Carver CS, Mulé S, & Joormann J (2013). Impulsivity and risk for mania: Towards greater specificity. Psychology and Psychotherapy: Theory, Research and Practice, 86(4), 401–412. 10.1111/j.2044-8341.2012.02078.x

Johnson EL, Miller Singley AT, Peckham AD, Johnson SL, & Bunge SA (2014). Task-evoked pupillometry provides a window into the development of short-term memory capacity. Frontiers in Psychology, 5, 1–8. 10.3389/fpsyg.2014.00218 [PubMed: 24474945]

Johnson SL, Tharp JA, Peckham AD, Sanchez AH, & Carver CS (2016). Positive urgency is related to difficulty inhibiting pre-potent responses. Emotion, 16(5), 750–759. 10.1007/s13398-014-0173-7.2 [PubMed: 27064288]

Joshi S, Li Y, Kalwani RM, & Gold JI (2016). Relationships between pupil diameter and neuronal activity in the locus coeruleus, colliculi, and cingulate cortex. Neuron, 89(1), 221–234. 10.1016/j.neuron.2015.11.028 [PubMed: 26711118]

Kaiser A, Bonsu JA, Charnigo RJ, Milich RA, & Lynam DR (2016). Impulsive personality and alcohol use. Journal of Studies on Alcohol and Drugs, 77(3), 475–482. 10.15288/jsad.2016.77.473

Kane MJ, Bleckley MK, Conway AR, & Engle RW (2001). A controlled-attention view of working-memory capacity. Journal of Experimental Psychology: General, 130(2), 169–183. 10.1037/0096-3445.130.2.169 [PubMed: 11409097]

Meese TS (2011). Basic vision: An introduction to visual perception. Oxford, UK: Oxford University Press.

Miyake A, & Friedman NP (2012). The nature and organization of individual differences in executive functions: Four general conclusions. Current Directions in Psychological Science, 21(1), 8–14. 10.1177/0963721411429458 [PubMed: 22773897]

Muhtadie L, Johnson SL, Carver CS, Gotlib IH, & Ketter TA (2014). A profile approach to impulsivity in bipolar disorder: The key role of strong emotions. Acta Psychiatrica Scandinavica, 129(2), 100–108. 10.1111/acps.12136 [PubMed: 23600731]

Pearson CM, Combs JL, Zapolski TC, & Smith GT (2012). A longitudinal transactional risk model for early eating disorder onset. Journal of Abnormal Psychology, 27(3), 320–331. 10.1002/nbm.3066

Pronin E (2013). When the mind races: Effects of thought speed on feeling and action. Current Directions in Psychological Science, 22(4), 283–288. 10.1177/0963721413482324

Ram N, & Gerstorf D (2009). Time-structured and net intraindividual variability: Tools for examining the development of dynamic characteristics and processes. Psychology and Aging, 24(4), 778–791. 10.1002/nbm.3066 [PubMed: 20025395]

Riley EN, Combs JL, Jordan CE, & Smith GT (2015). Negative urgency and lack of perseverance: Identification of differential pathways of onset and maintenance risk in the longitudinal prediction of nonsuicidal self-injury. Behavior Therapy, 46(4), 439–448. 10.1016/j.beth.2015.03.002 [PubMed: 26163709]

Roberts RJ, Hager LD, & Heron C (1994). Prefrontal cognitive processes: Working memory and inhibition in the antisaccade task. Journal of Experimental Psychology: General, 123(4), 374–393. 10.1037/0096-3445.123.4.374

Shields GS, Sazma MA, & Yonelinas AP (2016). The effects of acute stress on core executive functions: A meta-analysis and comparison with cortisol. Neuroscience and Biobehavioral Reviews, 68, 651–668. 10.1016/j.neubiorev.2016.06.038 [PubMed: 27371161]

Snowden RJ, O'Farrell KR, Burley D, Erichsen JT, Newton NV, & Gray NS (2016). The pupil's response to affective pictures: Role of image duration, habituation, and viewing mode. Psychophysiology, 53(8), 1217–1223. 10.1111/psyp.12668 [PubMed: 27172997]

Sperry SH, Lynam DPR, & Kwapil TR (2018). The convergence and divergence of impulsivity facets in daily life. Journal of Personality, 86, 841–852. 10.1111/jopy.12359 [PubMed: 29125631]

Steinhauer SR, & Hakerem G (1992). The pupillary response in cognitive psychophysiology and schizophrenia. Annals of the New York Academy of Sciences, 658(1), 182–204. 10.1111/j.1749-6632.1992.tb22845.x [PubMed: 1497258]

Tank AW, & Wong DL (2015). Peripheral and central effects of circulating catecholamines. Comprehensive Physiology, 5(1), 1–15. 10.1002/cphy.c140007 [PubMed: 25589262]

Victor SE, Johnson SL, & Gotlib IH (2011). Quality of life and impulsivity in bipolar disorder. Bipolar Disorders, 13(3), 303–309. 10.1111/j.1399-5618.2011.00919.x [PubMed: 21676133]

Whiteside SP, & Lynam DR (2001). The five factor model and impulsivity: Using a structural model of personality to understand impulsivity. Personality and Individual Differences, 30(4), 669–689. 10.1016/S0191-8869(00)00064-7

Whiteside SP, Lynam D, Miller J, & Reynolds S (2005). Validation of the UPPS Impulsive Behavior Scale: A four factor model of impulsivity. European Journal of Personality, 19, 559–574. 10.1002/per.556

Wilbertz T, Deserno L, Horstmann A, Neumann J, Villringer A, Heinze H-J, … Schlagenhauf F (2014). Response inhibition and its relation to multidimensional impulsivity. NeuroImage, 103, 241– 248. 10.1016/j.neuroimage.2014.09.021 [PubMed: 25241087]

Wright L, Lipszyc J, Dupuis A, Thayapararajah SW, & Schachar R (2014). Response inhibition and psychopathology: A meta-analysis of go/no-go task performance. Journal of Abnormal Psychology, 123(2), 428–439. 10.1037/a0036295

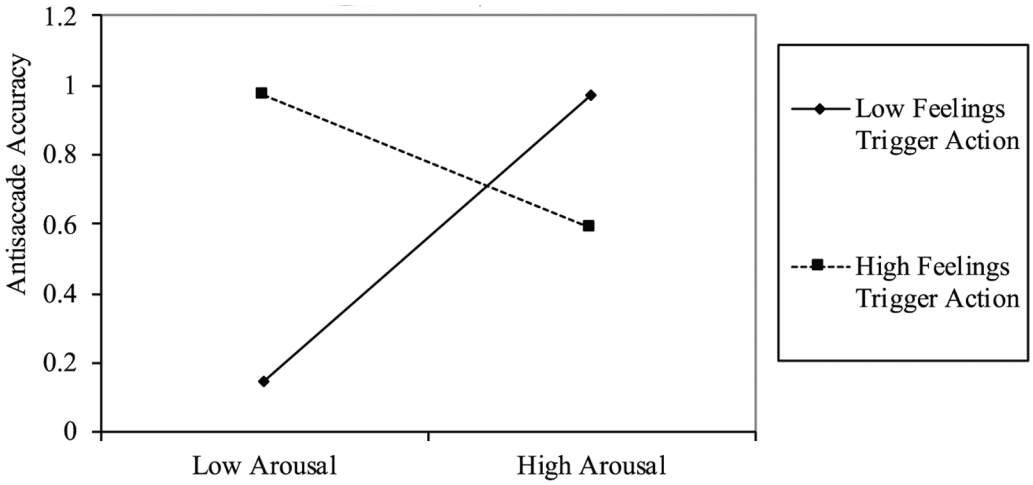

FIGURE 1.

Interaction between arousal, assessed as pupil dilation, and feelings trigger action predicting trial-level antisaccade accuracy

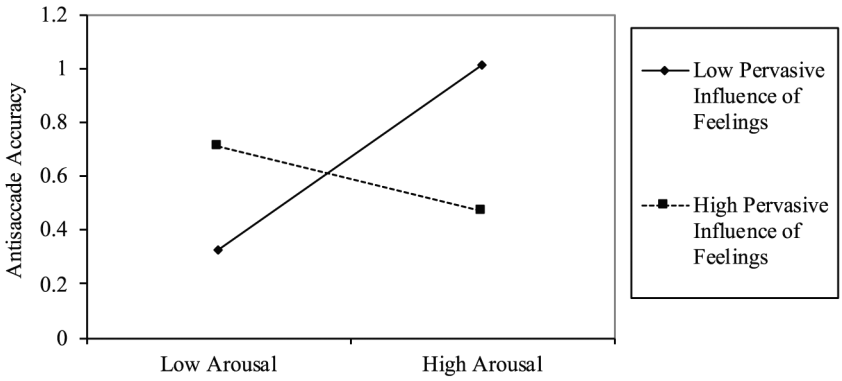

FIGURE 2.

Interaction between arousal, assessed as pupil dilation, and pervasive influence of feelings predicting trial-level antisaccade accuracy

TABLE 1

Correlations of impulsivity measures with antisaccade performance and pupil dilation

	Average antisaccade accuracy across trials[a]	Average pupil dilation across trials
Feelings trigger action	−0.04	−0.05
Pervasive influence of feelings	−0.11	−0.03
Lack of follow-through	0.03	−0.17

Note. No correlations were significant; (N = 85).

[a]Controlling for prosaccade performance.