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Publication Date

2022

Peer reviewed|Thesis/dissertation

Searching for the Process Linking Emotion-Related Impulsivity to Internalizing Symptoms:
Arousal-Induced Decays in Working Memory

By

Jennifer G. Pearlstein

A dissertation submitted in partial satisfaction of the

requirements for the degree of

Doctor of Philosophy

in

Psychology

in the

Graduate Division

of the

University of California Berkeley

Committee in charge:

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Summer 20222

Abstract

Searching for the Process Linking Emotion-Related Impulsivity to Internalizing Symptoms:
Arousal-Induced Decays in Working Memory

by

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Doctor of Philosophy in Psychology

University of California, Berkeley

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Emotion-related impulsivity, the trait-based tendency to respond impulsively to heightened emotions, is a transdiagnostic phenomenon associated with diverse forms of psychopathology and problematic behavior. Theory and research on the process of emotion-related impulsivity have identified deficits in cognitive control. Emotion-related impulsivity leads to problems during heightened emotions, which suggests that increased arousal may be core to problematic behavior. Arousal is also known to contribute to decays in some facets of cognitive control, especially working memory. A key need is to understand how arousal-induced decays in working memory relate to emotion-related impulsivity and whether arousal-induced decays in working memory mediate the relation between emotion-related impulsivity and internalizing symptoms.

Participants ($N = 100$) were recruited as part of a larger two-site study including people who experience a full range of internalizing and externalizing symptoms. Participants completed measures of internalizing symptoms, impulsivity, and potential confounds and then completed a novel N-back task including a within-task stressor to induce high arousal. Skin conductance and pupil diameter were collected at baseline and throughout the task to index arousal. Preliminary cleaning and analyses were conducted to exclude unusable data to yield a final sample ($N=82$).

Impulsivity was positively related to internalizing symptoms with significant large effects. Contrary to hypotheses, neither higher emotion-related impulsivity nor higher internalizing symptoms were related to poorer working memory performance, and neither interacted with arousal in relation to working memory performance. Arousal did not induce decays in working memory, and arousal-induced decay in working memory was not a putative mediator for the relationship between emotion-related impulsivity and internalizing symptoms.

Findings inform the emotional and neurocognitive processes involved in emotion-related impulsivity. Results did not support the role of arousal-induced decays in working memory in impulsivity or internalizing symptoms, however, future research is warranted to evaluate this model with other methods given the present findings contradict large and growing literatures on the role of arousal and cognition. Further pursuit of this model could identify the process underlying emotion-related impulsivity which may point to potential targets for intervention.

Searching for the Process Linking Emotion-Related Impulsivity to Internalizing Symptoms: Arousal-Induced Decays in Working Memory

The trait-based tendency to react in congruence with emotional impulses is referred to as emotion-related impulsivity. This trait is associated with diverse forms of psychopathology, indicating emotion-related impulsivity is a transdiagnostic correlate of psychopathology (Berg et al., 2015; Johnson, Carver, & Joormann, 2013). A meta-analysis of over 40,000 individuals found emotion-related impulsivity, when compared with other aspects of self-rated impulsivity (i.e., (lack of) Premeditation, (lack of) Perseverance, and Sensation Seeking), was the strongest predictor of every form of psychopathology studied, including depression, anxiety, eating disorders, borderline personality traits, suicidality, and non-suicidal self-injury (Berg et al., 2015). In clinical samples, this trait is related to lower quality of life, higher rates of comorbidity, self-injury, suicidality, aggression, and poor wellbeing (Auerbach et al., 2017; Muhtadie et al., 2014; Victor et al., 2011). Longitudinal research provides evidence that emotion-related impulsivity predicts the onset of eating disorders (Pearson et al., 2012), non-suicidal self-injury (Riley et al., 2015), gambling (Cyders & Smith, 2008), risky sexual behaviors (Zapolski et al., 2009), substance abuse (Kaiser et al., 2016; Smith & Cyders, 2016), and more severe depressive symptoms (Anestis et al., 2007). Emotion-related impulsivity appears to serve as a transdiagnostic vulnerability factor for internalizing, externalizing, and psychotic disorders (Carver et al., 2017). Given the robust cross-sectional and longitudinal evidence linking emotion-related impulsivity to psychopathology, an important next step is to identify the process by which emotion-related impulsivity leads to psychopathology.

Central to the definition of emotion-related impulsivity is the notion that impulsive responses are triggered by heightened emotion states. Although early work on emotion-related impulsivity characterized a factor defined as Negative Urgency, the tendency to act impulsively in response to *negative* emotion (Whiteside et al., 2005; Whiteside & Lynam, 2001), later work identified the additional factor of Positive Urgency, the tendency to act impulsively in response to *positive* emotion (Cyders & Smith, 2007). Substantial analytic evidence suggests that these two factors are very highly correlated and may form one higher-order construct of emotion-related impulsivity (Billieux et al., 2021; Carver et al., 2011; Cyders & Smith, 2007; Sperry et al., 2018). Consistent with the theory that heightened emotions trigger impulsive behaviors for people high in emotion-related impulsivity, laboratory research has verified increased risky decision-making, including drinking (VanderVeen et al., 2016) and binge-eating (Becker et al., 2016) during heightened emotion states for those high in emotion-related impulsivity as compared to those lower int his trait. Conversely, studies using ecological momentary assessment methods often fail to find increases in daily impulsivity during heightened emotions for those higher in emotion-related impulsivity (Sharpe et al., 2020; Sperry et al., 2016, 2018).

Affective science indicates core features of emotion states that may be important for understanding the process by which emotion-related impulsivity triggers risky decision-making. The widely used and empirically-supported circumplex model of affect (Russell, 2003) proposes that all affective states arise from the product of two properties: valence (emotions ranging from pleasant/positive to unpleasant/negative) and arousal (emotions ranging from quiet/still to active/energized). These properties can be plotted on a circumplex, with valence serving as the x-axis and arousal serving as the y-axis. Because both positive and negative emotions can trigger this form of impulsivity and its cognitive correlates (e.g. Dekker & Johnson, 2018; Pearlstein et

al., 2019), heightened arousal, rather than valence, may trigger impulsive behaviors for those high in emotion-related impulsivity.

With the aim to examine arousal-induced impulsive action, it is important to separate emotion-related impulsivity from general emotional reactivity. Indeed, a growing body of work distinguishes emotion-related impulsivity from high emotional reactivity. That is, mounting evidence indicates that emotion-related impulsivity is not related to increased reactivity to emotional stimuli, whether using film clips, failure, or reward to elicit responses, and across channels including subjective affect, psychophysiology, and facial affective behavior (Cyders & Coskunpinar, 2010; Johnson et al., 2016; Pearlstein et al., 2019). Furthermore, emotion-related impulsivity predicts symptoms of psychopathology after controlling for negative emotionality (Carver & Johnson, 2018; King et al., 2021). Taken together, emotion-related impulsivity does not appear to simply reflect emotional reactivity.

The Role of Cognitive Control in Emotion-Related Impulsivity

Deficits in cognitive control, which corresponds to the ability to apply resources flexibly towards goal-directed behavior, has been repeatedly associated with impulsivity (Bickel et al., 2012; Sharma et al., 2014). Cognitive control encompasses at least three distinct dimensions: updating, shifting, and inhibiting (Miyake et al., 2000). To date, impulsivity has been associated primarily with updating, encompassing working memory, and inhibiting.

Response inhibition, or the ability to override a prepotent response tendency, has been identified as an intuitive potential driver of impulsivity. Indeed, response inhibition tasks are often used as a laboratory analogue for impulsive behavior (for review, see Bari & Robbins, 2013; Cyders & Coskunpinar, 2011; Sharma et al., 2014). Whereas there is meta-analytic evidence for the associations between response inhibition and emotion-related impulsivity, caution is warranted because effect sizes are fairly small, particularly in nonclinical samples (Johnson et al., 2016). One potential explanation for the small effect sizes could be the failure to test response inhibition during heightened emotion states. Recent studies have considered the effect of affect inductions on response inhibition performance. Multiple fMRI studies have reported differential patterns of activation during response inhibition tasks for those high in emotion-related impulsivity during affective but not neutral states (Johnson et al., 2020). Behavioral effects have also emerged in the context of negative stimuli (Allen et al., 2018; Allen et al., 2021). In another study inducing positive affect, increased arousal led to decays in response inhibition performance only for persons high in emotion-related impulsivity (Pearlstein et al., 2019). Across studies, response inhibition appears to be moderately associated with emotion-related impulsivity and is subject to some differential effects in the face of emotion.

Alongside research associating emotion-related impulsivity with response inhibition, researchers have also considered the role of working memory, defined as the short-term maintenance of processed information (Baddeley, 2003). Working memory has a central role in two domains critically tied to both emotion-related impulsivity and psychopathology: self-control (Diamond, 2013) and emotion regulation (Schmeichel et al., 2008). Monitoring and revising goal-relevant information, especially in the presence of other goals or distractions, requires working memory (Miyake & Friedman, 2012). Therefore, the ability to override emotional impulses may be related to the ability to maintain and update information stored in working memory. Empirical literature documents working memory deficits correlate with multiple facets of impulsivity, including risk-taking and impulsive decision-making (Endres et al., 2011; Finn et al., 2002, 2015; Wesley & Bickel, 2014). The links between working memory and emotion-related impulsivity are not well established; some studies do not find associations

between working memory and emotion-related impulsivity (e.g. Lozano, 2015), whereas another study found that emotion-related impulsivity (i.e. urgency) was related to poor working memory capacity (Finn et al., 2015). More recent research has reported poorer working memory performance for those high in urgency as compared to those lower in the trait when distractors are present (Canale et al., 2019).

Working Memory is Important Transdiagnostically

Working memory deficits have been associated with diverse forms of psychopathology including anxiety (Moran, 2016), depression (Christopher & MacDonald, 2005), substance use (Bechara & Martin, 2004), antisocial personality (Bogg & Finn, 2010; Endres et al., 2011), psychotic disorders (Frydecka et al., 2014), among others. Further, decreased working memory performance is tied to key neural regions associated with psychopathology, including the frontoparietal network and the cingulo-opercular network (Etkin et al., 2013). Poor working memory performance precedes symptom onset and has been characterized as a vulnerability factor for psychopathology (Giakoumaki et al., 2011; Moran, 2016). Meta-analytic evidence supports the associations between working memory and internalizing symptoms, including anxiety (Moran, 2016), depression (Nikolin et al., 2021), and OCD (Abramovitch et al., 2013).

Working Memory is Sensitive to Changes in Emotion State

Because emotion-related impulsivity emerges in the context of heightened positive and negative emotions, theory should include a central role for shifts in emotional arousal in relation to neurocognitive processes. From this point onward, arousal will refer to the internal state that is triggered by stress, and this internal shift in arousal can lead to changes in cognition (see Figure 1). The neurocognitive process most sensitive to arousal-related decays has the potential to be central to the process of emotion-related impulsivity. A meta-analysis of over 50 studies compared the effects of stress on the three domains of cognitive control (shifting/cognitive flexibility, updating/working memory, and inhibiting/response inhibition); the meta-analytic findings suggested stress leads to decays in working memory performance, but not to decays in response inhibition (Shields et al., 2016).

Arousal has long been observed to impact cognitive performance (e.g. Eysenck, 2012; Hasher & Zacks, 1979; Karatekin et al., 2004; Yerkes & Dodson, 1908), though more recent neurobiological evidence sheds light on why working memory may be especially sensitive to stress. Neuroimaging findings have shown that activation of emotional processing regions (i.e., amygdala and ventrolateral prefrontal cortex) interferes with working memory performance (Dolcos & McCarthy, 2006), whereas in some studies, comparable effects have not been found for response inhibition (e.g., Brown et al., 2015). Acute cortisol administration improves response inhibition performance and impairs working memory (Shields et al., 2015). However, cortisol response alone does not explain decay in cognitive control, which suggests that additional pathways beyond cortisol are implicated in the process by which stress effects working memory (Shields et al., 2016). Beyond eliciting cortisol, stress is also known to influence sex hormone levels (Lennartsson et al., 2012), circulating inflammatory cytokines (Dhabhar et al., 2012), DHEA and catecholamines like norepinephrine (Arnsten, 2015; Lennartsson et al., 2012), and some of these have known effects on cognitive control. Both inflammatory (Marsland et al., 2017) and catecholaminergic mechanisms (Arnsten & Goldman-Rakic, 2003) have been associated with decreased working memory performance.

Several findings suggest that arousal-induced changes in working memory performance are a more important correlate of psychopathology than are deficits in working memory alone. Deficits in arousal-induced working memory, as compared to baseline working memory, have

been shown to be more strongly related to various internalizing disorders, including anxiety (Figueira et al., 2017), PTSD (Schweizer & Dalgleish, 2011; Zhang et al., 2013), and depression (Quinn & Joormann, 2015). Given the associations between stress reactivity, working memory and diverse forms of psychopathology, the planned project aims to assess stress-induced decays in working memory transdiagnostically.

The Present Investigation of Arousal-Induced Decays in Working Memory

Despite the evidence for robust links of psychopathology with arousal-induced working memory deficits, less is known about the underlying neurobiological process driving these effects. This study builds on human and animal literature that indicates a central role of noradrenergic activity in working memory decays (Chamberlain et al., 2006; Schoofs et al., 2008).

Two psychophysiological indices of arousal were used in the present investigation: skin conductance and pupil dilation. Skin conductance provides a continuous measure of autonomic arousal. Measuring skin conductance allowed for (a) a manipulation check to ensure the stress condition effectively elicited autonomic arousal; and (b) a test of the hypothesis that arousal predicts decays in working memory performance. Galvanic skin responses occur within 1-5 seconds of a stimulus (Dawson et al., 2016), which enabled the comparison of block level peaks in arousal but not trial by trial fluctuations. In contrast, pupil dilation provides a rapid marker of arousal (Beatty & Lucero-Wagoner, 2000). The pupil serves as a window into the autonomic nervous system: dilation reflects sympathetic activity and constriction reflects parasympathetic activity (Beatty & Lucero-Wagoner, 2000). Task-evoked pupil dilation corresponds to fluctuations in activity in the locus coeruleus, a neural region responsible for synthesis of norepinephrine (Joshi et al., 2016). Pupils dilate and constrict rapidly in response to stimuli so as to reach a peak within a second of stimulus onset, allowing for analysis of trial-level fluctuations in arousal . Pupil dilation is a rapid marker of arousal, enabling dynamic time-dependent analyses, which makes pupillometry a well-suited index of arousal to understand associations with cognitive performance and psychopathology. Pupil dilation provides a way to test the model that stress-induced norepinephrine release impacts working memory performance. Whereas skin conductance provided a stable, slower responding index of arousal to the stressor, pupil dilation provided a rapidly responding index of the temporal dynamics of arousal from one trial to the next.

In accordance with the Yerkes-Dodson law, which suggests arousal has a curvilinear relationship with cognitive performance (Yerkes & Dodson, 1908), moderate increases in task-evoked pupil dilation have been associated with improved cognitive performance (Rondeel et al., 2015). Emerging research has also validated the use of pupillometry for studying individual differences in psychopathology; people diagnosed with depression, compared to controls, display sustained pupil dilation in response to emotional stimuli (Siegle et al., 2003). This study extended these lines of inquiry by using pupillometry to assess arousal-related decays in cognitive performance and relation to psychopathology.

Goals and Hypotheses

The goal of this study was to integrate well-established factors into a model of how emotion-related impulsivity may be tied to decays in working memory in the context of arousal, which then contribute to internalizing symptoms. In this study, I conducted the first examination of whether emotion-related impulsivity is tied to arousal-induced decays in working memory. I also examined a novel model for how arousal-induced decay in working memory relates to emotion-related impulsivity and internalizing symptoms. This study included an innovative

version of the N-back working memory task to allow for a continuous stress induction and the examination of dynamic analyses to assess trial-by-trial changes in arousal using pupillometry, a rapid marker of noradrenergic activity. Data was gathered as part of a larger grant, which recruited a sample representing a wide range of internalizing and externalizing psychopathology. The hypotheses of the study were as follows. First, self-rated emotion-related impulsivity will relate to greater arousal-induced decays in working memory, measured by trial-level effects of arousal (pupil dilation) on N-back accuracy. Second, internalizing symptoms, as measured by a latent variable for self-report scales, will relate to greater arousal-induced decays in working memory, measured by trial-level effects of arousal on N-back accuracy. Third, the relationship between emotion-related impulsivity and internalizing symptoms will be accounted for by arousal-induced decays in working memory, the statistical mediator.

Method

Participants

Participants were recruited as part of a parent project in metropolitan regions of California and Florida through clinical referrals and print and online advertisements sent to support groups, online forums, and community clinics for populations known to have difficulties with emotion-related impulsivity, including mood and anxiety disorders and substance use disorders. Interested individuals were asked to provide consent, and then to complete an initial self-report eligibility questionnaire online, which assessed age, diagnosis of schizophrenia or other primary psychotic disorder, daily marijuana use, and current alcohol or substance use problems. Participants meeting these initial eligibility criteria were contacted to hear a brief project overview and complete additional screening by phone to address preliminary inclusion and exclusion criteria.

Inclusion criteria included receiving or seeking mental health treatment; sufficient functional impairment, defined as at least moderate (rating=5) impairment in at least one life domain as assessed by the Sheehan Disability Scale. Exclusion criteria included: head injury with loss of consciousness more than 5 min and/or with lasting effects, low cognitive abilities operationalized as an Orientation Memory Concentration test (Katzman et al., 1983) score of 7 or lower, presence of neurological disorders, daily antipsychotic medication or recreational drug use, use of sedating medications on the day of study completion, inability to attend the in-person laboratory sessions; lack of proficiency in English, or inability to read and comprehend the Informed Consent form, or medical conditions or medications that could interfere with diagnostic assessment (e.g., untreated endocrine disorders, HIV, syphilis, and past-year electroconvulsive treatment). A subset of participants was screened for inclusion in MRI sessions (e.g., ferrous metal that cannot be removed from the body, pregnancy, high risk for seizures). Potential participants completed a diagnostic interview to identify exclusion criteria (alcohol or substance use disorders in the past 6 months, lifetime psychotic disorders, or lifetime manic episode). There were no restrictions on race, ethnicity, or gender.

Participants ($N = 100$) were invited to participate in a laboratory session at the University of California Berkeley or the University of Miami. Most sessions occurred at the University of California, Berkeley ($n = 66$, 65%). Two participants chose to stop participating in the task; 11 participants' data was not collected properly due to technological errors (5 errors with behavioral data collection, 6 with eye-tracking acquisition errors); 3 participants' data did not have sufficient valid pupil data (>50% after removing blinks) and wore eyeglasses, 1 participant did not complete self-report measures, and 1 participant failed to follow instructions. The self-report

surveys included attention check items (e.g., '*Please answer "Agree" to this item*') and participants were required to accurately answer 50% of attention check items to be included. The sample included in analyses ($n = 82$) was 64.6% cis-gendered female (31.7% male, 3.7% non-binary/trans), $M_{age} = 29.87$, $SD_{age} = 10.32$). Participants reported their race as 22.0% Asian, 13.4% Black/African American, 1.2% Native Hawaiian/Pacific Islander, 13.4% Other/multiple races, 47.6% White (2.4% chose to decline to answer), and their ethnicities as 29.3% Hispanic/ Latino.

Procedures

All materials and procedures were approved by the Institutional Review Boards at respective universities before data collection commenced. Data collection occurred at a stand-alone session ($n = 71$) for participants who had already completed the parent project or after neuro-imaging sessions as part of the parent project ($n = 11$). For the latter, participants were given a break including a walk between buildings prior to participating in the present study. The present laboratory session lasted one hour and included symptom severity questionnaires, baseline psychophysiology, and the modified N-back task. The modified N-back paradigm included a stressor block of trials in which participants received socially evaluative feedback in the form of noxious noise. At the start of the session, participants completed current self-report measures and/or relaxed for 10 minutes before skin conductance sensors were applied and baseline skin conductance and pupil diameter were measured for 5 minutes pre-task while the participant sat quietly, then the N-back task was administered while pupil diameter was measured. Participants were debriefed at the end of the study session.

Measures

Diagnostic interviewing was conducted at an earlier study session to assess inclusion and exclusion criteria. Self-rated questionnaires were used to measure emotion-related and general impulsivity as well as broad internalizing symptoms of anhedonia, depression, generalized anxiety disorder (GAD), social anxiety disorder, and obsessive-compulsive disorder (OCD), which are the most consistent syndromes to emerge in modeling internalizing symptoms (e.g., misery/distress models; Watson et al 1995). For the purposes of this project, a single internalizing latent variable was modeled to use in subsequent models. Additional measures, the Risky Families and Externalizing Symptom Inventory were included as potential control variables and for exploratory analyses.

Structured Clinical Interview for DSM-V (SCID-V). The SCID-V ((First et al., 2015) is a semi-structured diagnostic interview used to assess Axis I diagnoses based on the DSM-V and was used to assess inclusion and exclusion. Trained interviewers completed didactic and interactive training and showed adequate inter-rater reliability before using the SCID-5 with participants. The study team conducted regular multi-site reliability meetings to protect against rater digression. The average kappa measured between the rater and the gold standard across raters was 0.82.

Sheehan Disability Scale. The Sheehan Disability Scale is a three-item interview developed to assess functional impairment (Sheehan, 1986). This scale was used to assess study inclusion criteria, operationally defined as a score of moderate functional impairment (rating of 5 or higher) in at least one life domain. Potential participants were asked to rate three domains (work/schoolwork, social life/leisure activities, and family life/home responsibilities) on a 0 (not at all) to 10 (extremely) scale. Potential participants were instructed to respond based on the worst month in the past six months for impairment due to mental health symptoms.

Three-Factor Impulsivity Index. The Three-Factor Impulsivity Index was derived from multiple scales and novel items measuring heterogeneous forms of lack of constraint over emotion, motivation, and impulses (Carver et al., 2011). The measure consists of three factor-analytically derived subscales, including two factors covering emotion-related impulsivity (Feelings Trigger Action and Pervasive Influence of Feelings) and a third factor of items covering impulsiveness without reference to emotion (Lack of Follow Through). Feelings Trigger Action covers the tendencies to engage in regrettable speech or action in response to positive or negative 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"). Pervasive Influence of Feelings refers to excessively broad impacts of (mostly negative) emotion on cognition (e.g., "My feelings greatly affect how I see the world") and motivation ("When I feel sad, it paralyzes me"). Lack of Follow Through refers to impulsiveness interfering with the completion of intended actions (e.g., "I am easily distracted by stray thoughts"). The emotion-related impulsivity factors have been shown to relate to early adversity, as well as symptoms of depression, anxiety, mania, externalizing disorders, and suicidality (Johnson, Carver, Mulé, et al., 2013). Lack of Follow Through is included to enable tests of discriminant validity of emotion-related as compared to non-emotion-related impulsivity. In this sample, the three subscales had high internal consistency reliability (Feelings Trigger Action: $\alpha = 0.91$; Pervasive Influence of Feelings: $\alpha = 0.86$; Lack of Follow-Through: $\alpha = 0.92$).

Mood and Anxiety Questionnaire – Short Form (MASQ). The MASQ - Short Form is a 62-item measure designed to assess depressive and anxiety symptoms in accordance with the tripartite model of anxiety and depression (Watson & Clark, 1991). Participants were asked to report severity of each symptom in the past two weeks (1 = not at all; 5 = extremely). The MASQ includes four symptom scales: (a) Anxious Arousal, measuring somatic tension and arousal (17 items; e.g., "was trembling or shaking,"); (b) Anhedonic Depression, measuring decreased interest, pleasure, and positive affect (22 items; e.g., "felt like nothing was very enjoyable"), (c) General Distress, which is comprised of General Distress-Anxiety, measuring nervousness and other nonspecific symptoms of anxiety (11 items; e.g., "felt uneasy"); and General Distress-Depression, measuring depressed/sad mood other nonspecific symptoms of depression (12 items; e.g., "felt sad,"). In this sample, internal alpha reliability across subscales was .85 with subscale scores ranging .81-.93. Because internal consistency within and between subscales was high and because modeling depended on the internalizing latent variable, with anxiety and depressive symptoms analyzed together, the subscales were combined and averaged into a total score for data reduction.

The Penn State Worry Questionnaire (PSWQ). The PSWQ is a 16-item measure used to assess pathological tendencies toward GAD (Meyer et al., 1990). The PSWQ was designed to assess the diffuse, excessive, and uncontrollable dimensions of pathological worry. Items are rated on a Likert scale (1 = not at all typical of me, 5 = very typical of me), and assess worry and anxiety (e.g., "My worries overwhelm me"). The PSWQ has been widely used and has been shown to discriminate GAD from other forms of anxiety (Fresco et al., 2003). The PSWQ demonstrated strong internal reliability as indicated by alpha = .95 in this sample.

Dimensional Obsessive-Compulsive Scale (DOCS). The DOCS is a 20-item measure used to assess OCD symptoms in the past month (Abramowitz et al., 2010). The DOCS contains four factor-analytically derived subscales that measure the common domains of OCD: (a) contamination, (b) responsibility for harm and mistakes, (c) incompleteness/symmetry, and (d) unacceptable (taboo) thoughts. Participants are asked to rate five items within each of these

subscales to determine (a) time occupied by obsessions and compulsions, (b) avoidance behavior, (c) associated distress, (d) functional interference, and (e) difficulty dis- regarding the obsessions and refraining from the compulsions. The DOCS has strong psychometric properties, including high internal consistency, sensitivity, and specificity (Thibodeau et al., 2015). In this sample, internal alpha reliability across subscales was .93 with subscale scores ranging .88-.94.

Social Interaction Anxiety Scale (SIAS). The SIAS is a 20-item measure that uses self-statements (e.g. "I get nervous if I have to speak with someone in authority (teacher, boss, etc.,).") to capture social anxiety in social situations such as interacting in dyads or groups (Mattick and Clarke, 1998). Items are rated on a 5-point scale from "not at all characteristic of me" to "extremely characteristic of me." The total score is a sum of all items after reverse scoring three positively worded items. The SIAS has been shown to correlate highly with other well-established measures of social anxiety (e.g. Heimberg et al., 1992; Mattick and Clarke, 1998) and has strong psychometric support, including high test-retest reliability, internal consistency, and discriminant validity across patient and control groups (Herbert et al., 2014). In this sample, internal alpha reliability was .94.

Externalizing Symptoms. Exploratory analyses consider the role of externalizing symptoms using the 160-item brief version of the Externalizing Spectrum Inventory (ESI; Patrick et al., 2013), a multidimensional measure of externalizing problems. Participants rate items as true, somewhat true, somewhat false, or false (e.g., "I enjoy pushing people around sometimes" and "I have stolen something out of a vehicle"). The ESI was developed based on genetic and structural-quantitative models of overlap among the externalizing syndromes and has been widely used in community and inmate populations (Venables & Patrick, 2012). The brief ESI captures tendencies toward a broad range of externalizing conditions, including psychopathic symptoms (e.g., fraud, theft, rule-breaking), drug and alcohol problems, sensation-seeking, and several types of aggression. The Disinhibition subscale was excluded due to the overlap with the impulsivity measure. In this sample, internal reliability was high across subscales (.80-.90). For data reduction in exploratory modeling, the ESI subscales were combined into a total score, which maintained high internal consistency (.90).

Risky Families. The Risky Families is a 13-item self-report measure of childhood adversity and stress (Taylor et al., 2004). Participants are asked to rate physical, mental, and emotional distress from the ages 5-15 (e.g., was verbally abused; was physically abused; observed quarreling or shouting between parents) on a Likert scale from 1 (not at all) to 5 (very often). The Risky Families demonstrate strong convergent validity with interview-based measures (Taylor et al., 2004). The Risky Families was included to as a potential control and to conduct exploratory analyses.

Working Memory Task. A modified version of the widely used, reliable, and well-validated N-back task was used to measure working memory performance (Chatham et al., 2011; Miyake & Friedman, 2012). Variations of the N-back task have been used across forms of psychopathology, such as PTSD (Schoofs et al., 2008) and depression (Quinn & Joormann, 2015). The N-back task has been shown to load well (e.g., .67) on the updating/working memory dimension of cognitive control (Miyake & Friedman, 2012) and is one of the most widely used working memory tasks in neuroimaging studies, with findings consistently pointing towards the activation of the frontoparietal system (Owen et al., 2005). The n-back has been shown to have adequate to high test-retest reliability and outperforms several other common executive functioning tasks (Soveri et al., 2018).

During the task, participants were presented with trials consisting of a fixation and one letter (stimulus) and were asked to indicate as quickly and accurately as possible whether the current stimulus matches a stimulus presented a number (n) of trials previously. This task requires working memory to maintain, update and organize task stimuli. Stimuli were presented using E-Prime Extensions for Tobii (Psychology Software Tools, Pittsburgh, PA). Each trial stimulus was presented for 500 milliseconds followed by a 1500 millisecond fixation screen, which means the tonic measure of pupillary response was a duration of 2000 ms per trial. This duration was selected to capture the full task-evoked pupillary response (e.g.Beatty, 1982). Stimuli were presented in white on a black screen and display brightness was standardized across trials and sites.

Before the N-Back task, a 30-second baseline pupil measurement was collected as well as a baseline sound segment to characterize pupillary response to the two sound stimuli across volumes. During these segments, participants viewed a blank black screen and heard the sounds included during the arousal manipulation across three volumes (none, low, high; standardized across sites). Participants then received instructions, completed practice trials, and completed the N-Back task. The N-Back task included three blocks of 2-back trials: a standard block (40 trials), a stressor block (100 trials, described below), and then a final recovery block (40 trials). In clinical samples, average rates of performance on the 2-back are around 75% (e.g. Sandström et al., 2012)). Participants received one point for each correct trial, such that analyses can examine trial-level performance, or overall accuracy (average performance across trials).

The psychometric properties of the working memory task were assessed. Internal consistency was calculated using a permutation approach based on a large number of random (without replacement) split halves of the data. For each permutation, the correlation between halves was calculated, with the Spearman-Brown correction applied (Spearman & Spearman, 1904). Randomly identified halves yielded strong internal consistency estimates ($r_{SB} = 0.95$, 95%CI [0.93,0.97]).

Arousal Induction. To induce arousal, the modified N-back procedure included a stressor block including socially evaluative noxious noise. Socially evaluative feedback reliably elicits distress (Vytal et al., 2016), and was provided in the form of a noxious buzzer noise during the stressor block. Noxious noise as a punishment on cognitive tasks has been used to elicit subjective and psychophysiological indicators of distress in humans (Heslegrave & Furedy, 1979), and has been shown to impact cognitive performance in animals (Arnsten & Goldman-Rakic, 2003). Because pupillometry is collected throughout the task, noxious noise was chosen as to not provide a visually or verbally distracting stimulus that would directly confound pupil measurements or working memory performance. Before the stressor block, participants were given additional instructions. More specifically, they were told that during this block of trials, an evaluator would closely monitor their behavior and that their performance will be compared to study participants who had already completed this task. Participants were informed that a "ding" signals strong performance and a buzzer signals poor performance compared to other participants who previously completed the study. Participants were told feedback was provided in order to help them improve their performance, including their speed and accuracy. Participants were also informed that the feedback may at times be delayed. All participants received pseudorandomized feedback: a "ding" on 5 of the of 100 trials and the noxious buzzer on 20 of the 100 trials, and the volume of the sound stimuli was standardized. After the stressor block and before the final task block, participants were given the instructions to try the task again without feedback.

Psychophysiological

Skin Conductance. As an index of sympathetic activity, skin conductance responses were acquired on the University of California, Berkeley subset of participants using 8-channel chassis BioLab acquisition software version 2.5 (Mindware Technologies, Gahanna, OH) at 10000 Hz through two snap electrodes attached to the non-dominant palm. Participants were given a nonabrasive soap to use and were instructed to hydrate and wash their hands. And ambient temperature and humidity was standardized and tracked to facilitate data collection and reduce confounds (Dawson et al., 2016). Before collecting baseline data, the proper placement of sensors were tested by visually inspecting the data while participants took a deep breath and responded to a loud clap. Skin conductance was calculated using the Mindware Technologies Ltd. EDA module to analyze and inspect the data in 1-minute epochs. Phasic skin conductance, known as skin conductance responses (SCRs) were collected as a manipulation check during the stressor block. The amplitude of SCRs were evaluated when changes >.05 microSeimen were detected. The total number of SCRs was calculated for each task segment. Tonic skin conductance, known as skin conductance levels (SCLs), were also considered across the baseline period and each task block to verify successful manipulation of arousal. SCL below 1 microSeimen, which is below the expected range of SCL values, reflects poor electrode placement or dry hands and was coded as missing for 1 participant. Tonic SCL was averaged across time for each task block. Of the 54 participants with skin conductance collected during their session, 8 had issues with data acquisition (e.g. software crashing). The subsample with usable skin conductance data was 43.

Eye-tracking. Pupil dilation was measured using an infrared eye tracker (T-120 Tobii Technologies, Dandyred, Sweden). Pupil dilation was recorded at 60 Hz. Participants viewed stimuli on a 17-inch computer monitor with 1280×1024 screen resolution. Participants used a headrest to minimize possible motion throughout acquisition. A nine-point calibration was conducted before acquiring data. The camera simultaneously recorded pupil diameter for both eyes.

Pupil data was cleaned by removing blinks and artifacts (procedures abased on Granholm, Asarnow, Sarkin, & Dykes, 1996). Data were identified as blinks when large changes in pupil dilation occurred too rapidly to signify actual dilation or contraction (Mathôt, 2013). Specifically, data was identified as a blink when pupil dilation velocity exceeded a threshold, in this case, one-millimeter pupil diameter change per one millisecond. Filtering was applied to remove relative outliers. More specifically, a local fit regression equation identified values more than 5 standard errors away from the locally-defined weighted mean and removed those values from the dataset, as described in Johnson et al., 2014. This process was applied separately to both eyes, which then was aggregated to provide a single pupil diameter measure for each time point. Pupil data for one eye was used when both eyes were not available. Data were excluded if less than 50% was valid after cleaning (n = 2).

Data Analysis

All analyses were pre-registered (https://osf.io/95ezp) and conducted using R version 4.0.2 (R Foundation for Statistical Computing, Vienna Austria). Reliability estimates for the working memory task were estimated using the splithalf package (Parsons, 2020). Latent variable construction was completed using the lavaan package (Yves Rosseel, 2012). Generalized linear mixed effect models were completed using the lme4 package (Bates et al., 2015) using restricted maximum likelihood (REML),). Significance values were estimated with the lmerTest package (Kuznetsova & Brockhoff, 2017). Bootstrapping was conducted for linear models using the lmeresampler package (Carpenter et al., 2003). Simple slopes for moderator

values one standard deviation above and below the mean, marginal effect plots, and Johnson-Neyman intervals were reviewed for significant mixed effect models (Bauer et al., 2005) and moderation was examined using the interActive tool (McCabe et al., 2018). The mediation model was computed using the mediation package (Tingley et al., 2014). All beta (ß) values were standardized.

Preliminary Analyses. Before conducting analyses, all variables were graphed and assessed for normality and potential control variables were examined. Graphs were visually inspected and values of skew and kurtosis were examined (Klein, 2011). Potential confounds were considered in relation to key study variables using t-tests and correlations. Because data collection occurred before and after the COVID-19 pandemic, date of participation was considered as a confound in relation to arousal and working memory performance.

Internalizing Symptom Latent Variable. The self-rated measures of internalizing symptoms, the MASQ, PSWQ, SIAS, and DOCS were standardized through mean-centering (i.e., transformed into a z-score), and then entered into a structural equation model to create a single latent variable for internalizing symptoms to be used in subsequent analyses. Considerable research suggests that these variables consistently form a single latent variable (e.g. misery/distress models; . To assess model fit, the chi-square (x^2) statistic (which is affected by sample size and therefore not emphasized), two absolute fit indices, and one relative fit index were examined. For absolute fit indices, we used the root mean square error of approximation (RMSEA) with < .08 as an indicator of fit (Browne & Cudeck, 1993) and the Standardized Root Mean Square Residual (SRMR) with < .08 as an indicator of fit (Hu and Bentler, 1999). For a relative fit index, we used the Comparative Fit Index (CFI) with a cut-off value of > .95 (Hu & Bentler, 1999). We did not use other relative fit indices, as CFI is highly correlated with the commonly used Tucker Lewis and Non-normed Fit indices (Kenny, 2015).

Individualized Estimate for Arousal-Induced Decays in Working Memory. The proposed mediation model relies on arousal-induced decays in working memory as the mediator (M). To reduce data for mediation modeling, an effect size for each individual participant was computed to quantify an individualized effect size (R^2) for the strength of the relationship between arousal (pupil dilation) and working memory (N-back). If not all effects are in the same direction, standardized ß will be used to preserve directionality.

Arousal Manipulation. The effectiveness of the stressor for eliciting arousal was measured by assessing multiple psychophysiological indicators innervated by the autonomic nervous system. Skin conductance (total SCRs and tonic SCL) and pupil dilation were compared across the baseline period and three blocks of the N-back task.

Primary Analyses. Statistical assumptions of bivariate normalcy were considered. Linearity was evaluated through visual inspection of the data as well as correlations between variables. All models implement bootstrapping procedures (1000 iterations unless otherwise specified). For models including trial-level pupil dilation and working memory accuracy, pupil data was lagged by one observation such that prior trial pupil dilation was used to predict subsequent trial performance. This time-lagged approach enables the assessment of dynamic temporal processes (Chiew & Braver, 2013; Ram & Gerstorf, 2009). This procedure was used to examine how arousal, as measured by pupil dilation, impacts subsequent working memory performance.

To examine hypothesis 1, a generalized linear mixed-effect model regressed the trial-level working memory accuracy on emotion-related impulsivity, prior trial pupil dilation, and the

interaction of motion-related impulsivity x prior trial pupil dilation. Hypothesis 1 support would be demonstrated by a significant interaction effect.

To examine hypothesis 2, a generalized linear mixed-effect model regressed the trial-level working memory accuracy on internalizing symptoms, prior trial pupil dilation, and the interaction of internalizing symptoms by prior trial pupil dilation. Hypothesis 2 support would be demonstrated by a significant interaction effect.

Hypothesis 3 requires a mediation model to determine whether individualized estimates of arousal-induced decays in working memory (the mediator: M) statistically mediate the relationship between emotion-related impulsivity (X) and internalizing symptoms (Y). This mediation model requires significant relations between X and M. The direct effect of X on Y (c') represents the difference in Y between two cases that differ by one unit on X and that are equal on M. The indirect effect of X on Y through M (ab) represents the product of the coefficient that connects X to M (a) and the coefficient that connects M to Y (b). A significant indirect effect indicates statistical mediation (Preacher & Hayes, 2004). To approximate the sampling distribution for the indirect effect, 1000 percentile bootstrap resamples will be used to create 95% confidence intervals (CIs; Preacher & Hayes, 2004). This analysis will be preliminary, given that individualized estimates tend to have a high level of error variance. Hypothesis 3 support would be demonstrated by a significant indirect effect, such that arousal-induced decays in working memory at least partially statistically mediate the relation between emotion-related impulsivity and internalizing symptoms.

A Priori Power Analyses to Determine Sample Size

A priori power analyses were determined based on simulations and through the use of G*Power (version 3.1.9.2; Faul et al., 2007)) and MedPower (Kenny, 2017) to estimate required sample size for primary analyses with a medium effect size (Cohen's $f = 0.25$). For Aims 1 and 2, we based sample size on standard recommendations for multilevel tests of fixed effects derived from Monte Carlo simulations, which generally converge on a minimum level-2 sample size of 50 (Bell et al., 2014; Maas & Hox, 2005). In addition, G*power was used for a priori sample size estimates for fixed effects and projects 73 participants would be needed to test three predictors (emotion-related impulsivity/internalizing symptoms, arousal, and their interaction) and three control variables (gender, age, and medication use). For Aim 3, MedPower projected a sample size of 90 to detect the indirect effect, based on prior work assessing the paths between X, emotion-related impulsivity, and M, arousal-induced decays in cognitive control (.30; Pearlstein et al, 2019); M, arousal-induced decays in working memory, and Y, internalizing symptoms, (.31; Quinn & Joormann, 2015); and X, emotion-related impulsivity, and Y, internalizing symptoms, (.38; Pearlstein et al., n.d.). Note that prior work has not directly tested these effects, and these are approximations based on the best available estimates. Given prior studies' rates of missing and invalid data (e.g., Pearlstein et al, 2019), it is expected ~7% of data will be unusable, thus 100 subjects were recruited for sufficient power to detect the hypothesized effects. Post-hoc power analyses were conducted based on detected effects since missing and unusable data exceeded this hypothesized estimate leading to a smaller sample than projected.

Results

Descriptive information and bivariate correlations of self-report, behavioral, and block 2 psychophysiological measures are shown in Table 1. All self-report, behavioral, and psychophysiological measures were within the acceptable ranges for skew and kurtosis (Klein,

2011). Partial correlations between working memory, tonic SCL and pupil dilation during block 2 controlling for block 1 are shown in Table 2.

Potential control variables of age, gender, site of data collection, medication use, and stress exposure were examined in relation to behavioral and psychophysiological measures. Temperature and humidity were considered in relation to skin conductance. Session date was also considered to determine whether behavioral performance or psychophysiological activation varied from before to after the advent of the COVID-19 pandemic. Effects for these potential control variables are shown in Table 3. Given the effects for age, stress exposure, and date of completion, these variables were considered for inclusion as controls in subsequent analyses, however, ultimately none of these were included for the following reasons. Age did not relate to psychophysiological variables. Stress exposure would have likely been over-control given the large correlations with self-report variables. Date did not impact the primary dependent variable (working memory performance) and the effect for pupil dilation was small. Combined with concerns about power, these controls were not included in subsequent models.

Preliminary Analyses

Internalizing Symptom Latent Variable. The self-reported measures of internalizing symptoms, the MASQ, PSWQ, DOCS, and SIAS were re-scaled and entered into a structural equation model to create a single latent variable for internalizing symptoms. The model (shown in Figure 2) had good fit, as indicated by $x^2(2) = 2.11$, $p = .35$; CFI = .99; SRMR = .03; and RMSEA = .04). Latent variable values were extracted for use in subsequent analyses. The latent construct based on internalizing measures was normally distributed based on visual inspection and tests of skew (0.02) and kurtosis (2.85). The latent variable for internalizing symptoms significantly correlated with Feelings Trigger Action ($r(80) = .38$, $p < .01$), Pervasive Influence of Feelings ($r(80) = .42$, $p < .01$), and Lack of Follow Through ($r(80) = .49$, $p < .01$).

Individualized Estimate for Arousal-Induced Decays in Working Memory. To preserve the temporally dynamic nature of arousal-induced decays in working memory, an individualized effect size for the strength of the relationship between arousal (prior trial pupil dilation) and working memory (N-back) was derived for each participant (ß's = -.60-.34, $M_{beta} = -.01$, $SD_{beta} = .05$; R^2's= .00-.01, $M_{Rsq} = -.08$, $SD_{Rsq} = .01$). Because estimates were both positive and negative, ß estimates were used in subsequent analyses. Individualized estimates (ß's) were normally distributed based on visual inspection and tests of skew (-0.77) and kurtosis (5.67).

Effectiveness of the Arousal Manipulation. The effectiveness of the stressor for eliciting arousal was evaluated by conducting two linear mixed effect models for average tonic SCL and pupil dilation to determine whether segments of the task (baseline, block 1, block 2 [stressor block], and block 3) differed significantly after accounting for the random effect of the participant. Where omnibus significant effects for task segment emerged, more specific contrasts to assess which blocks differed were examined by evaluating the contrasts between levels (i.e., task segment). As shown in Table 4 and Figure 3, task segment had an omnibus effect for tonic SCL($F(3, 127) = 28.16$, $p < .001$) and pupil dilation ($F(3, 243) = 12.59$, $p < .001$). Each segment was significantly different for average tonic SCL, and block 2 elicited the greatest average tonic SCL (β =.48, SE =.06, $t(127) = 8.34$, $p < .001$) across segments. For pupil dilation, arousal was meaningfully greater than baseline during all blocks (β's = .10-.27, SE =.05, $t(243)$'s = 2.00-5.69, $p < .001$); though block 2 (β = .10, SE =.05, $t(243) = 2.00$, $p = .05$) elicited significantly less pupil dilation than blocks 1 (β = .28, SE =.05, $t(243) = 5.70$, $p < .001$) or 3 (β = .21, SE =.05, $t(243) = 4.27$, $p < .001$). Partial correlations between tonic SCL and pupil dilation during

block 2 controlling for block 1 were small and insignificant (see Table 2), which suggests a lack of coherence across measures of arousal.

Using linear regression, none of the three impulsivity factors related to average tonic SCL ($|\beta|$'s = .01-06, *SE*'s = 02-04, t(27)'s = -.12-1.73, p's > .08) or pupil dilation ($|\beta|$'s = .01-11, *SE*'s = 06-.07, *t*(76)'s = -1.63-.42 *p's* > .11) during the second block controlling for the first block, which indicates comparable stress reactivity across impulsivity levels.

To ensure changes in pupil dilation during block 2 were in response to arousal as opposed to sound, two sets of analyses were performed. First, the pre-task sound test segment was analyzed using mixed effect modeling to examine the fixed effect of volume of the sound (none, low, or high) on pupil dilation (β = .00, *SE* =.00, *t* = -.69 , *p* = .49) with the random effect for the participant included. Second, trials with sound were compared to trials without sound through a mixed effect model evaluating the fixed effect of volume of the sound (none, low, or high) on the subsequent trial's pupil dilation (β = .00, *SE* =.00, *t* = -1.27 , *p* = .20), again with the random effect for the participant included. Although individual responses vary significantly across trials, Figure 4 illustrates an example of the temporally dynamic pupillary response to during a standard trial in block 1 and during a sound-trial in block 2. Together, these results suggest changes in pupil dilation were not in response to sound or volume, and thus volume was not included as a covariate in analyses.

Primary Analyses

Arousal-induced changes in working memory. A generalized linear effect model assessed whether prior trial pupil dilation was related to working memory accuracy by trial. Including the random intercept for each participant, higher arousal did not significantly reduce working memory accuracy (β = 0.01, *SE* =.06, *z* = .19, *p* = .84), in fact, the overall effect was very modestly in the positive direction. The inclusion of a random slope did not improve fit, suggesting that there were no pronounced individual differences in the magnitude or direction for the effect of pupil dilation on working memory accuracy. Alternative relationships including curvilinear (squared, cubic, and logged) were examined and no impact of arousal on working memory was found (all β's = -0.01-.01, *SE* =.01-.31, *z* = -.05-.65, *p* = .52-.95). As depicted in Table 4 and Figures 5 and 6, working memory accuracy was not meaningfully different across task blocks. Partial correlations between arousal and working memory during block 2 controlling for block 1 were also insignificant (see Table 2).

Relationship between emotion-related impulsivity and arousal-induced decay in working memory. Three parallel separate generalized linear mixed-effects models were examined for each of the three impulsivity factors. Each model regressed the trial-level working memory accuracy on the respective impulsivity factor, prior trial pupil dilation, and the interaction of impulsivity by prior trial pupil dilation. As shown in Table 5, Pervasive Influence of Feelings (β = 0.20, *SE* =.03, *z* = 2.14, *p* < .05) had a significant impact on trial-level working memory accuracy, however, the hypothesized interaction of Pervasive Influence of Feelings with pupil dilation was not significant. Feelings Trigger Action, Lack of Follow Through, and their interactions with pupil dilation were not related to trial-level working memory accuracy. Separate models considered the random effect for the slope of prior trial pupil dilation and the pattern of results remained the same, which is consistent with the models above that suggests that there were no meaningful individual differences in the magnitude or direction of effects.

Relationship between internalizing symptoms and arousal-induced changes in working memory. A generalized linear mixed-effect model regressed the trial-level working

memory accuracy on internalizing symptoms, prior trial pupil dilation, and the interaction of internalizing symptoms by prior trial pupil dilation. The small main effects for internalizing symptoms (β = 0.10, *SE* =.10, *z* = 1.69, *p* = .30) and pupil dilation (β = -0.02, *SE* =.06, *z* =-.07, *p* = .70) on working memory performance were not significant. Contrary to hypotheses, the effect of the interaction of internalizing symptoms and pupil dilation (β = -.10, *SE* =.07, *z* = 2.11, *p* < .17) on working memory performance was also insignificant.

Do arousal-induced decays in working memory mediate the relation between emotion-related impulsivity and internalizing symptoms? An initial linear regression confirmed the relationship between emotion-related impulsivity and internalizing symptoms. When entered into the same model, the main effect of Pervasive Influence of Feelings (β =.04, *SE* =.01, *t*(79) =3.84, *p* < .001), but not Feelings Trigger Action (β = 0.01, *SE* =.01, *t*(79) =1.17, *p* = .26), was significant for internalizing symptoms. Given Pervasive Influence of Feelings was also the only impulsivity factor that significantly related to working memory performance in mixed effect models, only this emotion-related impulsivity factor was tested in the mediation model.

The effect of Pervasive Influence of Feelings on internalizing symptoms was not mediated via arousal-induced decays in working memory. As Figure 7 illustrates, the regression coefficient of Pervasive Influence of Feelings with arousal-induced decays in working memory was small and insignificant (β = 0.07, *SE* =.12, *t*(72) =.61, *p* = .55) as was the regression coefficient of arousal-induced decays in working memory and internalizing symptoms (β = -.08, *SE* =.11, *t*(72) =--.72, *p* = .47). The significance of the effects was evaluated using bootstrapping procedures. Standardized indirect effects were computed for each of 1'000 bootstrapped samples. The bootstrapped standardized indirect effect was -.02, and not significant (*p* = .55), 95% confidence interval = -.17 to .03.

Exploratory Analyses of Externalizing Symptoms

Parallel with the internalizing model to evaluate hypothesis 2, a generalized linear mixed-effect model regressed trial-level working memory accuracy on externalizing symptoms (β = 0.21, *SE* =.10, *z* =2.10, *p* < .05), prior trial pupil dilation (β = -0.02, *SE* =.07, *z* = -.26, *p* = .80), and the interaction of externalizing symptoms by prior trial pupil dilation (β = 0.02, *SE* =.07, *z* = .23, *p* = .82).

A linear regression was used to evaluate the relationships between emotion-related impulsivity factors and externalizing symptoms. When entered into the same model, Feelings Trigger Action (β =.33, *SE* =.12, *t*(79) =2.63, *p* < .05), but not Pervasive Influence of Feelings (β = 0.07, *SE* =.13, *t*(79) =.52, *p* = .61) had a significant main effect on internalizing symptoms. Given Feelings Trigger Action was the only impulsivity factor with a significant effect on externalizing symptoms, this factor was tested in the exploratory mediation model.

The effect of Feelings Trigger Action on externalizing symptoms was not mediated via arousal-induced changes in working memory. As Figure 8 illustrates, the regression coefficient of Feelings Trigger Action with arousal-induced changes in working memory was small and insignificant (β = 0.04, *SE* =.12, *t*(72) =.37, *p* = .72) as was the regression coefficient of arousal-induced decays in working memory and externalizing symptoms (β = 0.14, *SE* =.12, *t*(72) =1.16, *p* = .25)). The bootstrapped standardized indirect effect was -.02, and the 95% confidence interval ranged from -.24 to .24. Thus, the indirect effect was not statistically significant (p=.92).

Post-hoc Analyses

Power. Because the included sample size (*n* = 82) was below the planned size in a priori power analyses, post-hoc power analyses were conducted. Post-hoc power curves were estimated

to assess whether our sample size would be adequate to detect level 1 effects in our mixed effect models (Kleinam, 2021). With the actual sample size ($n = 82$) and trials per participant ($k = 180$), large effects were powered at the >0.8 level and medium effects were powered at the 0.6 level; small effect sizes were not well powered (<0.5 level). To determine power for level 2 effects, simulations to determine the adequate sample size for mixed models were used. In addition to sample sizes estimates reported in a priori power considerations (i.e. greater than 50 yielded unbiased estimates, variance, and standard errors [Maas & Hox, 2005]), cross-level Monte Carlo simulations suggest the present study is sufficiently powered above the 0.8 level (Mathieu et al., 2012). Mediation effects were not sufficiently powered given the very small effects detected. Based on MedPower (Kenny, 2017), the path between X (Pervasive Influence of Feelings) and Y (internalizing symptoms) was detected at the >.99 level, however, effects were only powered at the .05 level to detect the paths between each of these variables and the moderator (arousal-induced decays in working memory. These post-hoc power analyses suggest caution when interpreting the indirect effects in the mediation model, however, the direct paths appear sufficiently powered.

  Post-hoc analyses using tonic SCL as a marker of arousal. Given that pupil dilation did not show expected increases during the stressor block compared to blocks 1 and 3, there was reason to doubt whether pupil dilation was a valid measure of arousal. Accordingly, I conducted parallel analyses to examine relationships with tonic SCL. Although SCL does not provide the temporal specificity for trial-level analyses, I examined whether increases in SCL in block 2 predicted diminished working memory performance during that block ($\beta = .01$, $SE = .02$, $t(41) = .50$, $p = .62$) and partial correlations among block 2 psychophysiological variables and working memory controlling for block 1 (see Table 2). I also evaluated a linear mixed effect model that regressed block-level working memory on tonic SCL ($\beta = .02$, $SE = .12$, $t(72) = .18$, $p = .86$). Finally, I implemented parallel linear models relating the average tonic SCL during block 2, controlling for block 1, to Feelings Trigger Action ($\beta = -.07$, $SE = .06$, $t(40) = -1.15$, $p = .26$), Pervasive Influence of Feelings ($\beta = .07$, $SE = .06$, $t(40) = 1.09$, $p = .28$), internalizing symptoms ($\beta = .06$, $SE = .04$, $t(40) = 1.79$, $p = .08$), and externalizing symptoms ($\beta = .03$, $SE = .04$, $t(40) = .78$, $p = .44$). I considered the effect of each of the above variables in linear models relating block 2 working memory performance, controlling for block 1, to tonic SCL during block 2, each individual difference variable, and the interaction between tonic SCL and each individual difference variable. There were no main effects or interaction terms across these models ($|\beta|$'s < .10, $SE's = .07-.12$, $|t(38)|$'s $=.30-.75$, $p's > .46$).

<div align="center">

Discussion

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  Emotion-related impulsivity is robustly associated with diverse forms of psychopathology and problematic behavior, which makes it important to understand the neurocognitive and affective processes by which this trait yields poor outcomes. I conducted the present study to examine a putative process that may underlie emotion-related impulsivity and its link with internalizing symptoms. Through the use of a novel paradigm to induce stress and monitor working memory continuously, I examined arousal-induced decays in working memory in relation to emotion-related impulsivity and internalizing symptoms. A key study strength was the analysis of dynamic trial-by-trial fluctuations in arousal and working memory performance. Consistent with hypotheses, emotion-related impulsivity was positively correlated with internalizing symptoms. Contrary to hypotheses, working memory performance was not significantly impacted by arousal—that is, working memory performance was consistent across

blocks of the task and prior trial pupil dilation did not impact subsequent trial working memory accuracy. Further, I did not find significant individual differences in arousal-induced decay in working memory. Of more import, arousal did not interact with emotion-related impulsivity or internalizing symptoms in relation to working memory performance.

In preliminary analyses of bivariate correlations, I replicated previous findings that emotion-related impulsivity is robustly associated with internalizing symptoms of psychopathology. The strong association of Feelings Trigger Action and Pervasive Influence of Feelings with the internalizing symptom composite is consistent with other studies that suggest emotion-related impulsivity is a transdiagnostic correlate of psychopathology (Berg et al., 2015; Johnson, Carver, & Joormann, 2013). Lack of Follow Through, the general impulsivity factor, was also related to the internalizing symptoms latent variable. The nonspecific profile of effects across impulsivity factors was contrary to hypotheses and other research, however, this result nonetheless converges with other work on transdiagnostic associations with general impulsivity (Castellanos-Ryan et al., 2016).

The first aim of the current study was to evaluate arousal-induced decay in working memory overall and in relation to individual differences in emotion-related impulsivity and internalizing symptoms. I first sought to replicate overall decays in working memory during periods of high arousal (Shields et al., 2016). Despite evidence that arousal was higher during block 2 based on tonic SCL, and although working memory performance during block 2 was numerically lower than in block 1 on average, working memory accuracy did not differ significantly across blocks; further, there was no evidence from pupil that arousal was higher in block 2, and dynamic trial-by-trial pupil dilation did not predict working memory performance. Across models, arousal did not have an effect on working memory within this study.

I then examined individual differences by conducting three parallel models to examine the three impulsivity factors, prior trial pupil dilation, and the interaction between emotion-related impulsivity and prior trial pupil dilation in relation to working memory performance. I hypothesized that the two emotion-related impulsivity factors but not the general impulsivity factor would interact with arousal in relation to working memory. Here again, findings did not support hypotheses, with null effects for Feelings Trigger Action or Lack of Follow Through, and, contrary to hypotheses, higher Pervasive Influence of Feelings related to significantly better working memory performance. Across models, the hypothesized interaction of arousal and the impulsivity factors on working memory was not supported.

The second aim was to evaluate arousal-induced decays in working memory in relation to internalizing symptoms. The hypothesis that internalizing symptoms, arousal, and their interaction would correlate with working memory was also unsupported. Whereas several findings have found relationships among internalizing symptoms and arousal-induced decays in working memory (Quinn & Joormann, 2015, 2020), and meta-analyses across internalizing disorders report poorer working memory (Abramovitch et al., 2013; Moran, 2016; Nikolin et al., 2021), in this sample, higher internalizing symptoms were associated with numerically though insignificantly improved working memory performance overall and in block 2 specifically.

The third aim was to assess a mediation model to determine whether arousal-induced decays in working memory explain the link between emotion-related impulsivity and internalizing symptoms. Despite the absence of support for arousal-induced decays in working memory, mediation modeling was conducted with Pervasive Influence of Feelings (X), arousal-induced decays in working memory (M), and internalizing symptoms (Y). Mediation was not supported. Given arousal did not induce decays in working memory, this was not a putative

mediator for the relationship between emotion-related impulsivity and internalizing symptoms. The effects detected were small and indeed were in an opposite direction of hypotheses, in that higher pupil dilation related to better working memory performance across the task.

The primary aims and hypotheses were focused on internalizing symptoms based on prior work and theory, however, parallel exploratory models were conducted using externalizing symptoms. Consistent with earlier work (Johnson et al., 2017), Feelings Trigger Action and Lack of Follow Through, but not Pervasive Influence of Feelings, related to externalizing symptoms with larger effects emerging for Feelings Trigger Action. The strong positive correlation coheres with the extensive literature documenting associations between Urgency and various externalizing syndromes (Derefinko et al., 2011; Kaiser et al., 2016; Murray et al., 2012). The observed significant bivariate positive correlation between externalizing symptoms and working memory accuracy contradicts prior work (e.g. Finn et al., 2015). As was the case for internalizing symptoms, externalizing symptoms did not relate to arousal-induced decays in working memory.

Despite the absence of evidence supporting arousal-induced decays in working memory in relation to Feelings Trigger Action or externalizing symptoms, exploratory mediation analyses were conducted to examine Feelings Trigger Action (X) and arousal-induced decays in working memory (M) in relation to externalizing symptoms (Y). Results mirrored the pattern in the internalizing model with Feelings Trigger Action impacting externalizing significantly, but no support for paths with M or mediation. Consistent with the results from the internalizing model, the effects detected were small and indeed were in an opposite direction of hypotheses, again with higher arousal relating to marginally better working memory performance.

The lack of support for study hypotheses is likely impacted most by three key methodological decisions: (1) the reliance on pupil dilation as the dynamic marker for arousal; (2) the relative lack of difficulty in the working memory task; and (3) the use of socially evaluative noxious noise as a stressor. I discuss the effect of these methodological considerations and the implications for the interpretation of results below.

First, all study aims relied on the use of pupil dilation, and pupil dilation did not appear to demarcate arousal specifically and sensitively in the present investigation. Whereas considerable research has shown that pupil dilation correlates with heart rate and skin conductance (Bradley et al., 2008; Wang et al., 2018), pupil dilation did not correlate with skin conductance measures in the present study, and unlike skin conductance-- which showed the expected increase during block 2 compared to blocks 1 and 3, pupil dilation was significantly greater in blocks 1 and 3 than in block 2. Although I initially intended to collect skin conductance across all participants, researchers at one site were not able to gather skin conductance. The limited numbers of skin conductance data combined with the relative lack of temporal specificity reduced the feasibility of considering key aims with skin conductance as an alternate metric to pupil dilation

The relatively reduced pupil dilation during block 2 contradicts prior work that has demonstrated more pupil dilation in response to noises of various types compared with silence during cognitive tasks (Antikainen & Niemi, 1983; Liao et al., 2016). Pupil dilation is innervated by multiple processes. Block 1 pupil dilation may have been the greatest due to the novelty, effort, and/or cognitive load involved, as these also elicit greater pupil dilation (Alnæs et al., 2014; Beukema et al., 2019; Siegle et al., 2008). It is possible that pupil dilation was also increased in block 3 due to significant increases in relief after the removal of the stressor, as pupil dilation is associated with emotions across positive and negative valence (Bradley et al., 2008). Pupil dilation has also been shown to diminish while mind-wandering (Unsworth & Robison, 2016), and it is possible that participants experienced more moments of mental

distraction during the second block while experiencing socially-evaluative threat. In addition, instructions provided to participants informed them that they had a chance to improve their performance during the final section of the task, which may have inadvertently encouraged greater engagement that was reflected in increased pupil dilation in block 3. Regardless of the mechanisms, pupil dilation did not appear to function as a marker of arousal in the present investigation.

Second, the design of the working memory task may have limited the feasibility to detect hypothesized results. The task had high internal consistency, however, average performance was substantially higher (86.4% accurate overall, 86.0% during block 2) than performance levels observed in previous research with psychiatric samples (around 75%) [Sandström et al., 2012]). In a meta-analysis, working memory load was shown to moderate the effect of stress on working memory with greater stress-induced decays at higher working memory loads (Shields et al., 2016), which suggests the high performance detected here may indicate that the 2-back was not a high enough working memory load to observe arousal-induced decays. During study design, adaptive versions of the task were considered in which load varies in difficulty based on performance, however, cognitive load was standardized across participants to reduce this confound on pupil dilation (Alnæs et al., 2014).

Third, the use of noxious noise to elicit social-evaluative threat may not have been a sufficiently potent stressor to detect effects. The use of noise to convey socially evaluative feedback was selected over other well-validated laboratory stressors such as shock, the cold pressor task, and the Trier Social Stress Test in order to (a) include a continuous stressor in the working memory task and (b) reduce sensory and psychophysiological confounds. Prior meta-analytic evidence supports the use of noise for eliciting working memory performance decays (Szalma & Hancock, 2011), however, the use of sound as feedback did not yield decays in working memory in the present investigation. The small and insignificant difference in performance across task blocks opposes research documenting decays in working memory in response to socially-evaluative threats (e.g. van Ast et al., 2014). Results provided partial support for the use of noxious noise to induce socially evaluative threat to elicit high arousal in that tonic SCL, but not pupil dilation, was significantly higher during block 2. Despite this evidence for arousal elicitation as measured by tonic SCL, results did not indicate poorer working memory performance during block 2 or in relation to arousal magnitude.

Beyond the methodological limitations related to the use of pupil dilation, the 2-back working memory task, and noxious noise to manipulate arousal, results need to be interpreted within the confines of additional limitations. First, the advent of the COVID-19 pandemic disrupted data collection, which impacted the sample size and may limit the generalizability of results. While the target number of data collection sessions was obtained, there were significantly more data exclusions ($n = 17$) due to technological and experimenter errors than anticipated, which reduced the sample size for analyses, and timeline limitations prevented additional data collection. Although analyses did not indicate significant differences in key parameters among those who participated before versus after COVID-19, it remains possible that those willing to participate in research during the earlier stages of reopening after the most acute phase of the COVID-19 pandemic represented a more able-bodied cross-section of the population, and the generalizability of results needs to be interpreted with caution. Second, due to technical errors with data recording, self-report data to confirm the manipulation of arousal was unusable. Although the study included two measures to verify arousal, the inclusion of

subjective affect ratings would have increased confidence that the task elicited subjective stress, especially given the unexpected decrease in pupil dilation during block 2.

Implications and Future Directions

Substantial evidence documents ties between emotion-related impulsivity with diverse forms of psychopathology and problematic behaviors. In the present study, I assessed a novel model of the process underlying emotion-related impulsivity and internalizing symptoms. Specifically, I examined whether arousal-induced decays in working memory related to emotion-related impulsivity, and statistically mediated the effects of emotion-related impulsivity on internalizing symptoms of psychopathology. Results replicated the robust association between impulsivity and transdiagnostic symptoms of psychopathology. Contrary to hypotheses, results failed to support the role of arousal-induced decays in working memory in relation to emotion-related impulsivity or internalizing symptoms. Perhaps the most important barrier in examining this theory is that participants generally showed high performance across the working memory task—limiting the ability to examine deficits in working memory. Also of concern, an unexpected inverse pattern of pupil dilation indicated that pupil dilation was not a sensitive or specific marker of arousal. Further, it is possible that the stress induction used was not sufficiently potent to detect effects. Together, it is possible that the present study did not include optimal methods to evaluate hypotheses.

Future research with improved methods is warranted given the current study cannot fully elucidate whether null results reflect methodological issues or lack of support for the model. The present findings contradict the large and growing literature documenting arousal can induce decays in working memory, and that the extent of such decay is related to internalizing symptoms. Other work has identified arousal-induced fluctuations in working memory using pupil dilation (e.g., Unsworth & Robison, 2017), which suggests that either the working memory load was not high enough to be vulnerable to stress or the use of noxious noise to elicit socially-evaluative threat was not effective at inducing arousal. Additional work is needed using alternative measures of working memory, as well as diverse psychophysiological and neuroimaging methods. Further pursuit of this model could identify the process underlying emotion-related impulsivity which could point to potential targets for intervention

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Figure 1 Stress elicits arousal leading to decays in working memory
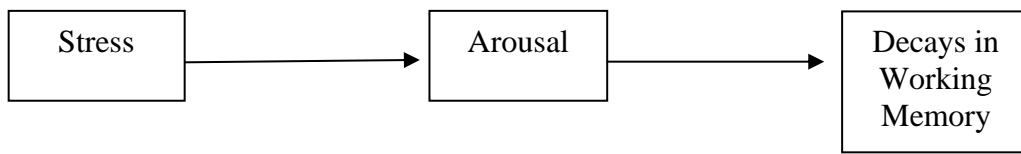
Note. Stress, an external stimulus, elicits arousal, an internal neurobiological response, which in turn effects working memory.

Table 1. Means, standard deviations, and correlations with confidence intervals

Variable	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Feelings Trigger Action	2.88	0.73														
2. Pervasive Influence of Feelings	3.60	0.86	.46** [.27, .61]													
3. Lack of Follow Through	3.04	0.91	.38** [.18, .55]	.61** [.45, .73]												
4. MASQ Total	1.40	0.56	.14 [-.08, .34]	.25* [.04, .45]	.21 [-.01, .40]											
5. DOCS Total	0.72	0.56	.20 [-.02, .40]	.05 [-.17, .26]	.14 [-.08, .34]	.25* [.03, .44]										
6. PSWQ Total	3.21	0.94	.29** [.08, .48]	.50** [.32, .65]	.45** [.26, .61]	.13 [-.09, .34]	.29** [.07, .48]									
7. SIAS Total	2.55	0.84	.18	.44**	.47**	.33*	.36**	.51**								

	M	SD	1	2	3	4	5	6	7	8	9	10	11	12
			[-.10, .43]	[.19, .64]	[.22, .66]	[.06, .55]	[.09, .58]	[.27, .69]						
8. Risky Families	2.04	0.67	.15 [-.07, .36]	.21 [-.01, .41]	.23* [.01, .43]	.11 [-.11, .32]	.15 [-.07, .36]	.28* [.06, .47]	-.01 [-.29, .27]					
9. Internalizing	0.00	0.78	.32** [.11, .50]	.48** [.29, .63]	.49** [.30, .64]	.37** [.17, .55]	.61** [.45, .73]	.80** [.71, .87]	.86** [.76, .92]	.23* [.01, .43]				
10. ESI Total	40.79	22.48	.35** [.14, .53]	.22 [-.00, .42]	.25* [.04, .45]	-.02 [-.24, .20]	.08 [-.14, .30]	.10 [-.12, .31]	.20 [-.08, .45]	.20 [-.02, .40]	.17 [-.05, .37]			
11. Working Memory Accuracy	0.86	0.11	-.04 [-.26, .18]	.24* [.02, .43]	.18 [-.04, .39]	.04 [-.18, .26]	-.07 [-.29, .15]	.15 [-.07, .36]	.20 [-.09, .45]	-.25* [-.45, -.03]	.13 [-.09, .34]	.26* [.04, .46]		
12. Block 2 Working Memory Accuracy	0.86	0.12	-.05 [-.27, .17]	.24* [.03, .44]	.17 [-.05, .38]	.02 [-.20, .23]	-.08 [-.29, .14]	.14 [-.08, .35]	.19 [-.09, .44]	-.25* [-.44, -.03]	.12 [-.10, .33]	.27* [.05, .46]	.98** [.97, .99]	
13. Block 2 Average Pupil Dilation	4.18	1.06	.02 [-.20,	.02 [-.20,	.01 [-.21,	.44** [.25,	-.09 [-.30,	-.17 [-.37,	-.07 [-.34,	-.04 [-.25,	-.05 [-.27,	-.18 [-.38,	.03 [-.19,	.01 [-.21,

	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14
			.23]	.24]	.23]	.60]	.13]	.06]	.22]	.18]	.17]	.05]	.24]	.23]		
14. Block 2 Average Tonic SCL	14.63	9.92	-.25	-.23	-.06	-.09	-.24	-.03	-.19	-.29	-.18	-.17	.12	.11	.14	
			[-.55, .10]	[-.54, .12]	[-.40, .30]	[-.43, .26]	[-.54, .12]	[-.38, .32]	[-.50, .17]	[-.59, .07]	[-.50, .18]	[-.49, .19]	[-.24, .45]	[-.25, .44]	[-.22, .47]	
15. Block 2 Total SCRs	27.25	15.53	-.24	-.04	-.04	.07	.02	.13	.02	-.32	.07	-.02	.17	.14	.14	.49**
			[-.54, .12]	[-.38, .31]	[-.38, .31]	[-.28, .41]	[-.33, .36]	[-.23, .46]	[-.33, .37]	[-.60, .04]	[-.29, .41]	[-.36, .33]	[-.19, .49]	[-.22, .46]	[-.22, .46]	[.17, .71]

Note. N's varied; variables 1-13 included 82 participants and 14-15 included 43 participants. M and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. DOCS = Dimensional Obsessive-Compulsive Scale total score; ESI Total = Externalizing Symptoms Inventory total score; MASQ = Mood and Anxiety Symptom Questionnaire total score; PSWQ = Penn State Worry Questionnaire total score; SIAS = Social Interaction Anxiety Scale total score. * $p < .05$. ** $p < .01$.

Table 2. Partial correlations between working memory, psychophysiological variables, and internalizing symptoms

Variable	Working Memory	Pupil Dilation	Tonic SCL
Pupil Dilation	-.01		
Tonic SCL	-.03	.03	
Internalizing	-.10	-.18	.27

Note. Values represent Pearson *r* partial correlations. *N's* varied; working memory, pupil dilation, and internalizing included 82 participants; tonic SCL included 43 participants. For working memory and the psychophysiological variables, measures collected during block 2 were examined controlling for measures during block 1. Internalizing symptoms was measured by the internalizing latent variable. None of these effects were statistically significant (p's > .05), though the relationship between internalizing and block 2 tonic SCL controlling for block 1 tonic SCL was a medium effect size and trending towards significance (p = .08).

Table 3. Consideration of potential covariates and confounding variables

Predictor	Outcome	df	r	p	ß	SE	t	p
Age	Working memory	80	-.44	<.001				
	Skin conductance	40	-.29	.06				
	Pupil dilation	79	-.09	.43				
Gender	Working memory	79			-.01 - .08	.03-.07	-.39- -1.25	>.21
	Skin conductance	39			-.26 - -.08	.08-.56	-1.62- -1.03	>.11
	Pupil dilation	78			-.14- -.06	.11-.27	-62- -.53	>.54
Site	Working memory	79			.07 - .09	.07-.09	.90- -1.07	>.29
	Pupil dilation	78			.17-.30	.29-.31	-1.04 - -.56	>.30
Medication use	Working memory	79			.13	.11	.15	.26
	Skin conductance	40			-.16	.28	.58	.56
	Pupil dilation	79			-.75	.44	-1.70	.09
Stress exposure	Working memory	80	-.25	.02				
	Skin conductance	40	.04	.71				
	Pupil dilation	79	.01	.91				
Temperature	Skin conductance	38			-.02	.08	-1.08	.29
Humidity	Skin conductance	38			.00	.00	.79	.43
Date of completion	Working memory	80			-.11	.24	.47	.64
	Skin conductance	40			-.00	.01	.00	.99
	Pupil dilation	80			-.23	.11	-2.15	.04
	Age	80			-.98	2.48	-.39	.78

Note. Df = Degrees of freedom; SE = standard error. Working memory scores reflect accuracy, calculated as the average performance across trials. Skin conductance refers to average tonic SCL. Degrees of freedom, partial correlations (r's), standardized betas (ß*'s*), t-statistics, and p value based on separate, parallel analyses. For working memory, skin conductance, and pupil dilation, measures from block 2 were used controlling for block 1. Date corresponds to the indicator variable for prior to or after the advent of the COVID-19 pandemic. *N*'s varied; for working memory accuracy and pupil dilation, $N = 82$; for average tonic SCL, $N = 43$.

Figure 2. Internalizing symptoms latent variable ($N = 82$)
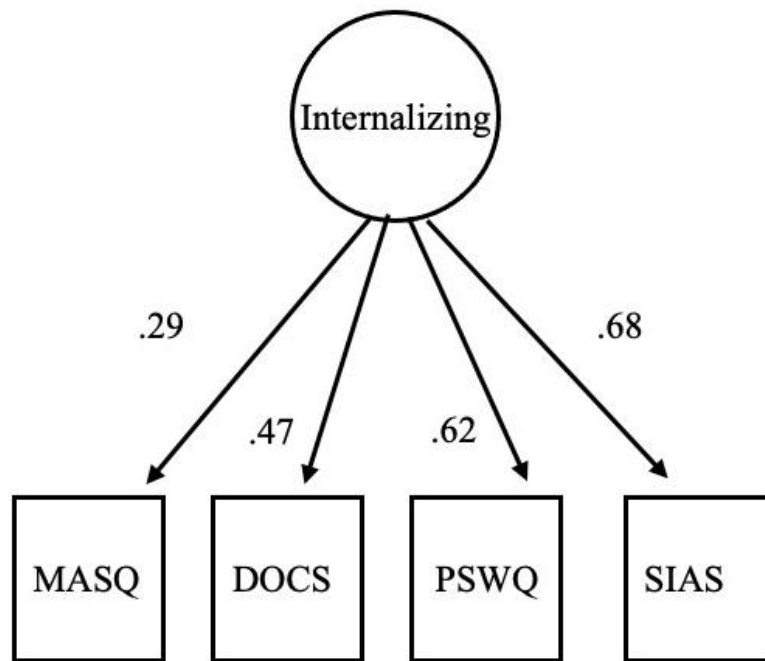
Note. DOCS = Dimensional Obsessive-Compulsive Scale total score; MASQ = Mood and Anxiety Symptom Questionnaire total score;; PSWQ = Penn State Worry Questionnaire total score; SIAS = Social Interaction Anxiety Scale total score. Variables were rescaled before modeling. All depicted paths are significant ($p < .05$). The above model had good fit, as indicated by $x^2(2) = 2.11$, $p = .35$; CFI = .99; SRMR = .03; and RMSEA = .04.

Figure 3. Aggregated pupillary responses during the working memory task ($N = 82$)
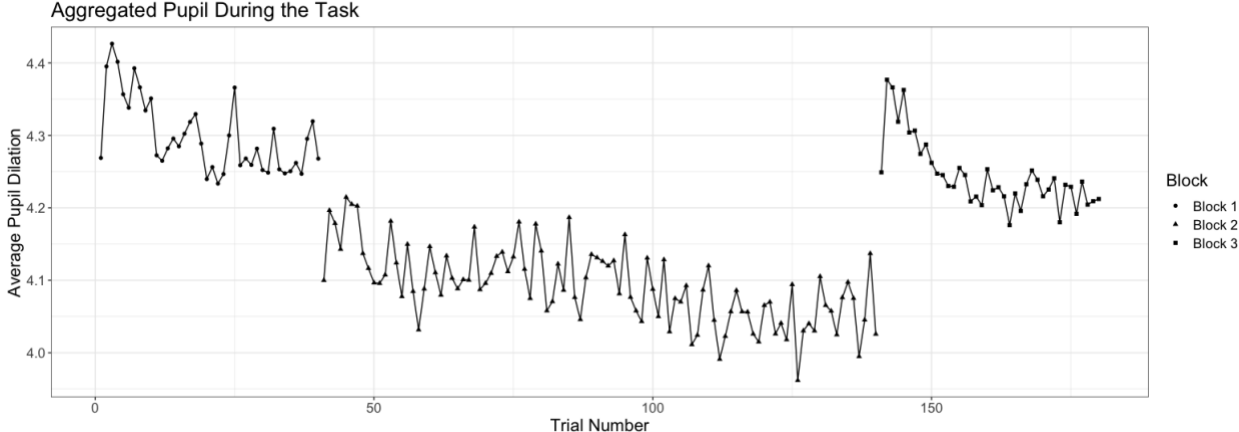
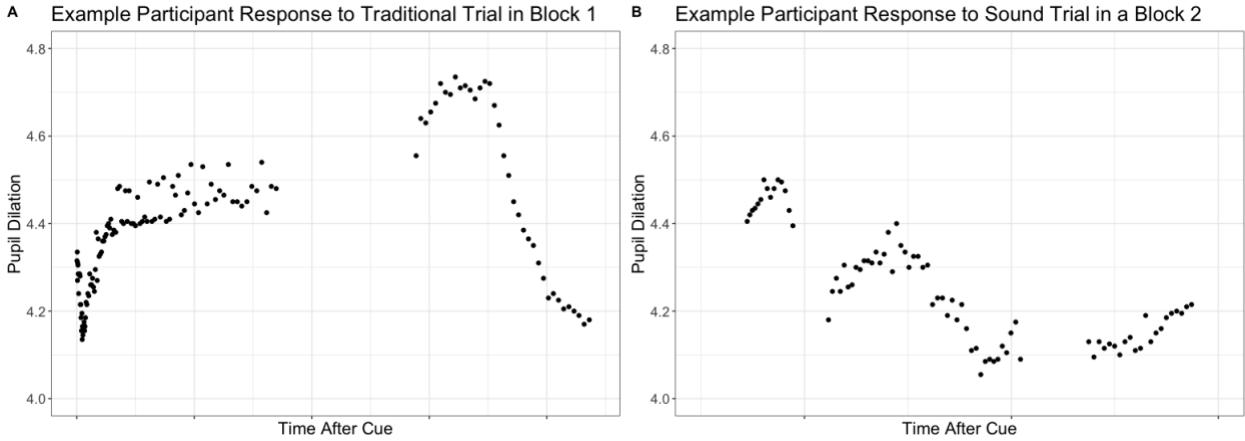Figure 4. Example participant pupillary response during a single trial.

Table 4. Means, standard deviations, and models comparing behavioral and psychophysiological variables across task blocks

Variable	Block 1		Block 2		Block 3		df	*F*	*p*
	M	*SD*	*M*	*SD*	*M*	*SD*			
Working memory	.87	.11	.86	.12	.87	.11	2, 160	1.78	.18
Pupil Dilation	4.36	.95	4.18	1.05	4.29	.98	3, 243	12.59	<.001
SCRs	5.88	4.21	26.65	14.63	5.70	4.09	3, 127	68.34	<.001
Tonic SCL	14.10	9.77	14.70	9.92	13.69	9.83	3, 127	28.16	<.001

Note. Df = Degrees of freedom. *M* and *SD* are used to represent mean and standard deviation, respectively. Working memory scores reflect accuracy, calculated as the average performance across trials in that block. *F* value, degrees of freedom, and *p* value based on separate, parallel linear models with block as the independent variable and the corresponding variable in the table as the dependent variable. *N*'s varied; for working memory accuracy and pupil dilation, $N = 82$; for total SCRs and average tonic SCL, $N = 43$.

Figure 5. Mean differences in working memory accuracy across blocks ($N = 82$)

Working Memory Accuracy
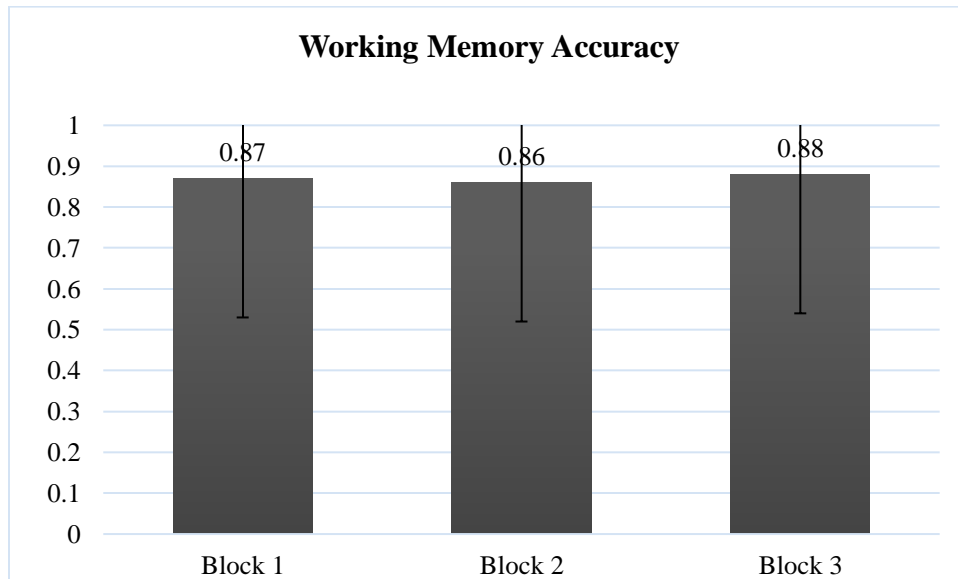
Note. Working memory accuracy was measured as the average across trials in each block, with 1 = correct and 0 = incorrect. Analysis of covariance confirmed no statistically significant differences across blocks ($F[2, 240] = .28$, $p = .75$).

Figure 6. Aggregated working memory performance ($N = 82$)

Aggregated Accuracy During the Task
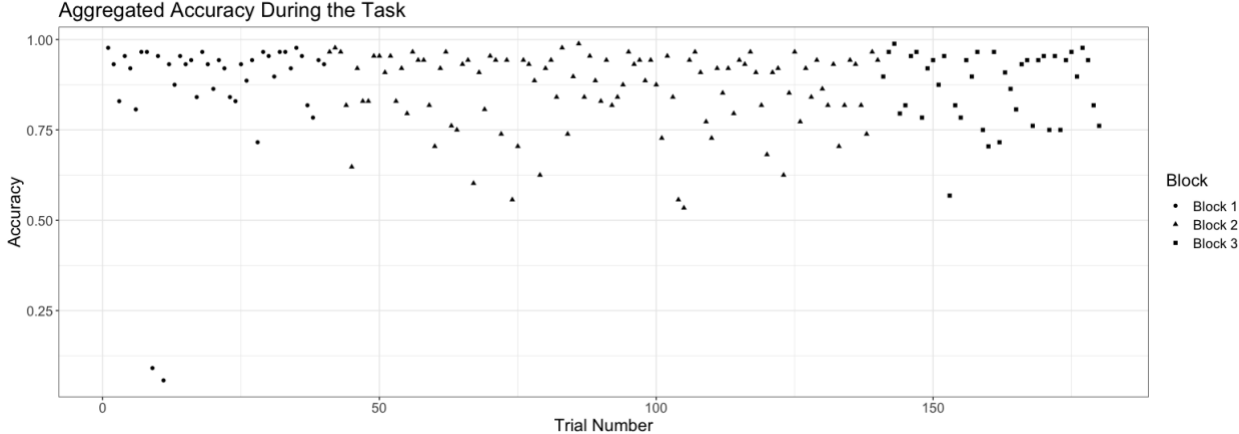
Block
· Block 1
▲ Block 2
■ Block 3

Table 5. Impulsivity and arousal (pupil dilation on prior trial) and their interaction in relation to working memory performance across all trials. ($N = 82$)

	Fixed Effects	ß	SE	p	Fit indices
Model 1: Feelings Trigger Action	Intercept	2.07	.10	<.01	AIC = 10246.7, BIC = 10284.4, log likelihood = -5118.3, $R^2 = .17$
	Pupil dilation (prior trial)	.09	.07	.20	
	Feelings Trigger Action	-.08	.10	.43	
	Pupil dilation X Feelings Trigger Action	.04	.64	.53	
Model 2: Pervasive Influence of Feelings	Intercept	2.08	.09	<.01	AIC = 10243.0, BIC = 10280.7, log likelihood = - 5116.5, $R^2 = .17$
	Pupil dilation (prior trial)	.07	.06	.31	
	Pervasive Influence of Feelings	**.20**	**.09**	**.03**	
	Pupil dilation X Pervasive Influence of Feelings	-.01	.06	.88	
Model 3: Lack of Follow Through	Intercept	2.07	.09	<.01	AIC = 10245.7, BIC = 10283.4, log likelihood = - 5117.8, $R^2 = .17$
	Pupil dilation (prior trial)	.08	.06	.19	
	Lack of Follow Through	.13	.09	.16	
	Pupil dilation X Lack of Follow Through	.02	.07	.79	

Note. ß = standardized beta, *SE* = standard error. All models include a random intercept (Model 1: variance = .70, *SD* = .84; Model 2: variance = .67, *SD* = .82; Model 3: variance = .70, *SD* = .83).

Figure 7. Mediation model in which arousal-induced decays in working memory explains the link between Pervasive Influence of Feelings and internalizing symptoms ($N = 82$)
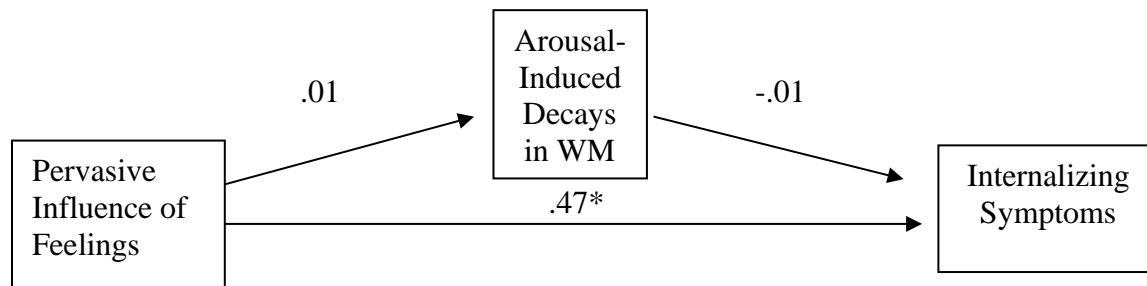
Note. WM = Working Memory, X =Pervasive Influence of Feelings, M = individualized estimate for Arousal-Induced Decays in Working Memory (WM). Y = internalizing symptoms latent variable. All paths represent standardized estimates. * $p < .01$.

Figure 8. Exploratory mediation model in which arousal-induced decays in working memory explains the link between Feelings Trigger Action and externalizing symptoms ($N = 82$)
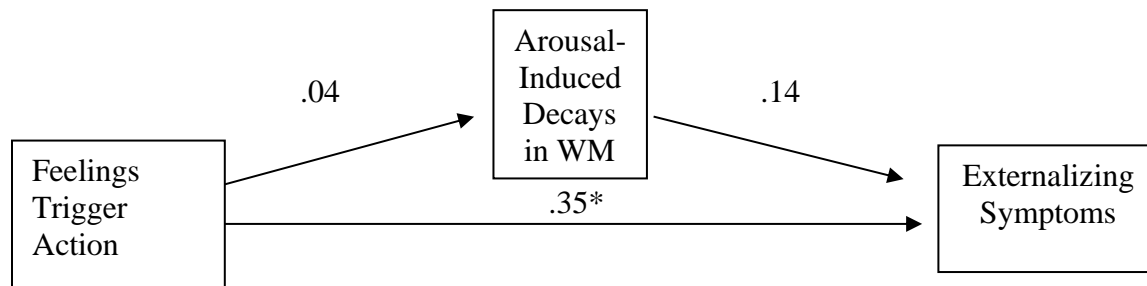
Note. WM = Working Memory, X = Feelings Trigger Action, M = individualized estimate for Arousal-Induced Decays in Working Memory (WM), Y = Externalizing Symptom Inventory total score. All paths represent standardized estimates. * $p < .01$.