The Role of Negative Urgency in Impulsivity and Anxiety

by

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ABSTRACT

Clinically significant anxiety and impulsivity are both highly prevalent and impair functioning in multiple domains. Historically, trait impulsivity has been considered the opposite of anxious avoidance, despite co-occurring presentations of anxiety and impulsivity across internalizing and externalizing disorder spectrums. Due to broad conceptualizations of impulsive behavior, it remains unclear whether anxiety could be positively associated with global impulsivity or specific facets of impulsivity. Negative urgency, or rash behavior that specifically occurs during negative emotional states, may provide common ground to examine the overlap between high anxiety and high impulsivity. Prior literature suggests that negative urgency is related to patterns of behavioral (e.g. experiential avoidance) and neural processes often associated with both anxiety and global impulsivity. This dissertation explored the role of negative urgency within relationships between anxiety, global impulsivity, experiential avoidance, and prefrontal cortex (PFC) activation.

In our first study, we examined self-report data collected from adult participants (n = 589) to test the indirect effect of anxiety on experiential avoidance through both negative urgency and global impulsivity. Contrary to our hypotheses, we found that there was no significant indirect effect of anxiety on experiential avoidance through negative urgency alone. However, follow-up analyses including data-driven factors of impulsivity revealed significant positive indirect effects of anxiety on experiential avoidance through emotion-based impulsivity

(i.e., negative and positive urgency) and hyperactivity. There was also a significant negative indirect effect of anxiety on experiential avoidance through a non-planning impulsivity factor. This study helps to inform future studies interrogating links between anxious avoidance and specific dimensions of impulsivity.

In our second study, we examined shared and distinct patterns of activation in the PFC associated with inhibitory control during emotional interference and motor inhibition tasks. Pre-adolescents (ages 9-10; n = 2264) from the Adolescent Brain Cognitive Development (ABCD) Study completed the Emotional N-Back (EN-Back) Task as a probe of emotional interference and the Stop Signal Task (SST) as a probe of motor inhibition during brain scanning. A conjunction analysis revealed shared activation across tasks in the inferior frontal gyrus (IFG); activation specific to the EN-Back task in the middle frontal gyrus (MFG); and activation specific to the SST in the dorsal anterior cingulate cortex (dACC) and dorsolateral PFC (dlPFC). These findings support distinctions in PFC involvement in emotional and non-emotional inhibitory control and suggest neural targets for future analyses using the ABCD dataset.

In our third study, we examined links between task-specific brain function identified in Study 2 and measures of anxiety and impulsivity. We aimed to explore the role of negative urgency in relationships between anxiety, global impulsivity, and PFC function. We found a positive relationship between anxiety and global impulsivity and a significant direct effect of anxiety on MFG activation during emotional interference. We found no significant indirect effects between anxiety, impulsivity, negative urgency, and PFC function. Considerations surrounding data characteristics and future analyses examining relationships between clinical symptoms and PFC function in developmental samples are discussed.

These findings highlight the importance of investigating links between anxiety and dimensions of impulsivity and provide support for further research interrogating nuances in behavioral and mechanistic relationships between these constructs. This research has important implications for the reclassification of diagnostic categories that may inform prevention and treatment efforts.

CHAPTER I

Background and Introduction to Three-Paper Project

Anxiety disorders are highly prevalent, with global estimates at approximately 7.3% in adults (Stein et al., 2017). They impair functioning in multiple domains (Kessler et al., 2005). Avoidance is a hallmark of anxiety and serves to reduce internal distress, but in turn, recursively perpetuates anxiety symptoms and further impairs social, occupational, and educational functioning (Klumpp et al., 2013). Anxiety disorders often arise in early adolescence and have been linked to developmental brain differences in threat reactivity (e.g. amygdala; (Swartz et al., 2014) and inhibitory control (e.g. Prefrontal Cortex; PFC; (Clauss et al., 2016) regions. Evidence based treatments for anxiety disorders (e.g. exposure therapy) specifically target avoidance behavior (Millner et al., 2017) and appear to strengthen PFC function underlying inhibitory control of threat reactivity in the amygdala (Young et al., 2017).

Trait impulsivity, in contrast, is broadly marked by lack of inhibitory control and increased approach-based motivational processes (Beauchaine et al., 2017) and is emblematic of externalizing disorders (e.g. Attention Deficit Hyperactivity Disorder [ADHD], and substance use disorders [SUD]; (Hershberger et al., 2017; Um et al., 2019a). Mechanistic conceptualizations of impulsivity often center on the Behavioral Approach System (BAS; (Depue & Collins, 1999). Similar to findings in anxiety, impulsivity also involves deficits in top-

down PFC control, but in the case of impulsivity, there are deficits in PFC control over ventral striatal dopamine release associated with reward-driven impulsivity (Hobkirk et al., 2019).

Importantly, there are strong comorbidities between anxiety and externalizing disorders with approximately 25% of individuals with ADHD and up to 21% of individuals with substance use disorders also meeting criteria for an anxiety disorder (Grant et al., 2004; Pasche, 2012; Schatz & Rostain, 2006a). The mechanistic parallel (with possible deficits in top-down inhibitory control in both anxious and impulsive disorders), coupled with high comorbidity rates, calls into question the traditional perspective that often places impulsive approach and anxious avoidance at the opposite ends of a single continuum (Barratt, 1965; Moustafa et al., 2017). The behavioral dyscontrol of impulsivity, like anxious avoidance, is also associated with imbalance between PFC and amygdala as well as striatum (He et al., 2019; Nikolova et al., 2016). Perhaps then, these two constructs of impulsive approach and anxious avoidance do not solely function in opposition.

Clinically, we sometimes see anxiety and impulsivity appearing simultaneously, manifesting, for example, in impulsive and even dangerous behaviors undertaken to escape a fear-inducing situation (e.g., an individual with a driving phobia speeding to minimize time on the highway). In a parallel fashion, impulsive reward-driven behavior (like substance use), can also be undertaken to escape the internal distress generated by fear and anxiety (e.g. an individual with social anxiety binge drinking to cope with anxiety about socializing at a party), and the escape behavior can increase risks in attempts to minimize short-term internal distress (Borges et al., 2017). An underlying drive to escape acutely discomforting internal states plays a role in both of these examples and has been labeled “experiential avoidance” (Hayes et al., 2004). In both cases, impulsive, risky behavior is connected to anxious-avoidant drive. In fact,

experiential avoidance has been linked to both anxiety and externalizing disorders, in terms of development and maintenance (Berghoff et al., 2012; Hayes et al., 1996). It is possible, then, that anxiety and impulsivity may in fact be distinct dimensions on separate continuums that could have interacting presentations.

Because of the classical association of anxiety with internalizing disorders and impulsivity with externalizing disorders, they have mostly been studied in isolation from each other, using separate metrics that are not sensitive to their potential intersections. Current measures and conceptualizations of anxiety are not sensitive to impulsive components that might shape particular behavioral symptom presentations in anxiety disorders. Reciprocally, classic measures and conceptualizations of global impulsivity often fail to account for internalizing symptoms and anxious distress that may be present in impulsive presentations. Negative urgency is a dimension of impulsivity that captures the role of negative emotion in impulsive behavior and has been shown to be related to both internalizing and externalizing spectrum disorders (Smith & Guller, 2014). Similarly, experiential avoidance captures the urge to escape distressing internal states. These parallels suggest that negative urgency is a related construct that may capture an aspect of the behavioral consequences of experiential avoidance. We propose that experiential avoidance and negative urgency can serve as useful conduits to interrogate behavioral, mechanistic, and personality-based dimensions at the intersection of anxiety and impulsivity, allowing us to study them together so we can better understand their previously neglected intersections.

Negative urgency has strong correlations with anxiety disorders and associated avoidance behaviors (Keough et al., 2018; Malivoire et al., 2019a; Pawluk & Koerner, 2016a); but the concept emerged from work on traits like impulsivity, underlying substance use disorders

(Cyders & Smith, 2008a). Given these origins, we posit that negative urgency may be uniquely positioned to 1) better profile clinical presentations at the intersection of anxious and impulsive behavior and 2) capture a characteristic intolerance of distress associated with impulsive anxiety and experiential avoidance. In this chapter, we first summarize the movement towards empirically supported reclassifications of diagnoses within transdiagnostic frameworks and explore possible reconfigurations of anxiety and impulsivity within these models. We then outline origins and previous applications of negative urgency within externalizing disorders and summarize existing literature on the relationship between negative urgency and anxiety. In considering potential similarities in the behavioral expressions of negative urgency and experiential avoidance in managing anxiety, we also present a theoretical profile of impulsive anxiety marked by intolerance of distress. With this presentation in mind, we identify specific aims for empirical testing of relationships between negative urgency, anxiety, and experiential avoidance, as well as diagnostic and clinical implications.

Transdiagnostic Approaches to Anxiety and Impulsivity

Behavioral symptom-based distinctions between anxiety and externalizing disorders within the DSM model have had some utility in efficient classification of psychological symptoms. However, these distinctions have resulted in separations in treatments and research that may not target multifaceted presentations of these conditions (Dalgleish et al., 2020). As such, researchers have developed transdiagnostic frameworks to reconcile mechanistic similarities and issues of comorbidity across categorical diagnostic classifications and heterogeneity within individual disorder categories. Forgoing the boundaries of categorical diagnosis allows researchers more flexibility to investigate areas of previously unexplained comorbidity.

Transdiagnostic approaches allow for a more nuanced examination of where anxiety and impulsivity overlap and diverge. The Research Domain Criteria (RDoC; Cuthbert & Kozak, 2013; Insel et al., 2010) aims to use a growing understanding of the brain to help us discover neurally instantiated dimensions of psychosocial functioning. Impulsivity has been implicated in many RDoC domains, including Negative Valence Systems (e.g. anxiety) for its role in fear learning, impulsive reactions to negative valence, and hyperarousal in trauma (Brooks et al., 2017). Impulsivity has also been separately integrated within the Cognitive Systems Domain (e.g., cognitive control) due to the involvement of dopaminergic PFC pathways associated with impulsivity in motor inhibition tasks.

The Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017), takes a dimensional, bottom-up approach to classifying psychopathology with six levels ranging from symptom-level indicators of vulnerability to a universal general vulnerability to psychopathology. Anxiety-based disorders are seen in the Internalizing Spectra, which accounts for ruminative negative affectivity. Impulsivity is reflected in the Disinhibited Externalizing Spectra, but is associated with syndromes seen outside of the Externalizing dimension, such as the Eating Pathology Subfactor (i.e. purging behavior; Fischer et al., 2008) and Distress Subfactor (Borderline personality traits; Sebastian et al., 2013) within the Internalizing Spectra. Given broad representation of impulsivity crossing domains in RDoC and overarching Super Spectra in HiTOP, researchers have investigated it as a functional interpretation of the general underlying vulnerability for psychopathology, or *p factor* (Carver et al., 2017; Johnson et al., 2013).

Outlining these possible reclassifications of impulsivity through transdiagnostic frameworks has two effects. First, it illustrates how transdiagnostic approaches allow for greater

flexibility in reconceptualizing the ways these constructs may interact, reflecting varied and nuanced clinical presentations. Second, in demonstrating how impulsivity may fit into multiple domains within a transdiagnostic framework (e.g. negative valence system, cognitive systems in RDoC), we reveal the breadth of processes encompassed by this construct.

A lack of consistency in definitions of impulsivity has undermined efforts to establish consistent relationships between impulsivity and anxiety. Empirical pursuits to examine this relationship have used common probes for particular dimensions of impulsivity such as motor inhibition, emotional reactivity, or reward salience (Dalley et al., 2011). However, these particular dimensions are often conflated with the broader construct of impulsivity, and thus, it has remained unclear which specific facets of impulsivity are positively associated with anxiety. In light of this longstanding issue, specific dimensions of impulsivity have been clarified. Negative urgency is one dimension of impulsivity that has been associated with both internalizing and externalizing disorders that captures impulsive behavior specifically enabled by heightened negative emotion. Negative urgency provides a narrow and more specific conduit to explore the intersection of anxiety and impulsivity. A more precise understanding of the dimensions of impulsivity that may drive relationships with anxiety will then allow for reconceptualization of these constructs within emerging transdiagnostic models.

Negative Urgency

The dimensional (Negative) Urgency, Premeditation, Perseverance, Sensation Seeking, and Positive Urgency (UPPS-P) Model (Whiteside & Lynam, 2001) was developed to better distinguish facets of impulsivity associated with psychopathology and standardize the construct across the field (Cyders & Smith, 2008a). Trait negative urgency is characterized within the Five-Factor Model of Personality (Costa & McCrae, 1990) by high neuroticism, low

conscientiousness, and low agreeableness. Although high neuroticism is a key facet of both negative urgency and mood and anxiety disorders, negative urgency has previously been distinguished from these internalizing presentations due to differences in factors of consciousness and agreeableness (Smith & Guller, 2014). Despite these potential differences across dimensions of personality, negative urgency is the dimension of impulsivity within the UPPS-P model that has been explored most thoroughly in the context of anxiety.

Little research has focused on relationships between anxiety and non-emotional dimensions of impulsivity (e.g. lack of premeditation, lack of perseverance). Positive urgency, or rash action in positive emotional states, has predominantly been associated with bipolar disorder (Muhtadie et al., 2014) and reward-driven psychopathology (e.g. pathological gambling, SUD; (Cyders & Smith, 2008b; Smith & Cyders, 2016a). Negative urgency is also strongly implicated in SUD and other externalizing disorders (e.g. Antisocial Personality Disorder), and measured in early adolescence, it is predictive of substance use behaviors (e.g. smoking, alcohol use) later in life (Smith & Cyders, 2016a). Negative urgency has also been linked to bulimia, which is highly comorbid with internalizing disorders such as anxiety (Fischer et al., 2008; Kaye et al., 2004). These relationships between trait negative urgency and both anxiety and comorbid disorders suggest that negative urgency is well positioned to serve as a conduit to explore impulsive anxiety.

Specific to anxiety related experiences, negative urgency can capture rash action driven by an extreme negative emotional state (anxious distress), potentially in spite of long-term consequences. Rash behaviors in a negative state may serve the perceived need to quickly escape an anxiety provoking situation (e.g. someone with a driving phobia speeding to exit a busy highway rather than driving with excess caution to avoid a crash) or directly run counter to an

intention to resolve anxiety by escalating negative situations (e.g. item 50 of the UPPS-P: “In the heat of an argument, I will often say things that I later regret”; Whiteside & Lynam, 2001). These varying presentations of negative urgency share a central theme of maladaptive responses in moments of distress, and these behavioral presentations may reflect underlying neural mechanisms associated with deficits in inhibitory control.

Patterns of neural function associated with negative urgency parallel those reported in the context of both anxiety and impulsivity, with deficits in top-down (i.e. PFC) regulation of inhibitory control. Neural activation associated with negative urgency has predominantly been examined in the context of SUD (Cyders et al., 2015a; Um et al., 2019b); therefore, research has centered on relationships between negative urgency and activation in regions implicated in reward salience and emotional reactivity. Specifically, reduced activation in PFC regions associated with emotion modulation and cognitive control (e.g. orbitofrontal cortex, OFC; anterior cingulate cortex; ACC; (Smith & Cyders, 2016a) and increased activation in emotion processing regions (e.g. amygdala and striatum) have been associated with negative urgency. These previous findings on negative urgency closely mirror the dual process model of neural activation associated with heightened emotional reactivity in anxious individuals. However, as a dimension of impulsivity, negative urgency may also be associated with broader deficits in inhibitory control not specific to emotional processes. Negative urgency has in fact been associated with reduced activation in regions associated with non-emotional inhibitory control (e.g. inferior frontal gyrus; IFG; Wilbertz et al., 2014). Both behavioral outcomes and patterns of neural activation associated with negative urgency- predominantly in the context of SUD- appear to reflect similarities with both anxiety and broader impulsivity. These similarities suggest that negative urgency may be well-positioned to capture nuances of impulsive profiles of anxiety.

Anxiety and Negative Urgency

Thus far, research has explored the relationship between negative urgency and anxiety in individuals with and without substance use. In the context of substance use, this relationship may reflect a characteristic lack of distress tolerance. Thus far, three studies have demonstrated that the relationship between anxiety sensitivity (i.e. fear of sensations and behaviors associated with anxiety; (Guillot et al., 2014) and substance use (e.g. tobacco, alcohol, cannabis) was mediated by negative urgency (Guillot et al., 2014; Kauffman et al., 2018; Keough et al., 2018), although one study examining this relationship did not find relationships between anxiety sensitivity or negative urgency and cannabis use (Sofis et al., 2020). It is possible then, that trait negative urgency may reflect, or be a consequence of, an acute intolerance of distress that could manifest through maladaptive coping behaviors (e.g., substance use).

The relationship between negative urgency and anxiety without the presence of SUD has focused on cognitive factors such as intolerance of uncertainty (Malivoire et al., 2019b; Pawluk & Koerner, 2016b), depressive symptoms (Altan-Atalay & Zeytun, 2020; King et al., 2021), and future oriented thinking (Altan-Atalay et al., 2020). Four such studies have reported a positive relationship between anxiety and negative urgency. One study examining relationships between negative urgency and internalizing symptoms showed no relationship between anxiety and negative urgency, but did demonstrate a positive relationship between negative urgency and depressive symptoms (Altan-Atalay & Zeytun, 2020). Through trait negative urgency, we may characterize impulsive profiles of anxiety by reactivity to distress and the use of often maladaptive short-term coping strategies. A potential avenue for continuing research may be probing the relationship between negative urgency and anxiety-based experiential avoidance.

Experiential Avoidance

Experiential avoidance centers on acute internal distress (physiological and psychological) rather than fears of situational, behavioral consequences and contingencies imposed by the outside world (Hayes et al., 2004). Experiential avoidance is associated with anxiety and stress disorders such as Generalized Anxiety Disorder (Mellick et al., 2019; Venta et al., 2012), Panic Disorder (White et al., 2006), and PTSD (Filipas & Ullman, 2006). Like negative urgency, experiential avoidance is also associated with SUD (Brem et al., 2017a; Cavicchioli et al., 2020; Luoma et al., 2020) echoing the characteristic of discomfort with distress captured by the relationship between negative urgency and the related but distinct construct of anxiety sensitivity (Kämpfe et al., 2012).

The link between experiential avoidance and negative urgency has not yet been explicitly studied; however, they are associated with similar disorders (e.g. anxiety, SUD). Furthermore, trait negative urgency has been associated with the use of short term coping mechanisms to alleviate distress associated with anxiety as opposed to long term, "slow-acting" strategies (e.g. emotion regulation (King et al., 2018a), supporting the possibility that negative urgency and experiential avoidance are related. For example, experiential avoidance and negative urgency have both been linked to self-harm without suicidal intention (e.g. non-suicidal self-injury; NSSI), a short term behavioral coping strategy that serves to divert psychological distress rather than resolve it in the long term (Nielsen et al., 2017; You et al., 2016).

If experiential avoidance is associated with distress caused by physiological and psychological facets of anxiety, and trait negative urgency is an inability to tolerate that distress which triggers impulsive subsequent action to escape the distress, then high levels of both may reinforce a feedback loop of short-term rash action taken to avoid negative emotional states.

Empirically examining the relationship between experiential avoidance and negative urgency will provide additional nuance in understanding profiles of anxiety.

Transdiagnostic and Clinical Implications

In outlining existing evidence for relationships between anxiety and impulsivity and their more nuanced subcomponents of experiential avoidance and negative urgency, we begin to uncover a potential theoretical profile of impulsive anxiety marked by high emotional distress, low distress tolerance, and reinforcement of short-term maladaptive coping strategies. This behavioral depiction of impulsive anxiety is likely supported by mechanistic similarities in failure of top-down PFC control over emotion processing centers (e.g. amygdala, striatum), as well as impaired motor inhibition, both of which have been associated with negative urgency. Greater specificity in defining dimensions of impulsivity provides an opportunity to clarify the role that impulsive action plays in anxiety, and negative urgency may be a construct well-positioned to add nuance to this relationship. A critical next step in exploring this possibility is to empirically test our hypothesized theoretical model of the constellation of relationships between anxiety, negative urgency, experiential avoidance, and non-emotional dimensions of impulsivity. Probing behavioral, symptom-driven relationships between these constructs, and patterns of underlying neural function, may allow for refinement in positioning anxiety and impulsivity within transdiagnostic frameworks.

The flexibility of transdiagnostic frameworks has provided researchers an outlet to consider impulsivity, and more specifically, negative urgency, not as a secondary piece of an individual's experience with anxiety related disorders, but as a driving and maintaining factor. Theoretically, the biologically based vulnerability to high emotional reactivity in tandem with a propensity to approach alleviatory action, seen in the foundational levels of the RDoC

conceptualization, interacts with biopsychosocial factors (e.g. family history of psychological conditions, environmental stressors) to shape expressions of anxiety related psychopathology (Cuthbert & Kozak, 2013; Insel et al., 2010). For some, as anxiety related psychopathology emerges, rash approach to risky action to avoid negative emotion results in quick alleviation of the negative emotion, affirming negative urgency as driving behaviors that serve to avoid short-term distress. Thus, as an individual continues to experience negative urgency, experiential avoidance and anxiety related psychopathology are maintained. Further investigation of negative urgency as a driving and maintaining mechanism of anxiety will provide clinicians and researchers with a more nuanced understanding of the different presentations of anxiety. The results of this work could lead to improved understanding of the most effective treatment approaches based on the nuanced relationships between anxiety and impulsivity.

Specific Aims

This dissertation examined how negative urgency, experiential avoidance, and neural function relate to both anxiety and impulsivity, and identified neural targets for future research to identify shared and distinct inhibitory control circuits central to anxious and impulsive processes. By better understanding the relationship between anxiety and impulsivity through negative urgency, we may also begin to examine relationships between impulsivity and anxiety-based avoidance, which could shape future behavioral treatments for anxiety. The following chapters focus on empirical studies to investigate these links as follows:

Chapter 2: We used self-report data collected from adult participants via Amazon Mechanical Turk (MTurk) to examine the indirect effect of anxiety on experiential avoidance through negative urgency and global impulsivity.

Chapter 3: We examined neuroimaging data during the Emotional N-Back (EN-Back) Task and Stop Signal Task (SST) from the well-established Adolescent Brain Cognitive Development (ABCD) Study to examine patterns of shared and distinct prefrontal cortex (PFC) activation during tasks associated with emotional and non-emotional forms of inhibitory control (emotional interference and motor inhibition respectively).

Chapter 4: Finally, we examined relationships between the unique patterns of PFC activation specific to emotional interference and motor inhibition identified in the analyses for Chapter 3 and self-reported measures of anxiety and impulsivity.

CHAPTER II

Relationships between Anxiety, Impulsivity, Experiential Avoidance, and Negative Urgency

Anxiety and impulsivity frequently co-occur and may present together in varied forms, yielding different targets for behavioral intervention. Trait measures have been developed to parse out specific dimensions of impulsivity based on emotional and personality factors; these dimensions may be differentially related to anxiety, providing nuance in profiles of co-occurring anxiety and impulsivity. On the surface, individuals may exhibit similar reactionary avoidance; however, these behaviors may serve different underlying anxious and impulsive motivations, which could benefit from different treatment approaches. For example, experiential avoidance, or efforts to alleviate emotional and physiological distress, may follow maladaptive reactionary responses to emotion (i.e., urgency) or lack of follow through on meaningful tasks (i.e., lack of perseverance). The aim of this chapter is to model relationships between anxiety and dimensions of impulsivity and examine relationships between their interactions and the outcome of experiential avoidance.

Although multiple dimensions of impulsivity have been linked to psychopathology, negative urgency is a dimension of particular interest because of its distinct relevance to anxiety and experiential avoidance. Due to its relevance to internalizing symptom profiles, emotion-based dimensions of impulsivity have been of major interest in clinical research. Urgency, as operationalized through the UPPS framework of impulsivity, captured impulsive behavior linked to increased emotional arousal, regardless of valence (Whiteside & Lynam, 2001). Positive

urgency was distinguished as a dissociable facet of impulsivity associated with positive rather than negative emotions due to differences in relationships with psychopathology, namely risky gambling, and bipolar disorder (Billieux et al., 2010; Carver & Johnson, 2018). Negative urgency, by contrast, has been more strongly associated with anxiety and has been shown to mediate the relationship between anxiety sensitivity and substance use (Guillot et al., 2014; Kauffman et al., 2018; Keough et al., 2018). Thus, negative urgency may be the dimension of impulsivity best positioned to examine a profile of anxiety and impulsivity marked by distress intolerance and avoidance.

Similarly, experiential avoidance is well positioned to capture the internal distress intolerance that may be central to profiles of anxiety and impulsivity driven by avoidance. Experiential avoidance is rooted in literature surrounding Acceptance and Commitment Therapy (ACT) and has been linked to several anxiety disorders (Filipas & Ullman, 2006; Mellick et al., 2019; Venta et al., 2012; White et al., 2006). Other forms of avoidance may reflect premeditated efforts to avoid situationally specific consequences, characteristic of overregulated presentations of anxiety (e.g., avoid driving on the highway for fear of an accident). By contrast, experiential avoidance manifests as short-term reactionary efforts to escape emotional distress, more consistent with impulsive presentations of anxiety (e.g., speeding to the nearest exit to leave the highway and alleviate anxious distress more quickly). Associations with impulsivity are evidenced by relationships between experiential avoidance and behaviors associated with externalizing disorders. In addition to substance use, prior literature has linked experiential avoidance to risky sexual behaviors (Brem et al., 2017b), eating pathology (e.g, binge eating; Della Longa & De Young, 2018), and non-suicidal self-injury (NSSI; Allen & Hooley, 2019). Furthermore, these relationships parallel those documented between similar short term, reflexive

coping behaviors (e.g., distraction, avoidance, suppression) and negative urgency (King et al., 2018b). To our knowledge, only one study has examined experiential avoidance and negative urgency together, and reported a positive correlation between experiential avoidance and negative urgency in a sample of individuals with disordered eating (Rodrigues et al., 2022). Within the context of anxious pathology, experiential avoidance itself, rather than associated coping behaviors, serves as a target for treatment.

Behavioral disinhibition and emotionally driven negative urgency are intuitively associated with anxiety. However, non-emotional dimensions of impulsivity such as motor inhibition have also been associated with anxiety such that greater anxiety is associated with more errors on inhibition tasks (Grillon et al., 2017; Iijima et al., 2018). Furthermore, hyperactivity (a hallmark of ADHD, a classically impulsive disorder) is commonly associated with greater anxiety (Prevatt et al., 2015; Schatz & Rostain, 2006b). An agnostic exploration of relationships between these constructs (i.e., including additional dimensions of impulsivity) may ultimately provide evidence that, regardless of emotional state, impulsive dimensions contribute to anxious avoidance.

Specific Aims

We aimed to model the indirect effects of trait anxiety on experiential avoidance through impulsivity. In particular, we aimed to distinguish the unique effects of negative urgency on this relationship from additional dimensions of impulsivity. Positive correlations between anxiety and the outcome of experiential avoidance have been well established and we expected our results to mirror previous findings. Research has also demonstrated strong comorbidities between anxiety and multiple dimensions of impulsivity. Therefore, we anticipated that both mediators of negative urgency and global impulsivity would be positively related to anxiety based on previous

research. Given parallels in findings between negative urgency and experiential avoidance, we also anticipated that these constructs would be positively correlated in our model. As a facet of impulsivity relevant to negative emotion, we hypothesized that anxiety would have a stronger indirect effect on experiential avoidance through negative urgency than global impulsivity.

Findings from this research will inform future studies examining impulsive presentations of anxiety. If trait negative urgency explains significant variance in the relationship between anxiety and experiential avoidance, further empirical studies can examine neural and behavioral facets of impulsive presentations of anxiety through negative urgency.

Method

Participants

Due to the constraints of data collection during the COVID-19 pandemic, the use of a well-known internet-based crowdsourcing platform, Amazon MTurk, allowed for access to a large, nationwide sample. Inclusion criteria for the study included adults between the ages of 18-55 based in the United States (M_{age}= 32.05, SD_{age}= 7.19; 79.78% White; 59.34% Male; 15.45% Sexual Minority; 43.11% < $50,000 Income; see Table II.1 for full demographic information).

Table II.1
Demographic Characteristics of the Sample (n = 589; M_{age} = 32.05, SD_{age} = 7.19)

	n	%
Race/Ethnicity		
White/ Caucasian	470	79.78%
Black/ African American	41	7.04%
American Indian/ Alaskan Native	4	0.72%
Asian	45	7.58%
Native Hawaiian/ Pacific Islander	1	0.18%
Latinx	26	4.33%
Multiple/Other	2	0.36%
Sex		
Assigned Male at Birth	350	59.34%
Assigned Female at Birth	239	40.66%

Gender		
Cisgender Male/Man	343	58.22%
Cisgender Female/Woman	235	39.90%
All Gender Minorities	11	1.88%
Sexual Orientation		
Heterosexual	498	84.55%
All Sexual Minorities	91	15.45%
Income		
≤ $10,000	16	2.73%
$10,000-$29,999	100	17.04%
$30,000-$49,999	137	23.34%
$50,000-$69,999	122	20.78%
$70,000-$89,999	44	7.50%
$90,000-$149,999	51	8.69%
≥ $150000	23	3.92%

Participants were paid consistent with United States federal minimum wage directly through the MTurk system. Informed consent was obtained prior to data collection and the study received an exempt status by the Institutional Review Board at the University of Michigan.

Rationale for the Present Sample

Data were collected from three cohorts (May 2020, June 2021, and October 2021) of adults. Although anxiety and impulsivity are often explored for their clinical significance, we proposed that both constructs are present on separate continuums across clinical and subclinical levels. Given intentions to establish proof of concept for relationships between anxiety, experiential avoidance, and negative urgency, it was important to first capture these relationships across the full spectrum of scores in an adult sample. Thus, participants were not specifically selected based on these dimensions.

MTurk Data Quality. Data collection via platforms such as MTurk has become more commonplace due to benefits of more rapid and widespread sampling and greater control over research design than use of secondary datasets. However, limitations of MTurk samples have

been well documented (Walters et al., 2018). Therefore, several measures were taken to ensure data quality during data collection and processing, consistent with prior literature (Agley et al., 2022; Parnarouskis et al., 2020a). At data collection, Human Intelligence Task (HIT) Qualifications were included to restrict participants to 1) MTurk workers with prior HIT approval rates of 90% or greater (i.e., percentage of tasks on MTurk previously completed by workers that were approved for their quality by requesters) and 2) MTurk workers based in the United States. In addition, "catch questions" were included within surveys to identify possible bot accounts and inattentive participants. The first catch question ("Please enter the last four digits of your MTurk ID") was completed by all three cohorts. Two additional questions were included during data collection for the third cohort to provide further indicators of data quality ("Who was the first president of the United States?", previously used in (Parnarouskis et al., 2020b); and "Pick 'Sometimes' for this question.", included within a survey with Likert scale answer choices.

Data were included in analyses if participants completed more than 90% of all surveys; if participants were unique (based on MTurk ID; duplicates were manually evaluated and the first duplicate was retained in analyses if all other quality checks were passed); and if completion time was between six minutes and one hour. Participants from the first and second cohorts were included in analyses if they passed the catch question (i.e. entered no more or less than four digits of an MTurk ID). Prior evidence suggests that free response items as attention checks provide the most definitive evidence of computer generated, rather than human responses (Dennis et al., 2019). Therefore, for the third cohort, we first evaluated responses to the added free response attention check (i.e. first president of the United States) to screen out AI workers. The most common incorrect answer was a variant of "John Hanson", and participants who

responded with this answer were excluded. If a different incorrect answer was given, responses to the other two catch questions were evaluated to eliminate potential inattentive human participants. Participants from the third cohort were retained if they passed at least 2 of the 3 attention checks. There were no differences in means or completion times between cohorts after data cleaning. The final sample included data from 589 participants across the three cohorts (Retention Rates: Cohort 1, 55.56%; Cohort 2, 61.58%; Cohort 3, 70.88%; Total, 64.30%).

Procedures

Assessment. Participants were instructed to complete a series of behavioral surveys on self-reported symptoms of anxiety, trait impulsivity, and experiential avoidance (For measure characteristics, see Table II.2).

Table II.2
Measure Characteristics

Measure	Items	Subscales of Interest (# of Items)	Scale of Scoring
Barratt Impulsiveness Scale – 11 (BIS-11)	30	Global Impulsivity (30)	Rarely/Never (1) – Always (4)
The Urgency, Premeditation, Perseverance, Sensation Seeking, and Positive Urgency Impulsive Behavior Scale (UPPS-P)	59	Negative Urgency (12) Lack of Premeditation (11) Lack of Perseverance (10) Sensation Seeking (12) Positive Urgency (14)	Agree Strongly (1) – Disagree Strongly (4)
The State Trait Anxiety Inventory (STAI)	40	State (20) Trait (20)	Rarely/Never (1) – Almost Always (4)
Acceptance and Action Questionnaire – II (AAQ-II)	7	Total Score (7)	Never True (1) – Always True (7)

Barratt Impulsiveness Scale-11 (BIS-11; Patton et al., 1995)**.** The BIS-11 is a well-validated measure of global impulsivity that consists of thirty statements regarding impulsivity that

participants responded to on a Likert-type scale. Ten items were reverse scored, and subscale scores (i.e., attentional, motor, and non-planning impulsiveness) and total sum scores were calculated as sums of all items as a primary measure of global impulsivity. Total scores can range from 30 to 120. Scores below 52 indicate excessive control, scores between 52-71 indicate normal impulsiveness, and scores of 72 or greater indicated high trait impulsivity. The BIS-11 has been previously demonstrated to have high internal consistency (Stanford et al., 2009). Within the present sample, internal consistency for the total score was good with Cronbach's alpha (α) = 0.88. Internal consistency for subscale scores ranged from $\alpha = 0.44$ (Perseverance) to $\alpha = 0.79$ (Motor) (Note: Cronbach's Alphas were calculated using unstandardized raw values).

The Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behavior Scale (UPPS-P; Whiteside & Lynam, 2001). The UPPS-P is a well validated measure of dimensional impulsivity that probes five dimensions of impulsivity (Negative Urgency, score range: 12-48; Lack of Premeditation, score range: 11-44; Lack of Perseverance, score range: 10-40; Sensation Seeking, score range: 12-48; and Positive Urgency, score range: 14-56) that map on to a higher order three factor structure of Sensation Seeking, Lack of Conscientiousness (including Lack of Premeditation, Lack of Perseverance), and Urgency (including Positive and Negative Urgency). Items for each subscale are summed separately. For the current sample, internal consistency ranged from $\alpha = 0.86$ (Lack of Premeditation to $\alpha = 0.96$ (Positive Urgency).

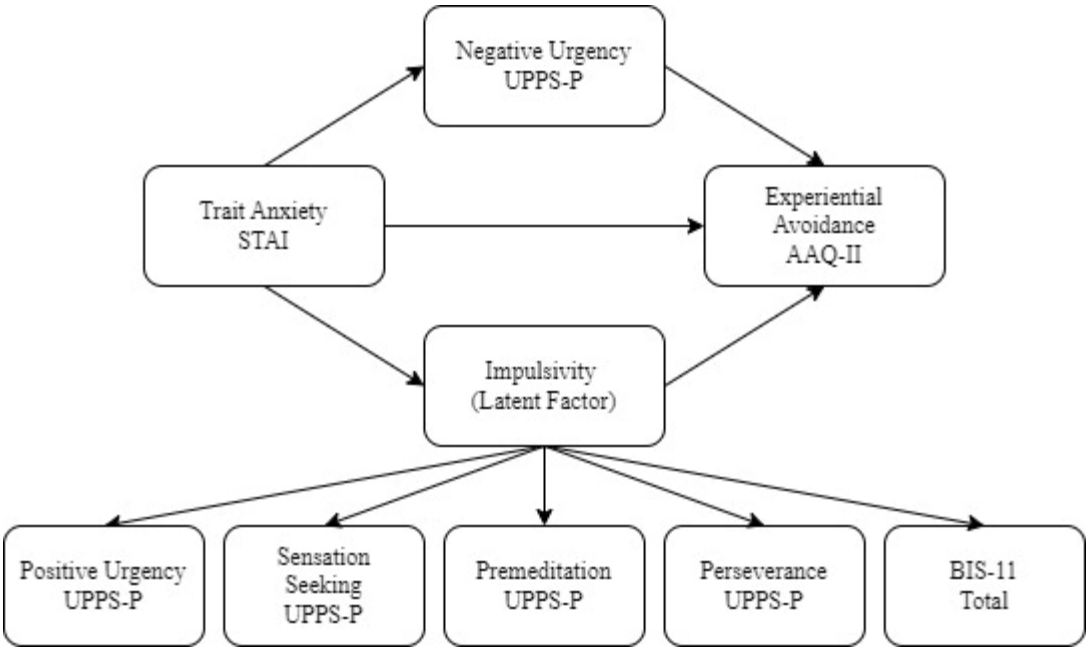
State Trait Anxiety Inventory (STAI; Spielberger, 1983). The STAI includes two subscales of trait (20 items) and state (20 items) anxiety that participants responded to on a Likert-type scale. Items for each subscale were summed separately with scores on each measure ranging from 20-80 and scores above 40 indicating significant anxiety. Both trait and state

subscales of the STAI have previously demonstrated high internal consistency for adult participants (Spielberger, 1983). The STAI is one of the most widely used measures of both state and trait level anxiety and has been used across a variety of samples (Elwood et al., 2012). The primary subscale for these analyses will be the Trait subscale. In the present sample, internal consistency for the trait subscale was $\alpha = 0.83$ and the state subscale was $\alpha = 0.89$.

The Acceptance and Action Questionnaire-II (AAQ-II;(Bond et al., 2011). The AAQ-II is a seven-item measure of experiential avoidance that participants responded to using a Likert-type scale. Scores range from 7 to 49, with scores of greater than 24-28 indicating clinically significant distress. The AAQ-II has previously demonstrated high internal consistency (Bond et al., 2011; Fledderus et al., 2012); in the present sample, internal consistency was $\alpha = 0.94$.

Analytic Strategy. To test the primary hypotheses, a confirmatory factor model (see Figure II.1 for conceptual model) was specified to examine relationships between trait anxiety and experiential avoidance through both the UPPS-P negative urgency subscale, and a latent global factor of impulsivity comprised of indicators from the BIS-11 and UPPS-P (i.e. UPPS-P subscales excluding negative urgency, BIS-11 total score).

Figure II.1
Conceptual diagram of confirmatory factor model



The structure for this latent factor of impulsivity was determined via an exploratory factor analysis (EFA) using an oblique geomin rotation. The EFA was conducted using best practices for EFA outlined by Watkins, (2018). Results of this EFA with best fit and conceptual rationale informed the structure of the latent factor of impulsivity that was included in a subsequent confirmatory factor analysis (CFA).

The hypothesis-driven CFA included the UPPS-P negative urgency subscale and the latent factor of global impulsivity (informed by the EFA) as two distinct mediators of the relationship between anxiety and experiential avoidance. Experiential avoidance served as the primary outcome variable that was regressed onto the independent variable of trait anxiety. Experiential avoidance was also regressed on both mediators and covariance was established between mediators. The CFA was conducted using maximum likelihood estimation, and the 95%

confidence interval for parameter estimates in the double mediation model was computed using percentile bootstrap procedure with 10,000 random draws.

Results

Descriptive Statistics. Descriptive statistics between all variables were conducted in MPlus Version 8.4 (Muthén & Muthén, 1998) and are provided in Table II.3. Sum scores were normally distributed and ranged from subclinical to clinical levels. All data were standardized (Z scores computed) to account for differences in scales of the psychometric measures. Intercorrelations between measures of impulsivity included in the EFA ranged from r = -0.05 to r = 0.78 (Table II.4).

Table II.3
Descriptive Statistics

Measure	Mean	SD	Min	Max	Kurtosis	Skew	α
UPPS-P							
1. Negative Urgency	26.62	8.84	12.00	47.00	-0.71	0.36	0.92
2. Positive Urgency	27.53	11.47	14.00	47.00	-0.41	0.77	0.96
3. Sensation Seeking	28.23	8.84	12.00	48.00	-0.81	0.10	0.91
4. Lack of Premeditation	19.62	5.74	11.00	44.00	2.33	1.17	0.86
5. Lack of Perseverance	19.49	5.93	10.00	38.00	-0.16	0.47	0.87
BIS-11							
6. Total	60.12	12.81	34.00	105.00	-0.13	0.42	0.88
AAQ-II							
7. Total	23.61	11.46	7.00	49.00	-1.08	0.32	0.94
STAI							
8. State	40.69	10.71	26.00	74.00	-0.20	0.66	0.89
9. Trait	45.56	9.17	29.00	71.00	-0.63	0.26	0.83

Table II.4
Zero-order Correlation Matrix

Measure	1	2	3	4	5	6	7	8
UPPS-P								

1. Negative Urgency	-							
2. Positive Urgency	0.78	-						
3. Sensation Seeking	0.35	0.52	-					
4. Lack of Premeditation	0.35	0.34	0.19	-				
5. Lack of Perseverance	0.47	0.31	-0.05	0.46	-			
BIS-11								
6. Total	0.71	0.64	0.27	0.49	0.58	-		
AAQ-II								
7. Total	0.60	0.45	0.08	0.11	0.46	0.57	-	
STAI								
8. State	0.48	0.31	-0.03	0.15	0.46	0.50	0.70	-
9. Trait	0.39	0.46	0.19	0.14	0.16	0.41	0.49	0.40

Exploratory Factor Analysis. A parallel analysis indicated two factors from the data above the 95th percentile estimates based on 100 simulated datasets (See Figure II.2 for screeplot). Therefore, a two-factor solution was specified for an EFA with maximum likelihood extraction with an oblique rotation. The EFA revealed that positive urgency and sensation seeking loaded strongly (i.e. greater than 0.35; Hair Jr et al., 2009) on one factor; while lack of perseverance, lack of premeditation, and the total BIS-11 score loaded strongly on a second factor (For factor loadings in the EFA, see Table II.5). According to established standards of

good fit (Hu & Bentler, 1998, 1999), this model moderately fit the data (χ^2 = 16.84, df = 1, p < 0.001; RMSEA = 0.16; SRMR = 0.02; CFI = 0.98; TLI = 0.83).

Figure II.2
Scree plot for exploratory factor analysis

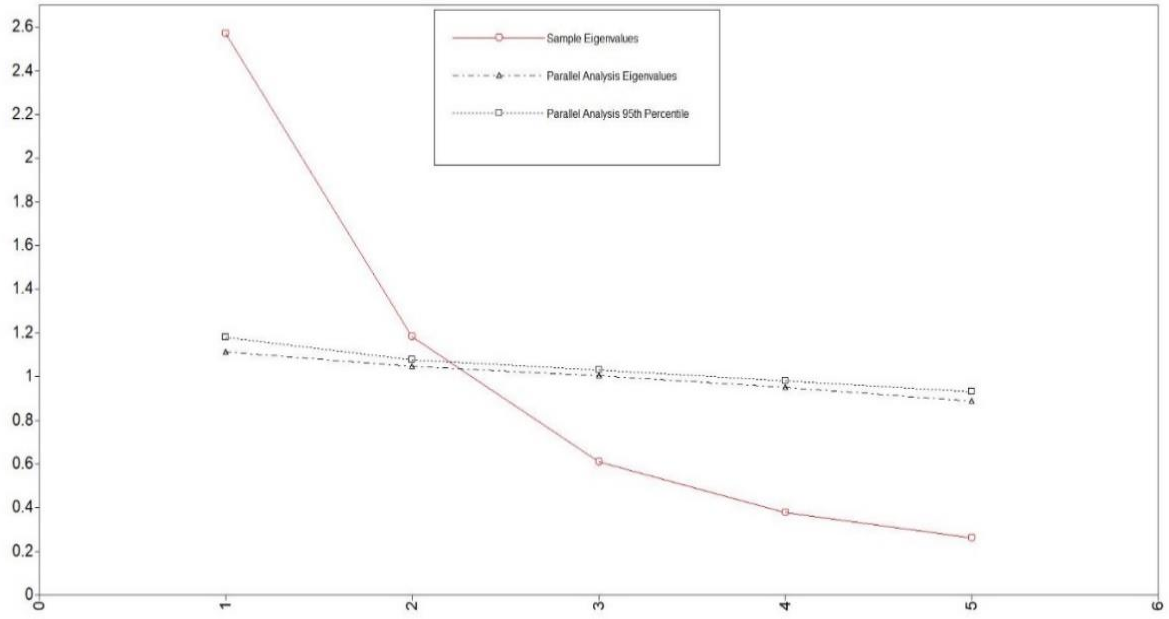


Table II.5
Factor loadings from exploratory factor analysis

	EFA	
	Factor 1	Factor 2
Positive Urgency	0.60*	0.45*
Sensation Seeking	0.71*	-0.002
Lack of Premeditation	0.002	0.59*
Lack of Perseverance	-0.31*	0.83*
BIS-11	0.17*	0.79*

Note. EFA = exploratory factor analysis. Oblique, geomin rotation used in EFA; factor correlation *r* = 0.30.

Confirmatory Factor Analysis. In keeping with our hypothesis-driven conceptual model including negative urgency and a latent factor of global impulsivity as mediators, a preliminary CFA was conducted using one latent factor for impulsivity (i.e., intercorrelations between

indicators not specified; factor mean set at 0 and variance set at 1) and negative urgency as mediators of the relationship between anxiety and the outcome variable of experiential avoidance. This model poorly fit the data (χ^2 = 1062.88, df = 18, p < 0.001; RMSEA = 0.31; SRMR = 0.18; CFI = 0.61; TLI = 0.39).

In an effort to improve model fit, a CFA was conducted with a bifactor model of impulsivity (informed by the results of the EFA) and negative urgency as mediators. Inter-factor correlations were specified between positive urgency and sensation seeking, and between lack of perseverance, premeditation, and the BIS-11 total for a bifactor model of latent global impulsivity. This resulted in an improvement in fit (χ^2 = 402.16, df = 14, p < 0.001; RMSEA = 0.22; SRMR = 0.11; CFI = 0.86; TLI = 0.71), but the model still did not meet conventional standards for a good fit. Results of this CFA are detailed in Table II.6 and graphically in Figure II.3.

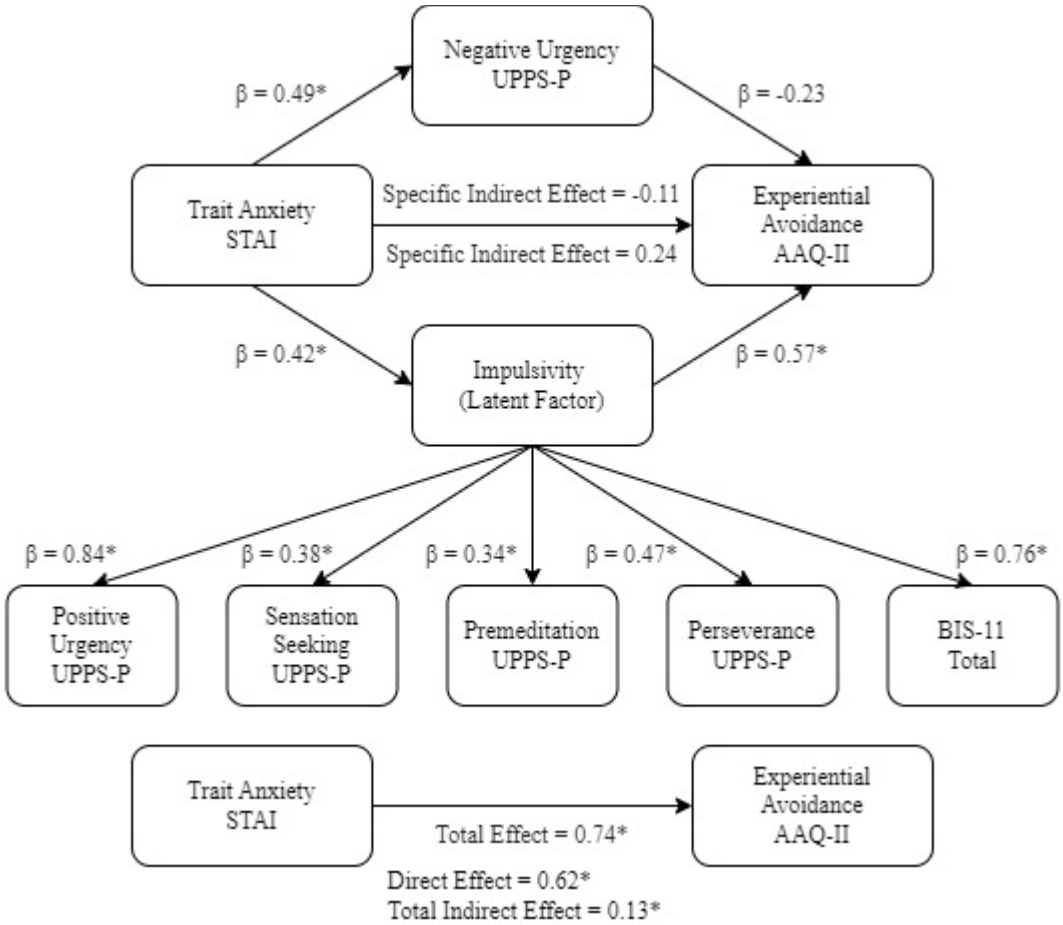
Table II.6
Results from confirmatory factor model

	STD	95% CI
Negative Urgency		
STAIT → NU	0.49	[0.42, 0.56] *
NU → EA	-0.23	[-0.77, 0.04]
Indirect Effect	-0.11	[-0.37, 0.02]
Impulsivity Latent Factor		
STAIT → IMP	0.43	[0.31, 0.54] *
IMP → EA	0.57	[0.30, 1.11] *
Indirect Effect	0.24	[0.11, 0.52] *
Direct & Total Effects		
Direct Effect	0.62	[0.52, 0.70] *
Total Indirect Effect	0.13	[0.06, 0.21] *
Total Effect	0.75	[0.71, 0.78] *

Note: STAIT = Trait Anxiety; NU = Negative Urgency; EA = Experiential Avoidance; IMP = Impulsivity Latent Factor; 95% CI = 95% confidence intervals for standardized coefficients; STD = standardized coefficients; * = p < 0.05. 95% confidence intervals derived via bias corrected bootstrap procedure with 10,000 random draws.

There was a significant direct effect of trait anxiety on experiential avoidance (b = 0.62). Trait anxiety was a significant predictor of both negative urgency (b = 0.49) and the latent factor of global impulsivity (b = 0.43). Negative urgency was not a significant predictor of experiential avoidance (b = -0.23); however, the global impulsivity factor did significantly predict experiential avoidance (b = 0.57). The indirect effect of trait anxiety on experiential avoidance through negative urgency was not significant (b = -0.11). The indirect effect through global impulsivity was small (b = 0.24) and approaching significance (p = 0.06). Both the total indirect effect (b = 0.13) and the total effect (b = 0.74) were statistically significant.

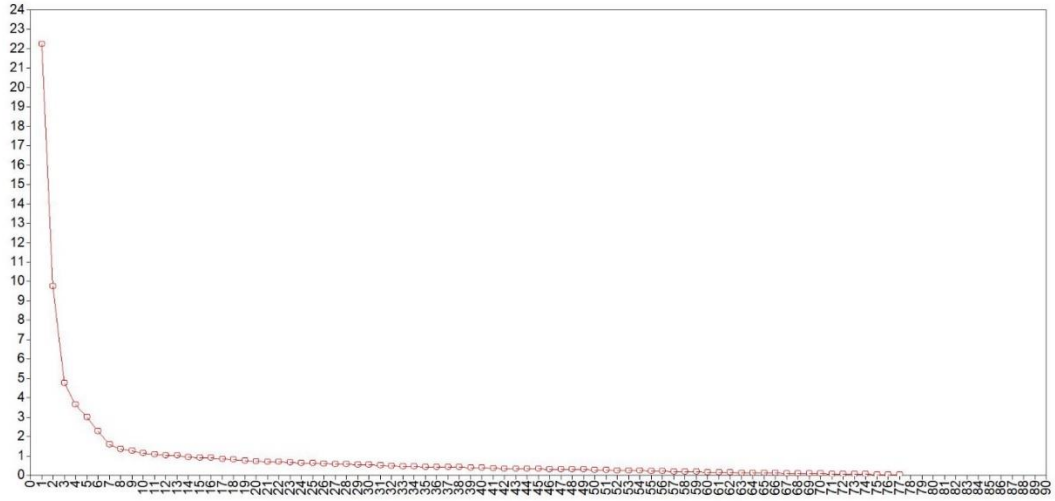
Figure II.3
Graphical model of confirmatory factor analysis



Follow Up Analyses. Our primary model of interest did not support initial hypotheses that the negative urgency subscale would explain more variance in the relationship between anxiety and experiential avoidance than global impulsivity. However, due to poor model fit we sought to further understand relationships between these variables of interest using a data-driven approach.

Item-level data from both the UPPS-P and BIS-11 were subjected to an EFA to agnostically examine the factor structure for indicators of impulsivity. A parallel analysis indicated a six-factor solution from the item-level data above the 95th percentile estimates based on 100 simulated datasets (See Figure II.4 for scree plot).

Figure II.4
Scree plot for item-level exploratory factor analysis



Therefore, a six-factor solution was specified for an EFA with maximum likelihood extraction with an orthogonal varimax rotation to allow for a simpler factor structure. This model demonstrated good fit (χ^2 = 7707.01, df = 3397, p < 0.001; RMSEA = 0.05; SRMR = 0.03). Loadings of items from both measures on each factor are presented in Table II.7. Items were

retained if they loaded robustly (i.e., > 0.30) on a factor; four items from the BIS-11 were excluded from further analyses due to insignificant loadings.

Table II.7
Item level loadings of exploratory factor analysis for factors of impulsivity (UPPS-P, BIS-11)

Measure (Subscale)	Item Content	Factor Loadings					
		1	2	3	4	5	6
Factor 1: Premeditation							
UPPS-P (PRE)	1. I have a reserved and cautious attitude toward life.	**0.53****	-0.12	0.25	0	-0.05	-0.09
UPPS-P (PRE)	6. My thinking is usually careful and purposeful.	**0.52****	0.11	-0.03	0.21	**0.31***	0.18
UPPS-P (PRE)	11. I am not one of those people who blurt out things without thinking.	**0.35****	0.17	-0.04	0.07	0.17	0.18
UPPS-P (PRE)	16. I like to stop and think things over before I do them.	**0.65****	0.2	-0.02	0.11	0.15	0.15
UPPS-P (PRE)	21. I don't like to start a project until I know exactly how to proceed	**0.55****	-0.04	0.14	0.05	-0.02	0.01

UPPS-P (PRE)	28. I tend to value and follow a rational, "sensible" approach to things.	**0.56****	0.17	-0.03	0.11	0.19	0.14
UPPS-P (PRE)	33. I usually make up my mind through careful reasoning.	**0.52****	0.18	-0.06	0.14	0.28	0.24
UPPS-P (PRE)	38. I am a cautious person.	**0.65****	0.03	0.18	0.03	0.02	0.06
UPPS-P (PRE)	43. Before I get into a new situation I like to find out what to expect from it.	**0.64****	0.17	0.09	0.21	-0.06	-0.01
UPPS-P (PRE)	48. I usually think carefully before doing anything.	**0.65****	0.1	0.04	0.17	0.24	0.21
UPPS-P (PRE)	55. Before making up my mind, I consider all the advantages and disadvantages.	**0.61****	0.16	0.02	0.12	0.19	0.11
Factor 2: Urgency							
UPPS-P (NU)	^2. I have trouble controlling my impulses.	0.15	**0.51****	0.12	0.05	**0.34***	**0.33***
UPPS-P (NU)	^12. I often get involved in things I later wish I could get out of.	0.04	**0.53****	0.2	0.1	0.27	**0.31***

UPPS-P (NU)	^17. When I feel bad, I will often do things I later regret in order to make myself feel better now.	0.05	**0.58****	0.11	0.03	**0.34***	0.3
UPPS-P (NU)	^22. Sometimes when I feel bad, I can't seem to stop what I am doing even though it is making me feel worse.	-0.06	**0.60****	0.15	0.05	0.26	0.29
UPPS-P (NU)	^29. When I am upset I often act without thinking.	0.11	**0.58****	0.09	-0.02	**0.32***	**0.39***
UPPS-P (NU)	^34. When I feel rejected, I will often say things that I later regret.	-0.01	**0.59****	0.06	0.02	0.3	**0.34***
UPPS-P (NU)	^39. It is hard for me to resist acting on my feelings.	0.08	**0.56****	0.17	-0.1	0.26	**0.36***
UPPS-P (NU)	^44. I often make matters worse because I act without thinking when I am upset.	0.03	**0.64****	0.13	-0.01	**0.32***	**0.32***
UPPS-P (NU)	^50. In the heat of an argument, I will often say things that I later regret.	-0.07	**0.49****	0.12	-0.01	**0.36***	**0.36***

UPPS-P (NU)	^58. Sometimes I do impulsive things that I later regret.	0.03	**0.51****	0.19	-0.07	**0.43***	**0.35***
UPPS-P (PER)	^47. Sometimes there are so many little things to be done that I just ignore them all.	-0.07	**0.42****	0.18	0.26	**0.32***	**0.32***
UPPS-P (PU)	^5. When I am very happy, I can't seem to stop myself from doing things that can have bad consequences.	0.13	**0.70****	0.19	0.07	0.05	0.21
UPPS-P (PU)	^10. When I am in great mood, I tend to get into situations that could cause me problems.	0.17	**0.78****	0.19	0.07	0.07	0.24
UPPS-P (PU)	^15. When I am very happy, I tend to do things that may cause problems in my life.	0.11	**0.75****	0.19	0.06	0.03	0.21
UPPS-P (PU)	^20. I tend to lose control when I am in a great mood.	0.16	**0.78****	0.16	0.06	0.04	0.16
UPPS-P (PU)	^25. When I am really ecstatic, I tend to get out of control.	0.14	**0.79****	0.2	0.07	0.1	0.18

UPPS-P (PU)	^30. Others would say I make bad choices when I am extremely happy about something.	0.17	**0.81****	0.16	0.03	0.04	0.18
UPPS-P (PU)	^35. Others are shocked or worried about the things I do when I am feeling very excited.	0.17	**0.79****	0.21	0.03	-0.04	0.14
UPPS-P (PU)	^40. When I get really happy about something, I tend to do things that can have bad consequences.	0.12	**0.78****	0.19	0.03	0.03	0.23
UPPS-P (PU)	^45. When overjoyed, I feel like I can't stop myself from going overboard.	0.1	**0.80****	0.21	0.07	0.07	0.15
UPPS-P (PU)	^49. When I am really excited, I tend not to think of the consequences of my actions.	0.12	**0.77****	0.16	0.07	0.09	0.21
UPPS-P (PU)	^52. I tend to act without thinking when I am really excited.	0.13	**0.75****	0.18	-0.02	0.14	0.24

UPPS-P (PU)	^54. When I am really happy, I often find myself in situations that I normally wouldn't be comfortable with.	0.08	**0.77****	0.24	0.04	0.04	0.15
UPPS-P (PU)	^57. When I am very happy, I feel like it is ok to give in to cravings or overindulge.	-0.03	**0.49****	0.23	-0.04	0.16	0.27
UPPS-P (PU)	^59. I am surprised at the things I do while in a great mood.	0.07	**0.71****	0.28	-0.02	0	0.25
Factor 3: Sensation Seeking							
UPPS-P (SS)	^3. I generally seek new and exciting experiences and sensations.	0.12	0.22	**0.58****	-0.18	-0.11	0.17
UPPS-P (SS)	^8. I'll try anything once.	0.09	0.17	**0.48****	-0.07	-0.01	0.15
UPPS-P (SS)	^13. I like sports and games in which you have to choose your next move very quickly.	0.08	0.22	**0.36****	-0.17	-0.23	-0.02
UPPS-P (SS)	^18. I would enjoy water skiing.	0.04	0.13	**0.68****	0	-0.09	0.08

UPPS-P (SS)	^23. I quite enjoy taking risks.	0.23	0.35	**0.62****	-0.13	-0.02	0.05
UPPS-P (SS)	^26. I would enjoy parachute jumping.	0.05	0.26	**0.69****	0.04	-0.08	0.03
UPPS-P (SS)	^31. I welcome new and exciting experiences and sensations, even if they are a little frightening and unconventional.	0.04	0.22	**0.65****	-0.18	-0.03	0.05
UPPS-P (SS)	^36. I would like to learn to fly an airplane.	0.01	0.16	**0.64****	0.06	-0.07	0.07
UPPS-P (SS)	^41. I sometimes like doing things that are a bit frightening.	0.11	**0.35***	**0.66****	-0.03	0	0.16
UPPS-P (SS)	^46. I would enjoy the sensation of skiing very fast down a high mountain slope.	0.03	0.21	**0.78****	0.03	-0.1	0.09
UPPS-P (SS)	^51. I would like to go scuba diving.	-0.07	0.07	**0.70****	-0.02	0.01	0.06
UPPS-P (SS)	^56. I would enjoy fast driving.	0.11	0.24	**0.61****	-0.01	-0.01	0.07
Factor 4: Perseverance							
UPPS-P (PER)	4. I generally like to see things through to the end.	0.26	0.18	-0.07	**0.56****	0.24	0.14

UPPS-P (PER)	^9. I tend to give up easily.	-0.06	**0.36***	-0.02	**0.39****	**0.37***	0.23
UPPS-P (PER)	14. Unfinished tasks really bother me.	**0.31***	0.01	0.01	**0.42****	0.04	0.12
UPPS-P (PER)	19. Once I get going on something I hate to stop.	**0.34***	-0.03	-0.11	**0.5****	0.01	0.06
UPPS-P (PER)	27. I finish what I start.	0.21	0.03	-0.1	**0.62****	**0.36***	0.27
UPPS-P (PER)	32. I am able to pace myself so as to get things done on time.	0.27	0.02	-0.13	**0.51****	**0.33***	0.22
UPPS-P (PER)	37. I am a person who always gets the job done.	0.17	0.03	-0.17	**0.59****	**0.42***	0.15
UPPS-P (PER)	42. I almost always finish projects that I start.	0.13	0.06	-0.07	**0.59****	**0.40***	0.26
Factor 5: Planning							
BIS-11 (ATT)	^9 I concentrate easily.	-0.02	0.11	-0.05	0.28	**0.67****	0.1
BIS-11 (ATT)	^20 I am a steady thinker.	0.24	0.2	-0.06	0.02	**0.64****	0.11
BIS-11 (CC)	10 I save regularly.	0.04	0.08	-0.01	0.08	**0.51****	0.07
BIS-11 (CC)	^15 I like to think about complex problems.	0.04	0.07	-0.28	0	**0.38****	-0.05
UPPS-P (NU)	^7. I have trouble resisting my cravings (for food, cigarettes, etc.).	-0.06	**0.37***	0.07	-0.01	**0.40****	0.27

UPPS-P (NU)	53. I always keep my feelings under control.	0.22	0.01	-0.16	0.13	**0.43****	0.21
UPPS-P (PER)	24. I concentrate easily.	0.09	0	-0.15	0.41	**0.49****	0.22
BIS-11 (PER)	^30 I am future oriented.	0.11	0.03	-0.13	0.12	**0.49****	-0.1
BIS-11 (SC)	^1 I plan tasks carefully.	**0.31***	0.15	0.13	-0.04	**0.52****	0.11
BIS-11 (SC)	^7 I plan trips well ahead of time.	**0.32***	0.24	0.13	0.01	**0.38****	0
BIS-11 (SC)	^8 I am self controlled.	0.21	0.22	-0.05	0.02	**0.64****	0.16
BIS-11 (SC)	^12 I am a careful thinker.	**0.34***	0.23	0.05	-0.06	**0.56****	0.09
BIS-11 (SC)	^13 I plan for job security.	0.1	0.14	0.02	0.14	**0.54****	-0.01
Factor 6: Hyperactivity							
BIS-11 (ATT)	5 I don't "pay attention."	-0.11	0.12	0.03	0.09	**0.34***	**0.38****
BIS-11 (ATT)	11 I "squirm" at plays or lectures.	0.01	0.24	0.07	0.13	0.03	**0.48****
BIS-11 (ATT)	28 I am restless at the theater or lectures.	0.07	0.2	0.03	0.13	0.06	**0.47****
BIS-11 (CC)	18 I get easily bored when solving thought problems.	0.1	0.3	-0.07	0.1	0.09	**0.44****
BIS-11 (COGIN)	6 I have "racing" thoughts.	-0.09	0.27	0.05	0.09	0.06	**0.51****
BIS-11 (COGIN)	24 I change hobbies.	0.12	0.24	0.17	0.19	-0.04	**0.49****

BIS-11 (COGIN)	26 I often have extraneous thoughts when thinking.	-0.08	0.19	0.12	0.2	0.06	**0.50****
BIS-11 (MOT)	2 I do things without thinking.	**0.30***	0.28	0.11	0	0.21	**0.55****
BIS-11 (MOT)	17 I act "on impulse."	0.24	0.26	0.15	-0.07	0.09	**0.67****
BIS-11 (MOT)	19 I act on the spur of the moment.	0.28	0.3	0.21	-0.08	-0.03	**0.67***
BIS-11 (MOT)	22 I buy things on impulse.	0.16	0.26	0.16	0.03	0.1	**0.60***
BIS-11 (MOT)	25 I spend or charge more than I earn.	0.18	**0.32***	0.1	0.13	0	**0.54***
BIS-11 (PER)	16 I change jobs.	0.14	0.24	0.21	0.2	-0.04	**0.43***
BIS-11 (PER)	21 I change residences.	0.22	0.28	0.18	0.18	-0.12	**0.46***
BIS-11 (PER)	23 I can only think about one thing at a time.	0.05	0.26	-0.08	0.22	-0.1	**0.32***
BIS-11 (SC)	14 I say things without thinking.	0.24	**0.36***	0.03	0.02	0.14	**0.58***
No Significant Loadings							
BIS-11 (CC)	27 I am more interested in the present than the future.	0.12	0.22	0	0.1	-0.09	0.29
BIS-11 (CC)	^29 I like puzzles.	0.12	0.09	-0.14	0.05	0.29	-0.12
BIS-11 (MOT)	3 I make-up my mind quickly.	0.23	0.12	0.12	-0.1	-0.38	0.17
BIS-11 (MOT)	4 I am happy-go-lucky.	0.19	0.12	0.18	-0.14	-0.44	0.18

** = primary factor loading > 0.30; * = secondary factor loading > 0.30; ^ = reverse scored item

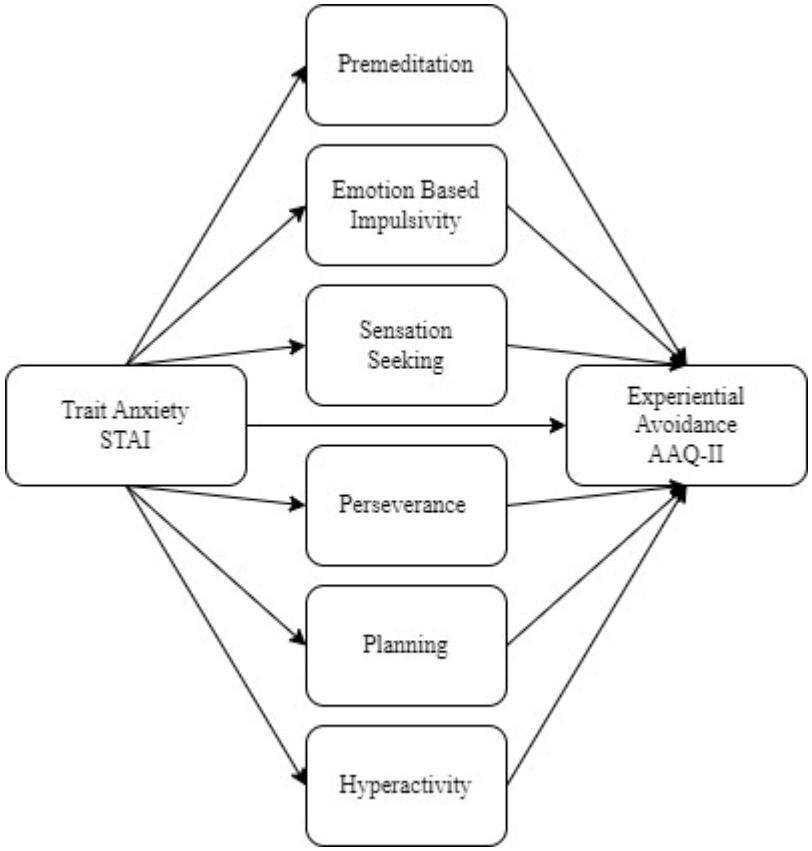
Factor 1 (UPPS-P Premeditation) was comprised of 11 items with factor loadings from 0.35 to 0.65. Conceptually, this factor consisted of items from the UPPS-P premeditation subscale related to caution and planning. Items loading on this factor were reverse scored such that higher scores on these items reflected lack of premeditation. Factor 2 (Urgency) was comprised of 25 items with factor loadings from 0.42 to 0.81. Items that loaded significantly onto this factor were largely from the negative and positive urgency subscales of the UPPS-P related to emotional reactivity. Factor 3 (Sensation Seeking) was comprised of 12 items with factor loadings from 0.36 to 0.78. This factor included items from the UPPS-P sensation seeking subscale. Factor 4 (Perseverance) was comprised of 8 items with factor loadings from 0.39 to 0.62. This factor consisted of items from the UPPS-P perseverance subscale and items were also reverse scored such that higher scores reflected a lack of perseverance.

Factor 5 (BIS-11 Planning) was made up of 13 items that were all reverse scored with factor loadings from 0.35 to 0.67. This factor included items from the BIS-11 related to future planning. This factor reflected similar themes to that of Factor 1 and was strongly correlated with Factor 1 ($r = 0.74$). However, only four items cross-loaded > 0.30 (range: 0.31-0.34) between Factor 1 and Factor 5. While Factor 1 was comprised solely of items from the UPPS-P premeditation subscale, Factor 5 consisted predominantly of items from the BIS-11. Therefore, it is possible that distinctions between these two factors are driven by measurement effects capturing subtle differences in similar substantive content surrounding lack of premeditation and planning. In order to distinguish these factors, Factor 1 was designated the (lack of) Premeditation factor while Factor 5 was labeled the (lack of) Planning Factor. Factor 6 (Hyperactivity) was comprised of 16 items with factor loadings from 0.32 to 0.67 from only the BIS-11. Items loading onto this factor reflect themes of hyperactivity (e.g., restless, fidgeting,

motor activity) parallel to the “Hyperactivity” factor in a three factor structure for ADHD (See Figure 1 of Park et al., (2018) for ADHD Hyperactivity factor loadings).

These results of the EFA were then used to inform the structure of factors included in a subsequent CFA. In keeping with our hypothesized model, the six factors of impulsivity served as mediators of the relationship between anxiety and experiential avoidance (See Figure II.5 for conceptual model).

Figure II.5
Conceptual model of confirmatory factor analysis informed by item-level exploratory factor analysis



Intercorrelations between factors were not specified and factor means were set at 0 with variances set at 1. In spite of good model fit in the data driven EFA, the revised impulsivity factor structure did not improve model fit to meet conventional standards of fit for the CFA (χ^2 =

12428.30, df = 3649, p < 0.001; RMSEA = 0.06; SRMR = 0.19; CFI = 0.73; TLI = 0.72). Specifically, modifications to the factor structure for dimensions of impulsivity did not contribute to sufficient improvement of model fit in the overall CFA with anxiety and experiential avoidance.

As a final attempt to elucidate granular relationships between our variables of interest, we conducted an item level EFA to examine the structure of our dependent variable, experiential avoidance. The AAQ-II is comprised of only seven items; therefore, one and two factor solutions were specified for an EFA with maximum likelihood extraction and oblique rotation. A two-factor model demonstrated good fit (χ^2 = 52.27, df = 3397, p < 0.001; RMSEA = 0.10; CFI = 0.95, TLI = 0.86; SRMR = 0.04). Three items conceptually related to painful memories, feelings, and experiences loaded robustly onto one factor with loadings ranging between 0.44 and 0.76, while the remaining four items associated with interference of emotions loaded robustly on a second factor with loadings ranging from r = 0.43 and r = 0.76. Factors were correlated at r = 0.41. Loadings of items on each factor are presented in Table II.8.

Table II.8
Item level loadings of exploratory factor analysis for the AAQ-II

	Factor Loadings	
Item Content	1	2
Factor 1: Painful Memories and Experiences		
1. My painful experiences and memories make it difficult for me to live a life that I would value	**0.758****	0.08
2. I'm afraid of my feelings	**0.549****	-0.023
4. My painful memories prevent me from having a fulfilling life	**0.438****	-0.144
Factor 2: Consequences of Emotions		
3. I worry about not being able to control my worries and feelings	0.061	**0.74****
5. Emotions cause problems in my life	-0.08	**0.585****

6. It seems like most people are handling their lives better than I am	-0.005	**0.76****
7. Worries get in the way of my success	0.114	**0.434****

** = primary factor loading > 0.30; factor correlation = 0.41

A subsequent CFA was once again conducted with trait anxiety as the primary predictor of interest and the previously described six factors of impulsivity as mediators. In this model, we specified a bifactor model for experiential avoidance based on results of the EFA for the AAQ-II. This revised factor structure did improve model fit; however, this model still did not meet conventional standards of good fit (χ^2 = 12431.66, df = 4161, p < 0.001; RMSEA = 0.06; CFI = 0.77, TLI = 0.76; SRMR = 0.18). Results of this follow up CFA are detailed in Table II.9 and graphically in Figure II.6.

Table II.9
Results from follow up confirmatory factor analyses

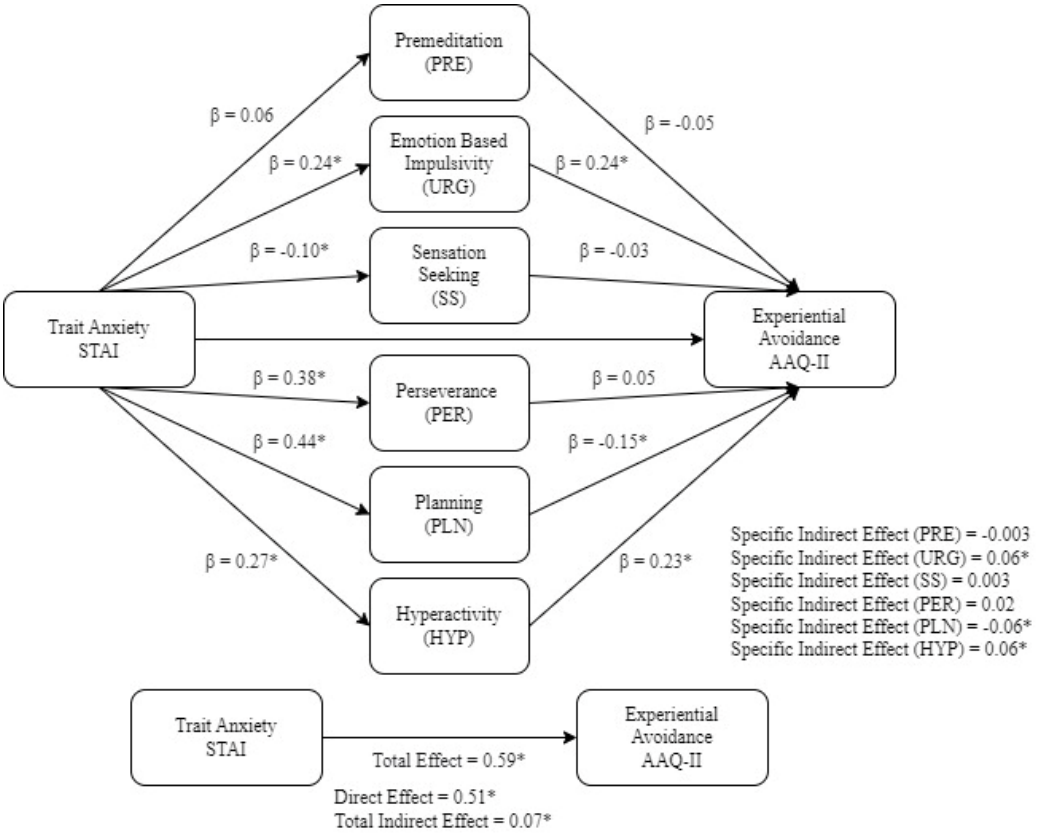
	95% CI	STD
Factor 1: Premeditation (F1)		
STAIT → F1	[-0.03, 0.14]	0.06
F1 → EA	[-0.20, 0.10]	-0.05
Indirect Effect	[-0.01, 0.01]	-0.003
Factor 2: Urgency (F2)		
STAIT → F2	[0.16, 0.31]*	0.27
F2 → EA	[0.10, 0.38]*	0.28
Indirect Effect	[0.02, 0.10]*	0.08
Factor 3: Sensation Seeking (F3)		
STAIT → F3	[-0.19, -0.02]*	-0.11
F3 → EA	[-0.13, 0.08]	-0.02
Indirect Effect	[-0.01, 0.01]	0.002
Factor 4: Perseverance (F4)		
STAIT → F4	[0.31, 0.45]*	0.41
F4 → EA	[-0.08, 0.18]	0.06
Indirect Effect	[-0.03, 0.07]	0.02
Factor 5: Planning (F5)		
STAIT → F5	[0.37, 0.51]*	0.47
F5 → EA	[-0.27, -0.01]*	-0.2

Indirect Effect	[-0.12, -0.004]*	-0.1
Factor 6: Hyperactivity (F6)		
STAIT → F6	[0.19, 0.34]*	0.3
F6 → EA	[0.09, 0.38]*	0.27
Indirect Effect	[0.42, 0.60]	0.08
Direct & Total Effects		
Direct Effect	[0.42, 0.60]*	0.65
Total Indirect Effect	[0.00, 0.15]	0.08
Total Effect	[0.53, 0.64]*	0.73

Note: STAIT = Trait Anxiety; NU = Negative Urgency; EA = Experiential Avoidance; 95% CI = 95% confidence intervals for standardized coefficients; STD = standardized coefficients; * = $p < 0.05$. 95% confidence intervals derived via bias corrected bootstrap procedure with 10,000 random draws.

Figure II.6

Graphical model of follow up confirmatory factor analysis



As in our previous CFA, there was a direct effect of trait anxiety on experiential avoidance ($b = 0.51$). Trait anxiety was a significant predictor of the urgency factor ($b = 0.24$),

the sensation seeking factor (*b* = -0.10), the perseverance factor (*b* = 0.38), the planning factor (*b* = 0.44), and the hyperactivity factor (*b* = 0.27). Significant predictors of experiential avoidance included the urgency factor (*b* = 0.24), the planning factor (*b* = -0.15), and the hyperactivity factor (*b* = 0.23). There were significant indirect effects of three out of our six impulsivity factors; urgency (*b* = 0.06) and hyperactivity (*b* = 0.06) demonstrated small positive indirect effects, such that individuals who experienced high anxiety and high urgency or hyperactivity demonstrated greater experiential avoidance. In contrast, the (lack of) planning factor demonstrated a small negative indirect effect (*b* = -0.06). Anxiety was positively correlated with (lack of) planning and with experiential avoidance while (lack of) planning was inversely related to experiential avoidance. The indirect effect was negative such that individuals who exhibited high anxiety and less planning tendencies demonstrated less experiential avoidance. Both the total indirect effect (*b* = 0.07) and the total effect (*b* = 0.59) of the overall model were statistically significant as well.

Monte Carlo Simulations. Prior to primary analyses, Monte Carlo simulations were run using MPlus statistical software to determine sample size for adequate power using the results from analyses on a preliminary sample (n = 250) following procedures outlined by (Muthén & Muthén, 2002). 1000 replications were run for Ns of 250, 500, and 750. For n = 250, 34% of replications yielded significant models, for n = 500, 58% of replications yielded significant models, and for n = 750, 79% of replications yielded significant models. Per results of these simulations, the collected sample of n = 916 would likely have been well powered for our analyses; however, due to efforts to eliminate potential inattentive participants and bots, analyses were conducted on a final sample of n = 589, which may not be adequately powered.

Discussion

Although avoidance is a hallmark of anxiety, impulsive profiles of anxious avoidance are frequently not captured by present diagnostic models. Furthermore, broad definitions of impulsivity may contribute to mixed findings in existing research exploring relationships between anxious and impulsive presentations. The present study contributes to an ever-growing body of transdiagnostic literature by examining the interactions between specific dimensions of impulsivity and internalizing symptoms such as anxiety and experiential avoidance. As an emotion-based dimension of impulsivity, negative urgency was of particular interest in these analyses given its previous links to both anxiety (Altan-Atalay et al., 2020; Altan-Atalay & Zeytun, 2020; King et al., 2021; Malivoire et al., 2019b; Pawluk & Koerner, 2016b) and experiential avoidance (Brem et al., 2017b; Rodrigues et al., 2022). Results of our initial hypothesis-driven model using subscales of established measures demonstrated positive correlations between anxiety and experiential avoidance, negative urgency, and global impulsivity. Additionally, this model revealed that the indirect effect of anxiety on experiential avoidance was not stronger through negative urgency, and a latent factor of global impulsivity better explained variance in this relationship. In isolation, these findings suggested that rash action driven by negative emotion, as measured by the widely used UPPS-P subscale, was not sufficient to explain the role of impulsive traits in anxiety-based experiential avoidance.

Follow up analyses conducted to elucidate data-driven factors of impulsivity provided further insight into more nuanced impulsive traits that contributed to experiential avoidance. These exploratory analyses yielded six factors of impulsivity, comprised of premeditation, emotion-based impulsivity (i.e., urgency), sensation seeking, perseverance, planning, and hyperactivity. As previously outlined, multiple measures of impulsivity have been generated through the lenses of underlying personality traits, involvement of emotion, and associated

cognitive factors (Carver & Johnson, 2018). Using multiple indicators of impulsivity allowed for a more agnostic generation of impulsive factors. These data-driven factors, rather than established subscales of individual measures, may provide utility in future studies examining relationships between impulsive dimensions and anxious symptomatology.

These data-driven factors provided nuance to our initial finding that negative urgency was not associated with anxiety and experiential avoidance. Our follow up CFA revealed that individuals with greater anxiety and emotion-based impulsivity (comprised of both negative and positive urgency) demonstrated greater experiential avoidance. This finding suggests that impulsive reactions to elevated emotion of any valence, in combination with anxiety, is associated with avoidance of internal negative emotional states. While research on anxious populations has placed emphasis on negative urgency, some studies have found positive associations between anxiety and broader emotion-based impulsivity (Johnson et al., 2013, 2017).

We predicted that the indirect effect of anxiety on experiential avoidance would be stronger via emotion-based rather than non-emotional dimensions of impulsivity. However, our data-driven model revealed a similar pattern with the non-emotional factor of hyperactivity. Specifically, individuals who exhibited greater anxiety and hyperactivity demonstrated greater experiential avoidance. These results are in line with previous literature demonstrating a high cooccurrence of ADHD hyperactivity symptoms and clinically significant anxiety (Jacob et al., 2014; Reimherr et al., 2017). No current research has examined the relationship between hyperactivity and experiential avoidance. However, one study found that compared to healthy participants, individuals with ADHD had greater difficulty tolerating emotionally discomfiting situations without engaging in impulsive behaviors- an experience akin to experiential avoidance

(Cavelti et al., 2019). It is possible then that motor-based impulsivity (e.g., restlessness, fidgeting) may serve as a way to cope with internal distress or may be an outward manifestation of it.

While indirect effects of anxiety on experiential avoidance through both emotion-based impulsivity and hyperactivity were positive, the indirect effect through the planning factor was negative. In other words, high anxiety and lower planning was associated with less experiential avoidance. This finding aligns with initial expectations that in comparison to emotional dimensions, non-emotional dimensions of impulsivity would not be positively associated with experiential avoidance. However, planning did not mirror hyperactivity, which is also a non-emotional dimension of impulsivity. Furthermore, this result does not align with prior literature linking greater emotion regulation, reflection, and mindfulness to reduced experiential avoidance (Naragon-Gainey et al., 2017).

The finding that high anxiety and lower planning were associated with less experiential avoidance suggests that there may be additional factors that influence relationships between impulsive dimensions and experiential avoidance. Additional measures would be needed to fully elucidate underlying drivers of these results; however, parallels between the (lack of) premeditation and planning factors provide some clues for further exploration. The premeditation factor was not a significant mediator, but it demonstrated the same directionality of relationships with anxiety and experiential avoidance (i.e., a positive relationship with anxiety, negative relationship with experiential avoidance, and a negative indirect effect). As previously outlined, items loading onto both premeditation and planning factors reflected similar themes of lack of self-discipline, responsibility, or caution (e.g., UPPS-P Item 16: “I like to stop and think things over before I do them”). A speculative interpretation could be that individuals who are anxious

and lack planning tendencies may be less reflective, or less likely to link their anxiety to internal cues (e.g., emotion, physical sensations) and painful memories associated with experiential avoidance. To our knowledge, there is no prior literature linking lack of premeditation or planning (or similar constructs including self-discipline or future orientation) and experiential avoidance. In all, these results suggest that presentations of anxiety in conjunction with different dimensions of impulsivity yield different patterns of experiential avoidance.

Limitations. These analyses are limited by low power due to a small sample size that may contribute to poor model fit for results of the CFA. Although data from 916 participants were collected, our sample was limited to 589 participants following MTurk data quality checks. Due to the strength of relationships, we would anticipate that a larger sample would reinforce these patterns of results and future studies should examine these questions in larger samples. Furthermore, although we aimed to recruit equivalent numbers of men and women, generalizability of findings may be limited due to homogeneity of racial background and sexual orientation across our sample. Previous studies have demonstrated that individuals who hold sexual, racial, or gender minority identities exhibit elevated rates of psychopathology (Fox et al., 2020), and in particular, may engage in risky behaviors commonly associated with impulsivity (Jones et al., 2018). The unique psychosocial stressors (e.g., racism, homophobia) and lack of structural support that individuals with minoritized identities face may exacerbate relationships between negative urgency and experiential avoidance.

Furthermore, poor model fit of the CFA may reflect variance not captured by measures administered in this study, data quality notwithstanding. For one, this study did not measure anxiety sensitivity, which has previously been implicated in the relationship between anxiety and negative urgency (Guillot et al., 2014; Sofis et al., 2020). The inclusion of measures of anxiety

sensitivity, such as the Anxiety Sensitivity Index (Reiss et al., 1986), could provide more insight into how fears of sensations associated with emotions may interact with impulsive traits and contribute to experiential avoidance. Furthermore, this study did not directly measure or exclude externalizing psychopathology (e.g., substance use, ADHD). Externalizing symptoms unaccounted for in these analyses may have contributed unexplained variance in these relationships. In order to specifically isolate the interaction between anxious symptomatology and impulsive traits, future studies may exclude substance use or ADHD symptoms or include measures as covariates.

Finally, these analyses may be impacted by potential floor effects of item responses. This study aimed to examine impulsive traits and anxious symptoms across continuums that transcend conventional clinical cutoffs, and therefore, data collection was not restricted to clinical samples. Although subscale scores across all of our measures spanned from subclinical to clinical ranges, individual items- particularly on measures of impulsivity- demonstrated low endorsement rates. Findings from this unrestricted sample would likely benefit from replication in a sample with a greater range of symptom endorsement, especially in the clinical range.

Future Directions. Results from this research provide a foundation to examine individual differences in profiles of co-occurring anxiety and impulsivity that could yield different targets for behavioral treatments. In particular, future research should examine relationships between emotion-based dimensions of impulsivity and experiential avoidance accounting for externalizing psychopathology, anxiety sensitivity, and means of behavioral avoidance. Due to the cross-sectional nature of our data, it is not possible to interpret directionality of relationships; future studies may reexamine these relationships within a longitudinal cohort to better understand causal links between anxious and impulsive constructs.

Additionally, our findings provide evidence for relationships between impulsive dimensions and experiential avoidance, future research may examine coping behaviors (e.g., "escape" related behaviors, methods of alleviating distress in the absence of problematic substance use) associated with these constructs.

Conclusion. In summation, the present study demonstrated that the relationship between anxiety and experiential avoidance is differentially mediated by emotional and non-emotional dimensions of impulsivity. Although the indirect effect of anxiety on experiential avoidance through negative urgency was not significant, the indirect effect was significant via emotion-based impulsivity and hyperactivity. Conversely, we found a significant negative indirect effect via planning such that greater anxiety and low planning tendencies were associated with less experiential avoidance. Evidence that both emotional and non-emotional impulsive factors may have different relationships with experiential avoidance further motivates analyses of neural function in association with clinical symptoms, to be outlined in Chapters 3 and 4. Examining shared and distinct regions of prefrontal cortex activation involved in emotional and non-emotional inhibitory control processes (Chapter 3) and comparing relationships between these patterns of activation and clinical symptoms (Chapter 4) will provide further insight into neural correlates that could underlie correlations observed in these analyses.

CHAPTER III

Prefrontal Activation Associated with Emotional Interference and Motor Inhibition in the Adolescent Brain Cognitive Development (ABCD) Dataset

Prefrontal cortex (PFC) function has broadly been associated with a wide array of processes (e.g. emotional appraisal, conflict management, motor inhibition) relevant to inhibitory control related to psychopathology (Braver, 2001; Dixon et al., 2017; Hu et al., 2016). Prefrontal inhibitory control is engaged during emotional interference to maintain attention on a primary task while disengaging from emotionally salient distractors. Prefrontal inhibitory control is also engaged during motor inhibition tasks to interrupt prepotent motor responses. Studies have demonstrated reduced inhibitory control in individuals with psychopathology during both emotional (e.g. emotional interference) and non-emotional (e.g. motor inhibition) tasks (Braver, 2001; Dixon et al., 2017; Hu et al., 2016). Emotional interference has relevance to anxiety and motor inhibition has relevance to impulsivity. Lack of inhibitory control underlying both may be a crucial component in a general "p factor" underlying all psychopathology (Bari & Robbins, 2013; Sotres-Bayon et al., 2004; Weafer et al., 2019).

Our goals are to understand how facets of inhibitory control (emotional interference and motor inhibition) manifest in the brain, and then to explore relationships between brain-based processes and clinical symptoms (i.e., anxiety and impulsivity). Therefore, in this chapter, we interrogated potential shared and unique patterns of PFC function underlying inhibitory control

associated with emotional interference and motor inhibition. We aimed to uncover PFC function associated with inhibitory control processes shared between both emotional and non-emotional tasks. Studies have examined neural activation associated with these processes separately or in parallel, but few studies have specifically interrogated the overlap in patterns of activation related to both emotional (emotional interference) and non-emotional (motor inhibition) facets of inhibitory control. In the present study, we take a transdiagnostic approach to examining overlapping and distinct patterns of PFC activation during emotional interference and motor inhibition using data from early adolescents in the ABCD dataset.

Emotional Interference

Emotional interference in non-clinical samples has been conceptualized as a function of bottom-up processing of emotional input combined with top-down PFC regulation (Marwood et al., 2018). Previous research on emotional interference in adult and adolescent samples has implicated regions involved in: modulating emotional responses and conflict management, namely the ACC (Stollstorff et al., 2013); and decision making and working memory such as the dlPFC (Kohn & Fernández, 2020). In addition, the middle frontal gyrus, a region associated most commonly with language and possibly involved in reorienting attention, has been implicated in emotional interference (Colich et al., 2017; Japee et al., 2015).

Clinical samples – particularly those with anxiety – have demonstrated differences in both performance and neural activation associated with emotional interference (Goodwin et al., 2017a). Studies have found that when compared to healthy controls, participants with anxiety demonstrate increased attention to negative stimuli and slower reaction times on emotional interference tasks (Veerapa et al., 2020). Some studies have shown that in comparison to healthy

individuals, those with anxiety have reduced activation in the PFC during emotional interference (Villemonteix et al., 2017). However, other research has suggested that individuals with anxiety may recruit additional PFC activation to maintain performance (Ladouceur et al., 2005a).

Table III.1
Studies examining neural activation during negative emotion (or combined emotional) vs. neutral contrasts of the EN-Back Task

Citation	Sample	Task Contrasts	dlPFC	ACC	Amygdala	Putamen	Additional Regions	Psychopathology
(O'Brien et al, 2020)	Pre-Adolescents	2-Back, 0-Back: Emotional (Happy, Fearful) vs. Neutral Faces	↓	↓ Caudal	↑ L,R	↓	↓ Caudate; ↓ Nucleus Accumbens (L)	− Psychotic-Like Symptoms; − Depression
(Mueller et al, 2017)	Adolescents	0-Back: Angry vs. Neutral	↑		–		– Nucleus Accumbens	None
	Adults		–					
(Mullin et al, 2012)	Adults	2-Back: Fearful vs. Neutral Faces	↑	−	↑ R	↑ R	↑ Inferior Parietal Cortex (L,R)	↑ Bipolar Disorder
(Bertocci et al, 2012)	Adults	2-Back: Fearful vs. No Face	–	–	–	–		– Depression; − Bipolar Disorder
(Sun et al., 2020)	Adults	0-Back: Combat vs. Neutral Images	−	↑ Subgenual	↑ L,R			− PTSD

Note. dlPFC = dorsolateral prefrontal cortex; ACC = anterior cingulate cortex Directionality of activation for contrast and correlations with psychopathology indicated (↑ = Increased; ↓ = Decreased; − = None)

Emotional N-Back Task. Emotional interference has been examined using common probes of working memory and cognitive control with emotionally salient stimuli (e.g., Affective Stroop Task, EN-Back Task). The EN-Back was designed to probe working memory capacity and cognitive load while processing emotional and non-emotional information (Casey et al., 2018a). In addition, this task may be used to probe differences in cognitive processes due to interference from emotional (mainly negative) stimuli. fMRI studies have examined neural function during the negative vs. neutral image contrast (i.e., activation during trials including negative stimuli vs. trials including neutral stimuli) on the EN-Back Task. Consistent with other probes of emotional interference, these studies have found differences in PFC (dlPFC, ACC) and amygdala activation during negative compared to neutral trials (For review of studies, see Table III.1). Directionality in patterns of neural function and relationships with clinical symptoms have also yielded mixed findings across studies. To our knowledge, one study has examined neural function during negative vs. neutral face trials on the EN-Back Task using the ABCD dataset to probe relationships between neural function and both depression and psychotic-like symptoms (O'Brien et al., 2020a). This study demonstrated that all participants had reduced activation in dlPFC and ACC and increased activation in amygdala during emotional vs. neutral face trials. Studies examining relationships between internalizing symptoms and neural function on the EN-Back Task have predominantly focused on the working memory condition. Two studies have examined relationships between symptoms of bipolar disorder and depression and neural function during emotional vs. neutral contrasts (Bertocci et al., 2012; Mullin et al., 2012). One study reported that greater bipolar symptom severity was associated with greater activation in dlPFC, amygdala, and putamen during fearful faces compared to neutral faces (Mullin et al.,

2012). By contrast, the other study reported no differences in neural function during the fearful face vs. no face contrast and no relationship with clinical symptoms (Bertocci et al., 2012).

Motor Inhibition

Deficits in inhibitory control during emotional interference appear most clinically relevant to anxiety; in contrast, motor inhibition (i.e., ability to inhibit prepotent responses), is often employed as a cognitive and neural proxy for impulsivity (Roberts et al., 2011). Well established neural indices of motor inhibition in healthy samples include motor control regions (e.g., supplementary motor area; SMA) and inferior frontal gyrus (IFG; Weafer et al., 2019). In addition, some studies and meta analyses have linked motor inhibition with activation in emotional processing regions, including ACC (Rubia et al., 2001a), dlPFC, and insula (Nee et al., 2007a). Like emotional interference, behavioral performance and patterns of activation associated with motor inhibition have differed between healthy controls and clinical samples. Impaired motor inhibition has been associated with ADHD, OCD, and psychotic disorders, but is not as strongly linked with internalizing disorders such as anxiety and depression (For meta-analysis, see Lipszyc & Schachar, 2010).

Stop Signal Task. Motor inhibition has been classically assessed with paradigms such as the Go/No-Go or Stop Signal Tasks (SST; Logan et al., 2014) which do not include emotional stimuli. fMRI studies have examined neural function during failed stop trials vs. go trials (i.e., activation during trials where participants fail to inhibit response when a stop cue is presented vs. trials where a go cue is presented) on the SST. This task has been widely established as a probe of motor inhibition, and neural activation associated with failed inhibition on this task aligns with findings from other common tasks (e.g., Go/No-Go, Flanker) in IFG, SMA, and ACC (Rubia et al., 2001a). Two studies thus far have reported relationships between neural activation

on the SST and clinical symptoms using the ABCD dataset. In one study, differences between children with and without parental history of alcohol use disorder were only found in cerebellum for failed inhibition, while no differences were found in frontal regions (Lees et al., 2020). Another study found decreased activation in orbitofrontal and parietal regions during failed inhibition in participants with ADHD and high irritability (Lee et al., 2022). To our knowledge, studies have not yet been conducted on the ABCD sample examining activation during SST and internalizing symptoms. Although studies have consistently demonstrated that anxiety is associated with increased reaction time on the SST (Lipszyc & Schachar, 2010a), only a few studies have examined relationships between activation during the SST and internalizing symptoms in adult samples. One study found that people with anxiety and depression who use cannabis showed greater negative and positive urgency than healthy controls, but there were no differences in patterns of activation on the SST (Spechler et al., 2020a). By contrast, another study only demonstrated a relationship between trait anxiety and amygdala activation on the SST, but not between trait anxiety and vmPFC activation (Li et al., 2009).

Early Adolescent Prefrontal Development Associated with Anxiety and Impulsivity

Examining shared and distinct patterns of PFC inhibitory control over the course of development may illuminate risk for clinically significant psychopathology over the lifetime. Onset of both anxiety disorders (Ströhle et al., 2018a) and externalizing disorders marked by high impulsivity (e.g. ADHD; O'Neill et al., 2017) often precede full development of PFC in early adulthood (Arain et al., 2013). However, research has demonstrated variability in PFC development in early adolescence that may precede the onset of both clinically significant anxiety and impulsivity. In particular, childhood anxiety has been associated with age-related differences in PFC-amygdala connectivity (Gee et al., 2013; Gold et al., 2016; Wu et al., 2016a).

One longitudinal analysis of early adolescents has demonstrated changes in connectivity between amygdala and dlPFC associated with increasing anxiety and low behavioral inhibition between age 10 to age 13 (Abend et al., 2020). Furthermore, a meta-analysis of developmental (age range: 9-21 years) neuroimaging studies showed that externalizing disorders marked by impulsivity (i.e., Oppositional Defiant Disorder; ODD, Conduct Disorder; CD, ADHD) are also associated with reduced activation in frontal regions such as bilateral ACC and OFC in addition to amygdala and striatum (Noordermeer et al., 2016). These findings from an early adolescent sample suggest that 1) there is evidence of differences in PFC activation and connectivity with bottom-up processing regions in pre-clinical and clinical samples compared to healthy adolescents, and 2) these differences have been associated with or are predictive of both anxiety and impulsivity. This suggests that there is merit to further examining PFC activation in an early adolescent sample.

Rationale for the Present Sample

The purpose of this study was to examine shared and distinct patterns of activation in the PFC across emotional and non-emotional forms of inhibitory control. We chose the large, multisite ABCD dataset to answer these questions because it allowed us to examine patterns of neural function associated with two separate inhibitory control tasks completed by the same participants. This dataset includes behavioral, psychometric, and neuroimaging data from over 11,000 early adolescent children and has been previously used to assess transdiagnostic relationships across psychological constructs and relationships between neural function and psychological symptoms (Rapuano et al., 2020; O'Brien et al., 2020). Previous research has examined PFC activation during emotional and non-emotional tasks separately, with little

opportunity to conduct within-subject comparisons of neural activation across tasks. Prior studies that have probed activation across tasks (Rubia et al., 2001a; Swick et al., 2011) have lacked the sample size of a multisite dataset like ABCD, limiting statistical power and generalizability of findings. The use of this dataset provides an unprecedented opportunity to examine PFC activation across established tasks of emotional interference and motor inhibition in a well-powered and more representative sample of adolescents across the United States. Further examination of PFC activation in a large, well powered dataset of adolescents may provide critical insight into patterns of activation that could convey risk for the future development of psychopathology. Early detection of possible neurobiological indicators of psychopathology could allow for a greater focus on methods of prevention earlier in development.

Specific Aims

The present study examined neuroimaging data from the EN-Back Task and SST from the ABCD dataset. We aimed to identify shared and distinct PFC activation associated with emotional interference and motor inhibition. Findings of shared regions of activation during both tasks would likely reflect common processes that govern both emotional and non-emotional inhibition. We had the following predictions:

First, consistent with previous studies on emotional interference using the EN-Back Task, we hypothesized that reduced activation would be observed in regions including the dlPFC, vmPFC, and ACC during negative compared to neutral face trials. Second, based on meta-analyses and reviews of neural activation during the SST, we hypothesized that reduced activation in IFG, ACC, and dlPFC would be observed during failed stop vs. go trials. Third, we

hypothesized that similar patterns of PFC activation would be shared across both tasks in regions including the dlPFC and ACC.

Method

Participants

The ABCD Study is a multisite, longitudinal dataset including neurocognitive and psychometric data from 9–10-year-old children (Casey et al., 2018b) collected from 22 sites across the United States. The ABCD Release 4.0 included task-based neuroimaging data and psychometric data from 11,876 participants (DOI 10.15 154/1523041). For full ABCD Study recruitment procedures and sample information, see (Barch et al., 2018a; Garavan et al., 2018). For these analyses, data from children with ADHD, psychotic-like symptoms, developmental delays, and substance use risk based on clinical thresholds from the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS; Kaufman et al., 1997) were excluded. Given that a primary measure of interest for Chapter 4 was the UPPS-P negative urgency scale, participants without negative urgency scores were also excluded. Finally, participants were excluded for excessive head motion during MRI scans (< 4 minutes per run of data with FD<0.9), poor coregistration or warping, or poor behavioral performance based on ABCD performance flags (for breakdown of inclusion criteria for the current sample, see Table III.2). After exclusions, the final sampled consisted of 2264 participants (M_{age} = 10.02, SD_{age} = 0.63; 56.22% Female; 63.68% White; 47.75% >$100,000 Income). For full demographic information, see Table III.3.

Table III.2
Inclusion criteria for current sample

Inclusion Criteria	n
Total Baseline Arm 1 Participants	11878
No Substance Use	11847
Completed Negative Urgency Subscale	11726
No Risk for Psychosis	11559
No ADHD	9324
No Developmental Delays	8628
Completed SST	5188
Completed EN-Back	4369
Acceptable MRI Data	2264

Table III.3
Demographic Characteristics of the Sample
(n = 2266; M_{age} = 10.02, SD_{age} = 0.63)

	n	%
Race/Ethnicity		
White	1443	63.68%
Hispanic	392	17.30%
Black	178	7.86%
Asian	50	2.21%
Other	203	8.96%
Sex		
Female	1274	56.22%
Male	992	43.78%
Income		
≤ $50,000	133	17.87%
$50,000-$100,000	405	28.51%
≥ $100000	646	47.75%
Not Specified	1082	5.87%

Procedure

Emotional N-Back Task (EN-Back). The EN-Back task is a modified version of a working memory task that includes conditions of differing cognitive load with emotional and non-emotional stimuli. The task included 24 unique faces of each emotion type and 24 non-emotional images of buildings, during which participants indicate whether an emotional (happy, fearful), non-emotional (neutral) face, or building "lure" matches a fixed "target" image at the beginning of the task block (0-Back), or a "target" image presented two trials prior to the current trial (2-Back). This task allowed for assessment of emotional interference, or the ability to inhibit emotional interference in order to make a correct response. Trials are presented for 2000ms with 500ms ITIs. Participants completed two runs of this task lasting approximately five minutes each with eight task blocks (four 2-Back, four 0-Back) and four fixation blocks. Only negative and neutral face trials from the 0-Back condition were used in the present analyses to specifically probe patterns of activation associated with emotional interference as opposed to cognitive load.

Stop Signal Task (SST). The SST is an established measure of motor inhibition that requires participants to repeatedly respond to a "Go" cue and inhibit responses when a "Stop" cue appears. In this task, participants indicated the direction of an arrow facing left or right (Go

Trials) or withhold responses when the left or right arrow is followed by a "Stop" cue (upward arrow; Stop trials). Initially, Go-trials were terminated after 1000ms if a response was not made. Stop cues were presented 300ms after presentation of left or right arrow cue and terminated after 300ms. Task was adapted in real time, such that a tracking algorithm decreased or increased trial durations by increments of 50ms to maintain an accuracy of 50% successful and 50% unsuccessful stop trials and 80% successful go trials for each participant. Intertrial intervals (ITIs) were presented for variable durations between 700-2000ms with fixation crosses superimposed on black backgrounds. Participants completed two runs of this task with 180 trials (150 Go/ 30 Stop trials) each. This task was used to identify patterns of PFC activation associated with non-emotional motor inhibition during the failed stop vs. go trials contrast.

fMRI Preprocessing and Analysis Methods. Imaging protocols were harmonized across ABCD sites and scanners (Casey et al., 2018b; Hagler et al., 2019). Preprocessing of whole brain functional MRI data followed a standardized preprocessing procedure previously used for preprocessing of ABCD Data (see Appendix 1 modified from Sripada et al., 2021), using fMRIPrep version 1.5.0 including fieldmap correction, coregistration to the T1 anatomical image, and realignment. Functional data were projected onto fsaverage surface space and normalized to the MNI brain template in volume. Nuisance regressors (i.e., including five principal components from cerebrospinal fluid, and five from white matter) were calculated from the MNI volume image.

For the present analyses, surface data and subcortical volume data were then combined into a Connectivity Informatics Technology Initiative (CIFTI) image in the standard HCP space (HCP 32k, with 91,282 grayordinates). Images were then scaled to have a grand mean of 10,000 and entered into first level processing for task-based analyses. CIFTI formatted files were used

rather than traditional Neuroimaging Informatics Technology Initiative (NIFTI) files. CIFTI formatted files are comprised of both 2-dimensional cortical surface data and 3-dimensional subcortical volumetric data, while NIFTI files only contain volumetric data (Dickie et al., 2020). Therefore, CIFTI files provide benefits including greater statistical sensitivity, elimination of issues related to spatial smoothing with volumetric data, and greater fidelity to the anatomical surface. Although it is possible to map subcortical regions using standard MNI coordinates, this coordinate system is not recommended for cortical surface data. Subject-level differences in cortical surface structure result in lack of correspondence with volume-based coordinates. Instead, studies including CIFTI data in analyses have reported correspondence (i.e., % overlap between data and regions defined by atlas) with different cortical atlases to provide greater accuracy in identification of regions of activation (See Liu et al., 2020; Sheng et al., 2019).

A fixed-effects general linear model was used to assess whole brain neural activation during Stop Trials vs. Go Trials for the SST and during negative (fearful) vs. neutral face trials during the 0-Back condition of the EN-Back Task. Whole brain analyses were conducted for both tasks and cluster peaks were identified using a) the Desikan-Killiany Tourville Atlas (DKT; Klein & Tourville, 2012); b) the HCP Multimodal Parcellation (HCP-MMP 1.0; Glasser et al., 2016); and c) the Yeo 7-Network Atlas (Yeo et al., 2011). Based on regions identified across these three atlases, peak vertices in PFC were identified and then individually reviewed via visual inspection using the Human Connectome Project Workbench.

Conjunction Analysis. Given prior evidence that the SST and EN-Back may activate similar inhibitory control regions, we aimed to establish shared activation between tasks. A conjunction analysis was conducted to identify overlapping patterns of activation between the

EN-Back Task and SST. The conjunction was calculated using maps of T scores from both the EN-Back Task and SST (threshold at $p < 0.05$, FDR corrected, 10,000 permutations).

Results

Consistent with Liu et al., (2020), for each task activation, we report the Area ID, peak statistical value, vertex number, and overlap with the DKT Atlas, and the HCP Multimodal Parcellation (HCP-MMP 1.0; Glasser et al., 2016; Table III.4). Results beyond the scope of these analyses are outlined in Appendix 2.

Table III.4
FDR Corrected Cortical Results

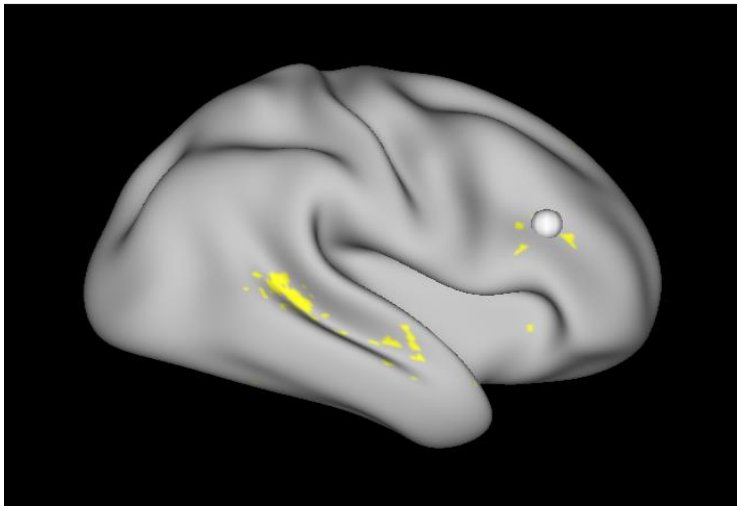
Hemisphere	Area ID (mm^2)	Peak T	Vertex	Cluster Overlap: DKT Atlas (Desikan et al., 2006)	Cluster Overlap: HCP MMP Atlas (Glasser et al., 2016)
EN-Back Task- Negative vs. Neutral Face Trials					
R	10	4.236	19070	Rostral Middle Frontal (100%)	IFSp (Inferior Frontal; 100%)
Stop Signal Task- Stop vs. Go Trials					
L	4	7.19	2767	Superior Frontal (29.9%); Precentral (22.1%); Insula (14.0%); Pars opercularis (14.0%); Caudal Middle Frontal (6.9%); Caudal Anterior Cingulate (5.7%);	6r (Rostral Area 6; 9.7%); 6a (Anterior Area 6; 8.1%); FOP4 (Frontal Opercular Area 4; 7.3%); SCEF (Supplementary and Cingulate Eye Field; 7.0%); 6ma (Anterior Area 6m; 5.6%);

R	32	7.296	2903	Superior Frontal (76.5%); Caudal Anterior Cingulate (14.6%); Caudal Middle Frontal (5.9%);	6ma (Anterior Area 6m; 16.6%); SCEF (Supplementary and Cingulate Eye Field; 15.6%); 6a (Anterior Area 6; 11.5%); p32pr (Prime Area p32; 10.7%); SFL (Superior Frontal Language Area; 10.6%); a32pr (Prime Anterior Area 32; 10.5%); 8BM (Area 8BM; 9.1%);
R	40	4.971	19492	Pars Opercularis (34.3%); Insula (24.6%); Precentral (18.6%); Lateral Orbitofrontal (10.2%); Pars Triangularis (7.9%);	6r (Rostral Area; 21.7%); AVI (Anterior Ventral Insular Area; 11.4%); FOP4 (Frontal Opercular Area 4; 10.9%); MI (Middle Insular Area; 9.1%); 44 (Area 44; 8.7%); FOP5 (Frontal Opercular Area 5; 8.1%); PEF (Premotor Eye Field; 5.5%);
Conjunction					
R	7	3.046	19174	Parsopercularis (100%)	IFJa (Inferior Frontal; 100%)

EN-Back Task. Results demonstrated greater activation during negative compared to neutral trials on 0-Back trials in the right rostral middle frontal gyrus (100% overlap; Figure III.1).

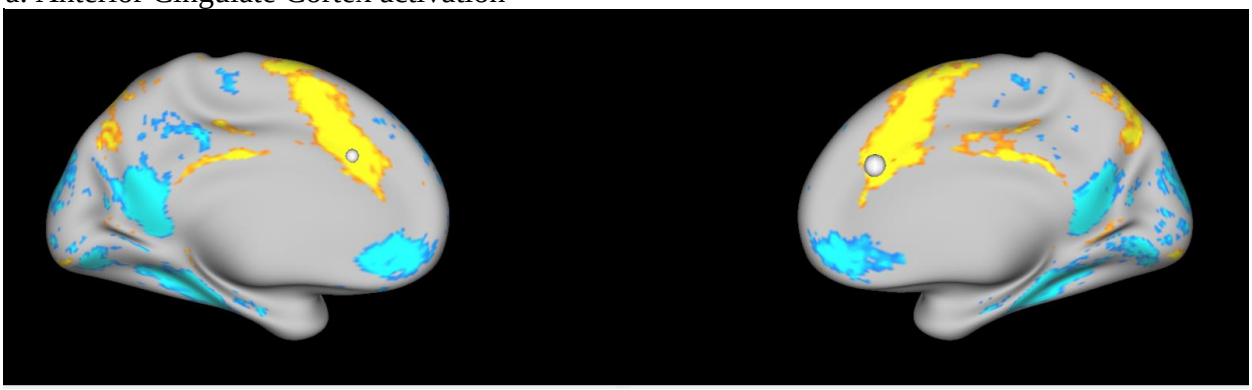
Stop Signal Task. For the Stop vs. Go Trial contrast on the SST,

Figure III.1
Neural activation during the EN-Back Task

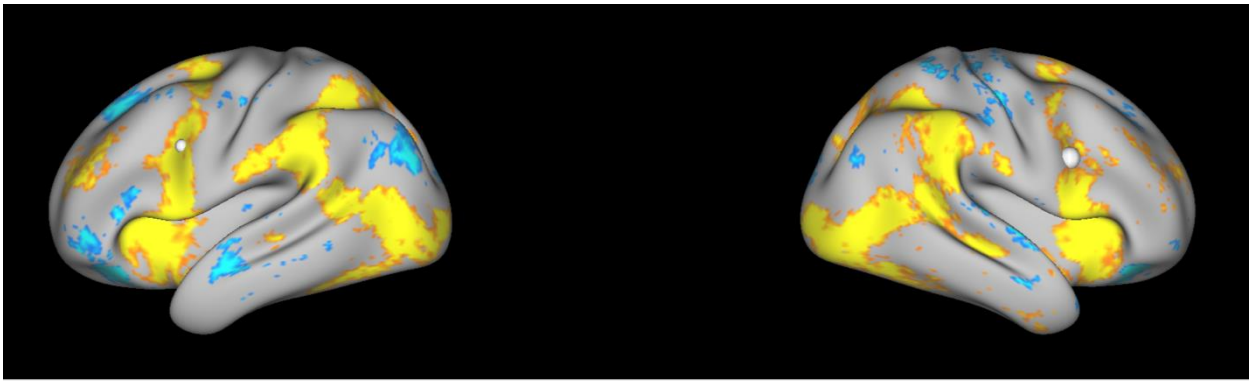


results revealed greater activation in regions including the bilateral superior frontal gyrus (Left: 29.9% overlap; Right: 76.5% overlap), caudal anterior cingulate (Left: 5.7% overlap; Right: 14.6% overlap), caudal middle frontal gyrus (Left: 6.9% overlap; Right: 5.9% overlap), left precentral (22.1% overlap), left insula (14.0% overlap), and the left pars opercularis (14.0% overlap) for stop compared to go trials (Figure III.2a and b).

Figure III.2
Neural activation during the SST
a. Anterior Cingulate Cortex activation



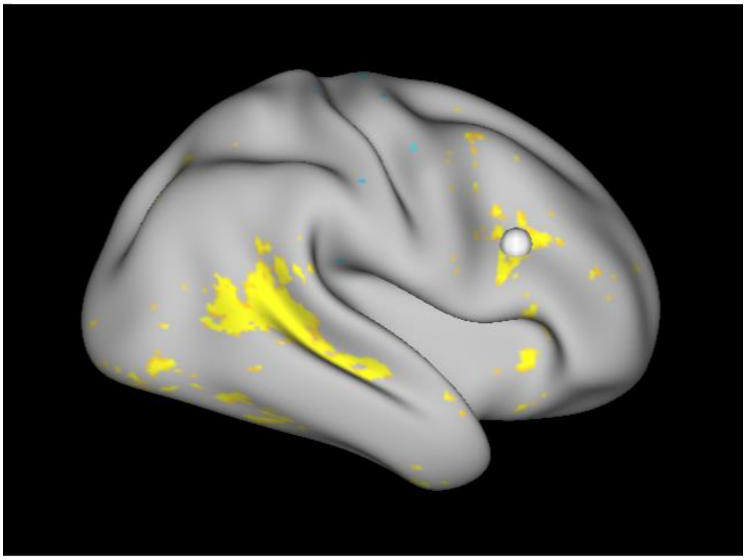
b. Dorsolateral prefrontal cortex activation



Conjunction Analysis.

Results of the conjunction analysis revealed common patterns of activation across both tasks in the right pars opercularis (100% overlap; Figure III.3).

Figure III.3
Results of the Conjunction Analysis



Discussion

The purpose of this study was to examine overlapping and distinct patterns of PFC activation associated with emotional interference and motor inhibition processes within this sample of pre-adolescents. A growing body of work has centered on the identification of neurobiological factors early in development that may forecast future clinical symptoms and contribute to diagnostic and prevention efforts. PFC function and its role in multiple facets of inhibitory control has been of particular focus for its relevance across the spectrum of psychological conditions. Despite the longstanding focus on neurobiological mechanisms associated with multiple forms of inhibitory control, findings have been mixed. The ACC (Stollstorff et al., 2013; Sun et al., 2020) and dlPFC (Kohn & Fernández, 2020; Mueller et al., 2017; Mullin et al., 2012; O'Brien et al., 2020b) are often linked to emotional interference; however, studies have yielded inconsistent patterns of activation in these regions (See Table III.1). In contrast, the SMA and IFG have been well established as regions involved in motor

inhibition (Rubia et al., 2001b), but some research has also implicated regions commonly associated with emotion (e.g., ACC, Rubia et al., 2001; dlPFC, Nee et al., 2007). Therefore, it remains unclear which prefrontal regions may be commonly involved across multiple forms of inhibitory control, and which regions may be involved in specific emotional and non-emotional processes.

Our results demonstrated activation in regions encompassing bilateral dACC, superior frontal gyrus, insula, and dlPFC during the SST, activation in the rostral MFG during the EN-Back Task, and common activation across both tasks in the IFG. SST activation was classified by cortical atlases across multiple prefrontal areas, with activation consistent with the dACC (Figure III.2a) and dlPFC (Figure III.2b) as reported in prior studies probing cognitive control (For comparable regions of activation in ACC and dlPFC, see Mullin et al., 2012). The region of activation common to both tasks was consistent with the right IFG as reported in prior studies (for a comparable region of activation in IFG, see Hughes et al., 2013).

Contrary to our hypotheses for the EN-Back task, we only found activation in the MFG during this task. Activation in the MFG has not previously been found during the EN-Back negative emotion contrast. However, activation in MFG has been reported in studies including similar emotion-based tasks (i.e., task-irrelevant emotional distractors, particularly negative compared to neutral face distractors; Colich et al., 2017; Kennedy et al., 2021; Wang et al., 2008), suggesting that it could play an important role in management of emotional interference. Previous literature has suggested that the MFG directs attentional control between top-down and bottom-up processing (Japee et al., 2015). Greater disengagement from negative affective faces has also been associated with greater resting state and task based functional connectivity between MFG and regions such as the fusiform gyrus (Bae et al., 2019) and amygdala (Sebastian et al.,

2017). Therefore, one interpretation of this finding could be that the MFG may contribute to disengagement from irrelevant emotion information and reengagement with the primary working memory task.

We predicted that activation in ACC and dlPFC would be associated with emotional interference and may be shared between tasks. The ACC and dlPFC have been linked to both emotional and non-emotional interference processing due to their roles in conflict monitoring and attention to task-relevant information respectively (For meta-analysis, see: Xu et al., 2016). Surprisingly, we found that these regions were only engaged during the SST. We examined activation solely during 0-Back trials to target PFC activation associated with emotion, rather than cognitive load; however, it is possible that an interaction between higher cognitive load (e.g., 2-Back) and emotion is required to sufficiently engage ACC and dlPFC. Notably, a previous study using EN-Back data from the ABCD sample showed reduced activation in both ACC and dlPFC during emotional compared to neutral faces when both 0-Back and 2-Back trials were included (O'Brien et al., 2020b).

Additionally, we hypothesized that the IFG would be involved in motor inhibition but not emotional interference. It is interesting then, that activation in this region was common across both tasks. This finding suggests that functions governed by the IFG are central to both preventing emotional interference and effectively inhibiting a motor response. Although the IFG has predominantly been implicated in non-emotional motor inhibition (Nee et al., 2007b), activation in this region has also been demonstrated during emotional variants of multiple motor inhibition tasks (for meta-analysis, see Cromheeke & Mueller, 2014). The IFG has been implicated in suppression of both task irrelevant information (e.g., emotion) and suppression of motor responses, particularly with increased task difficulty (Hughes et al., 2013). Our finding

suggests that the IFG could be involved in the shared process of overriding both emotion and prepotent response behavior.

Finally, we expected that activation in ACC and dlPFC may be shared across tasks given some prior evidence implicating these regions in motor inhibition (Rubia et al., 2001a). Previous studies have predominantly reported reduced activation associated with failure to inhibit responses, particularly in clinical samples (Bourque et al., 2017; Luijten et al., 2014; Moran et al., 2018). In contrast, we found increased activation in ACC and dlPFC during failed inhibition. One study demonstrated increased activation in ACC for adolescents without, compared to those with, ADHD during unsuccessful inhibition, and increased dlPFC activation across all subjects during stop vs go trials regardless of successful inhibition (Pliszka et al., 2006). Our finding aligns with baseline activation patterns reported in the ABCD sample; increased activation in ACC and dlPFC have been reported during both correct and incorrect stop vs. go trials (Chaarani et al., 2021). It is possible that differences in activation in these regions associated with failed inhibition could emerge later on in development (Larsen & Luna, 2018). Another possible interpretation surrounding increased ACC activity during failed inhibition could be that failed inhibition engages both cognitive and emotional functions. One study demonstrated that increased ACC activity during incorrect stop trials on the SST was associated with greater subjective frustration associated with performance (Spunt et al., 2012). In summation, these findings provide greater insight into prefrontal control unique to managing emotional interference and non-emotional motor inhibition and shared across tasks.

Limitations. Findings from the present analyses are subject to task-based limitations. For one, the primary function of the EN-Back Task is to examine working memory across cognitive load conditions, rather than measuring interference of emotionally salient stimuli. Previous

studies using this task have demonstrated findings in prefrontal regions specific to emotion contrasts (e.g., fearful or negative vs. neutral faces; Mueller et al., 2017; Mullin et al., 2012; O'Brien et al., 2020; Sun et al., 2020). However, effects of emotion may be constrained by limited salience of emotional face stimuli, lack of explicit instructions to attend to emotion (i.e., capturing implicit emotional processing only), and cognitive load diluting emotional effects. Future studies comparing non-emotional vs. emotional inhibitory control may include behavioral tasks explicitly probing or manipulating emotion (e.g., fear conditioning, emotion recognition paradigms), or tasks that employ more salient stimuli such as the International Affective Picture System (IAPS; Lang et al., 2005). Use of an explicit emotion task may reveal distinct prefrontal activation associated with emotional but not unemotional inhibitory control.

Future Directions. These analyses were conducted on baseline data from a longitudinal sample. Therefore, these analyses may be repeated when this cohort is older to probe within-subject developmental changes in PFC function associated with emotional interference and motor inhibition. Furthermore, these analyses were specifically focused on prefrontal regions due to their clinical relevance to impulsivity and inhibitory control; however, there were findings of significant activation in regions beyond the scope of this study (Appendix 2). Future studies using this longitudinal sample should examine functional connectivity between top-down PFC control and bottom-up regions (e.g., amygdala; Gee et al., 2013; Gold et al., 2016; Wu et al., 2016). Network-based analyses would provide additional context to understanding mechanistic drivers of inhibitory control involved in emotional and non-emotional tasks.

Conclusions. The present study aimed to explore patterns of PFC function associated with both emotional and motor inhibition processes. PFC function has been unequivocally linked to inhibitory control processes; however, in line with findings from self-report data outlined in

Chapter 2, these results reveal commonalities in neural function associated with emotional and non-emotional processing. Use of secondary data from the ABCD Study was critical to addressing our aims, as it allowed for comparison of task activation within the same sample, and provided a large, well powered sample to adequately capture potential differences in PFC function. In addition, an early developmental sample allowed us to capture patterns of PFC function that could predict ongoing development of clinical symptoms as this cohort ages. Finally, these analyses were conducted using cortical surface data via the CIFTI file format. Cortical data more closely adheres to the surface structure of the brain than traditional volumetric averages, providing greater accuracy in mapping activation. In summation, these findings provide support for the unique involvement of the MFG in emotional interference and dACC and dlPFC in motor inhibition. In addition, we identified the IFG as a region common to both emotional and non-emotional inhibition. These analyses lay the foundation to explore relationships between these patterns of PFC activation and clinical symptoms in Chapter 4, and track changes in these patterns of activation as the ABCD Study cohort ages.

Chapter IV:

Relationships between Negative Urgency and Prefrontal Activation Associated with Emotional Interference and Motor Inhibition

As posited in Chapter 1, negative urgency may be best positioned to capture the intersection of anxious and impulsive clinical presentations due to its relevance to negative emotion and externalizing disorders. In Chapter 2, we probed relationships between anxiety and dimensions of impulsivity, including negative urgency, and examined effects of their interactions on the outcome of experiential avoidance. Building on these analyses focused on clinical symptoms, we now aim to understand relationships between negative urgency and neural function underlying forms of inhibitory control relevant to both anxiety and impulsivity. In Chapter 3, we uncovered differences in PFC function associated with inhibitory control governing emotional interference (relevant to anxiety) and non-emotional motor inhibition (relevant to impulsivity). Relationships may differ between these patterns of PFC activation and trait negative urgency.

In this chapter, we examine relationships between anxiety symptoms, impulsive traits (including negative urgency) and PFC function associated with emotional interference and motor inhibition. Specifically, we explored the indirect effects of 1) anxiety on MFG activation associated with emotional interference through negative urgency and global impulsivity (Figure IV.1); and 2) global impulsivity on both ACC and dlPFC activation associated with motor

inhibition through negative urgency and anxiety (Figure IV.2). Examining these relationships with negative urgency will provide additional mechanistically driven insight into whether and how this construct functions as a conduit between anxiety and global impulsivity.

Neural Function Associated with Negative Urgency

In addition to similarities between behavioral presentations of negative urgency, anxiety, and global impulsivity, emerging literature interrogating the neural correlates of negative urgency has mirrored neurocognitive deficits associated with anxiety and impulsivity. Relationships between trait negative urgency and neural function have predominantly been examined in the context of substance use. Thus, regions involved in reward salience including the dorsal striatum and caudate have been linked to negative urgency. In keeping with conceptualizations of neural function associated with other forms of psychopathology, negative urgency has been conceptualized as a failure of top-down regulation over emotion (Cyders et al., 2014). Like anxiety, negative urgency has been associated with hyperactivation in emotion processing regions (e.g., insula, amygdala; Xiao et al., 2013), and reduced activation in PFC regions (e.g., lateral orbitofrontal cortex; OFC, vmPFC). In absence of SUD, few studies have examined relationships between negative urgency and PFC inhibitory control over emotional interference and motor inhibition. However, behavioral similarities between negative urgency and both anxiety and impulsivity, as well as PFC functional similarities in both anxiety and impulsivity, may point to a similar association between negative urgency and reduced PFC inhibitory control.

Negative Urgency and Emotional Interference

As previously outlined, PFC regions implicated in managing emotional interference (i.e., disengaging from irrelevant emotion distractors) include the ACC (Stollstorff et al., 2013),

dlPFC (Kohn & Fernández, 2020), and MFG (Colich et al., 2017; Japee et al., 2015). Neural function associated with emotional interference has relevance to anxiety. Individuals with anxiety show heightened attention to negative stimuli (Goodwin et al., 2017b) and reduced emotion regulation (Picó-Pérez et al., 2017). On cognitive tasks including negative stimuli, individuals with anxiety demonstrate poorer performance or slower reaction times to maintain performance when compared to healthy controls (Ladouceur et al., 2005b; Villemonteix et al., 2017). Imaging studies employing emotional interference tasks have linked anxiety with reduced activation in emotion regulation regions (e.g. superior frontal gyrus, ACC, MFG), hyperactivation in emotion processing regions (e.g., amygdala), and decreased functional connectivity between PFC and amygdala (Fitzgerald et al., 2019; J. Li et al., 2020a).

Negative urgency has been associated with similar patterns of neural function and in turn, we anticipated similar relationships with PFC control over emotional interference as seen with anxiety. Some previous literature examining neural function associated with negative urgency mirrors the dual process model of neural activation associated with emotional interference in anxious individuals. Findings suggest reduced activation in PFC including orbitofrontal cortex (OFC) and anterior cingulate cortex (ACC; Smith & Cyders, 2016a) and increased activation in amygdala and reward processing regions (e.g. striatum) are associated with negative urgency. However, other findings reflect a compensatory model of hyperactivation in PFC regions in addition to emotion processing regions, potentially to manage emotional interference. Previous literature has reported positive relationships between negative urgency and greater activation in the OFC and amygdala (Cyders et al., 2015b) in healthy social drinkers and greater activation in vlPFC in addition to insula and dorsal striatum (Chester et al., 2016; Cyders et al., 2015b) in healthy participants.

Negative Urgency and Motor Inhibition

Although reduced PFC inhibitory control over emotional interference may be more intuitively associated with psychopathology, previous literature has also demonstrated differences in regions associated with motor inhibition in clinical samples. Poor performance on motor inhibition tasks, even in absence of emotionally salient stimuli, has been associated with multiple forms of psychopathology including externalizing disorders (e.g. ADHD) and anxiety (Lipszyc & Schachar, 2010b). Inhibitory control during motor inhibition tasks has been probed as a neural proxy for the personality trait of impulsivity (Roberts et al., 2011). As a dimension of impulsivity, negative urgency may also be associated with deficits in motor inhibition irrespective of emotional processing. Across healthy and clinical samples, motor inhibition is associated with motor control regions (e.g., SMA), and the IFG (Weafer et al., 2019). Some studies have also implicated the ACC and dlPFC in motor inhibition (Nee et al., 2007b; Rubia et al., 2001b). To the best of our knowledge, only two studies thus far have examined relationships between negative urgency and neural activation during non-emotional versions of a motor inhibition task (the SST). One study demonstrated a relationship between negative urgency and reduced activation in the IFG during Stop vs. Go Trials in participants with no psychopathology (Wilbertz et al., 2014). However, another study demonstrated no relationship between task activation in the IFG and negative urgency during this contrast in participants with anxiety or depression and cannabis use disorder (Spechler et al., 2020b).

Negative urgency has relevance to multiple dimensions of psychopathology, across existing diagnostic categories, and may reflect central underlying deficits in PFC function. Negative urgency may capture specific impulsive actions that could contribute to the relationship between anxiety and PFC activation specific to a lack of emotion regulation. Conversely,

negative urgency may capture the specific role of negative emotion in the relationship between global impulsivity and PFC activation specific to inhibitory control. Examining the role of negative urgency in relationships between clinical symptoms and these neural processes will provide additional insight into the potential position of negative urgency at the intersection of impulsivity and anxiety.

Rationale for Present Sample

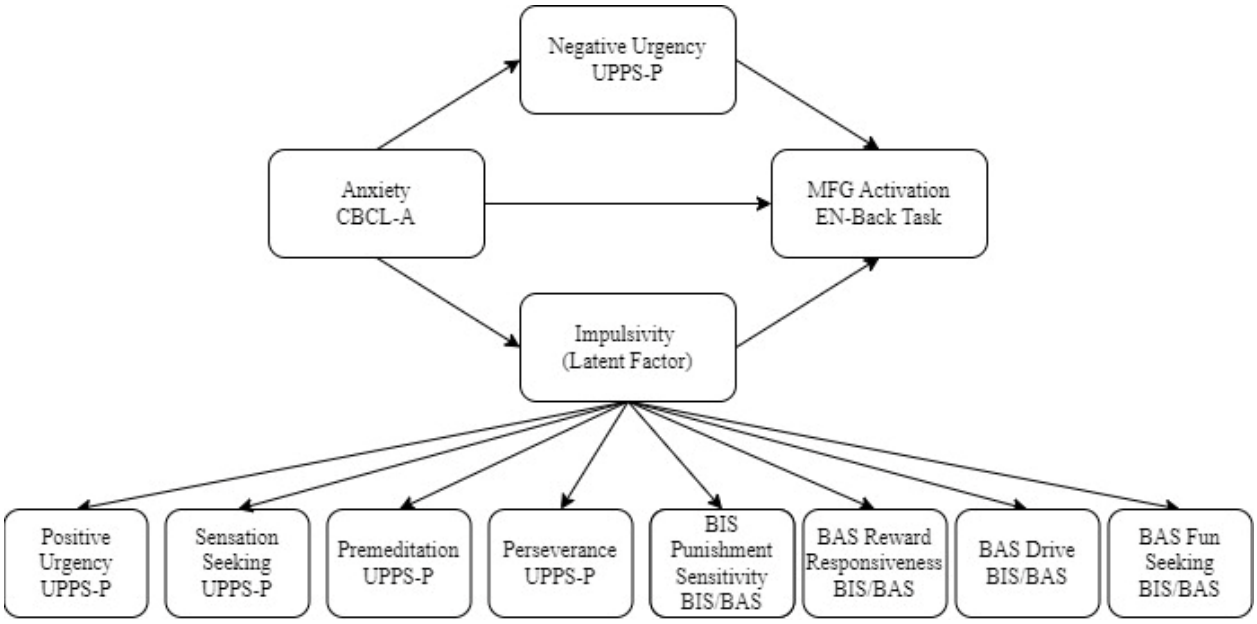
As previously outlined, use of the ABCD Dataset allowed for comparisons of PFC activation across common probes of emotional interference and motor inhibition in a large sample of early adolescents. Similarly, negative urgency has previously been identified in early adolescent samples as a risk factor for development of later psychopathology, namely substance use disorders (Settles et al., 2012; Smith & Cyders, 2016b). Negative urgency has also been found to mediate relationships between anxiety disorders and substance use behaviors in adolescence (Wolitzky-Taylor et al., 2016). Thus, an early adolescent sample allowed us to capture variation in negative urgency, anxiety, and global impulsivity and their relationships with PFC function.

Specific Aims

The conjunction map generated in Chapter 3 revealed unique patterns of activation specific to 1) emotional interference (the negative vs. neutral face contrast of the EN-Back Task) in the MFG and 2) motor inhibition (Stop vs. Go Trials on the SST) in the ACC and dlPFC. In order to better characterize the position of negative urgency among anxious and impulsive traits related to inhibitory control, task-based PFC function and indicators of anxiety and impulsivity were subjected to structural equation models.

We first examined the indirect effect of anxiety on activation in the right MFG specific to emotional interference on the EN-Back Task through negative urgency and additional indicators of impulsivity (See Figure IV.1 for conceptual model of relationships). We hypothesized that anxiety would be inversely related to MFG activation during negative compared to neutral face trials and that negative urgency would be the dimension of impulsivity that explained the greatest variance in these relationships.

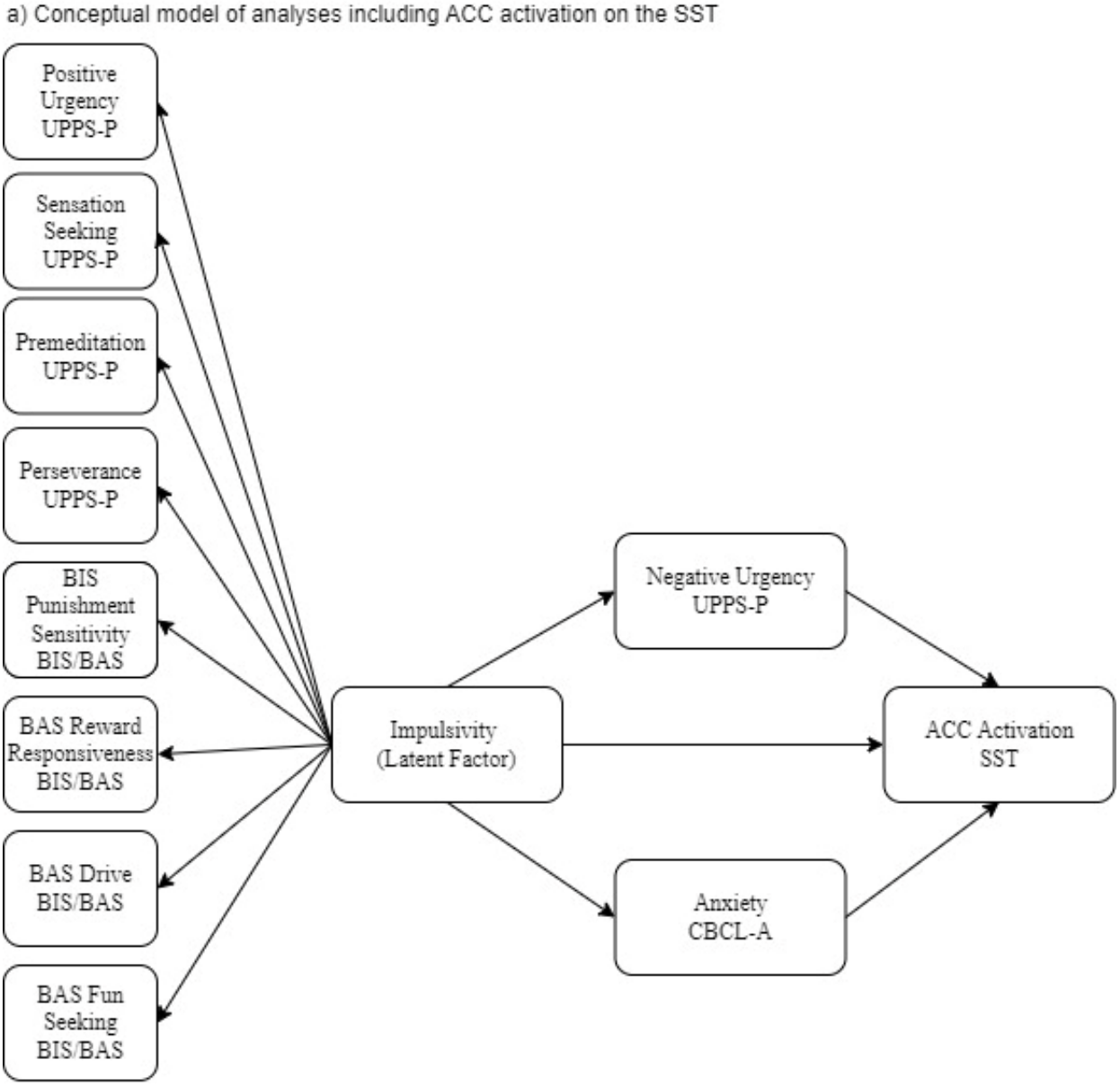
Figure IV.1
Conceptual model of relationships between anxiety, impulsivity, and activation on the EN-Back Task

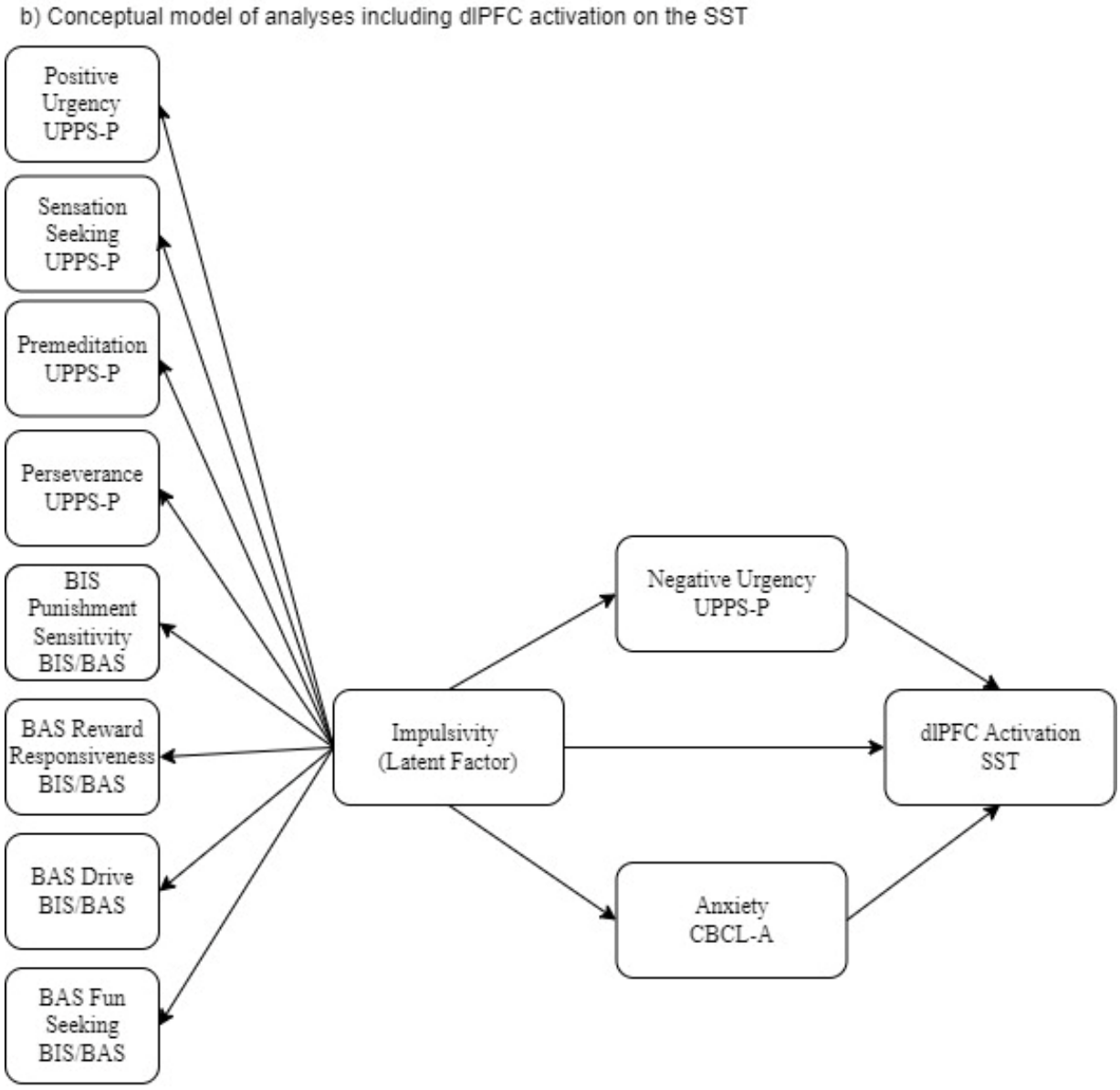


Similarly, we used indicators of anxiety and negative urgency to examine the role of negative emotion in the relationship between non-emotional dimensions of impulsivity and activation specific to motor inhibition on the SST in both the bilateral caudal ACC and dlPFC (See Figure IV.2 for conceptual model of relationships). Although anxiety has previously been associated with performance and neural activation on the SST, we anticipated that the indirect

effect of non-emotional global impulsivity on motor inhibition would be stronger through negative urgency than anxiety.

Figure IV.2
Conceptual model of relationships between anxiety, impulsivity, and activation on the SST





Method

Participants

Psychometric and neuroimaging data from the same participants in the ABCD dataset used in Chapter 3 were included in the present analyses. For full exclusion criteria for the current sample, see Table III.3.

Procedures

Assessment. For full ABCD Study procedures, see (Barch et al., 2018a; Garavan et al., 2018). In addition to MRI procedures outlined in Chapter 3, participants and parents were instructed to complete a series of behavioral surveys including measures of anxiety and trait impulsivity (For survey characteristics, see (Barch et al., 2018b; Watts et al., 2021a).

The Behavioral Inhibition System/Behavioral Activation System Scale (BIS/BAS; Carver & White, 1994). The BIS/BAS is a well-established measure of behavioral inhibition and approach that participants responded to using a Likert-type scale. The youth version of the BIS/BAS modified for use in the ABCD Study included 20 items (For full details, see Pagliaccio et al., 2016). Subscales include one BIS (Punishment Sensitivity) subscale and three BAS subscales (Drive, Reward Responsiveness, and Fun Seeking). Items were summed separately for subscales.

Short Form of The Urgency, Premeditation (lack of), Perseverance (lack of), Sensation Seeking, Positive Urgency, Impulsive Behavior Scale- Children, ABCD Version (S-UPPS-P-C; (Watts et al., 2021b)**.** The full version of the UPPS-P is a well validated measure of dimensional impulsivity that has been widely used in adult samples. The Short Form of the UPPS-P for Children was developed specifically for the ABCD Study sample and includes four items (modified to be appropriate for children) from each of the original subscales (Negative Urgency, Lack of Premeditation, Lack of Perseverance, Sensation Seeking, Positive Urgency; Barch et al., 2018). Using the ABCD sample, Watts et al., (2020) found that the short form demonstrated the same factor structure as the original scale, and established convergent and discriminant validity with measures of psychopathology (e.g. K-SADS-5, CBCL) and impulsivity (e.g. BIS/BAS). Items for each subscale were summed separately.

The Child Behavior Checklist (CBCL (Achenbach, Thomas, 2013)**.** The CBCL is an established measure used to assess parent-reported anxiety symptoms in children between the ages of 6-18. The CBCL-A is a DSM-oriented subscale including 10 statements that parents of participants responded to on a Likert-type scale (Kendall et al., 2007).

Analytic Strategy. Beta weights from the right MFG for negative vs neutral 0-back trials on the EN-Back task and both caudal ACC and dlPFC for stop vs go trials on the SST were included in cross-sectional structural equation models. First, we conducted an EFA including multiple indicators of impulsivity (i.e., subscales of the UPPS-P and BIS/BAS excluding negative urgency) to establish the factor structure of a latent factor of impulsivity. The latent factor of impulsivity, informed by the EFA, was included in subsequent confirmatory factor models. In the first CFA, parent-reported anxiety was used as the predictor variable, negative urgency and the latent factor of impulsivity served as mediators, and activation in the MFG on the EN-Back task was the outcome variable. Two CFAs were conducted using PFC activation on the SST to examine the effects of emotion-based constructs (i.e., negative urgency, anxiety) on the relationship between global impulsivity and motor inhibition. In these models, the latent variable of impulsivity was the predictor variable, and negative urgency and anxiety served as mediators. ACC activation on the SST was included as the outcome variable for one CFA and dlPFC activation on the SST was included as the outcome variable on the other CFA. Analyses were conducted using maximum likelihood estimation, and the 95% confidence interval for parameter estimates in the confirmatory factor model was computed using percentile bootstrap procedure with 10,000 random draws.

Sum scores were calculated for psychometric variables according to standardized scoring practices outlined for each measure. Descriptive statistics, correlations between variables of interest, and factor analyses were conducted in MPlus Version 8.4 (Muthén & Muthén, 1998).

Results

Descriptive Statistics. Descriptive statistics are provided in Table IV.1. Sum scores for measures of impulsivity were normally distributed and ranged from subclinical to clinical levels; however, anxiety scores were skewed heavily toward lower scores. Data were transformed to account for non-normal distribution of the data; while no transformations fully normalized the data, a log transformation yielded the strongest effect on distribution out of all transformations attempted. A constant of 1 was added to all CBCL Anxiety scores and scores were log transformed to account for non-normal distribution (See Figure IV.3 for histograms).

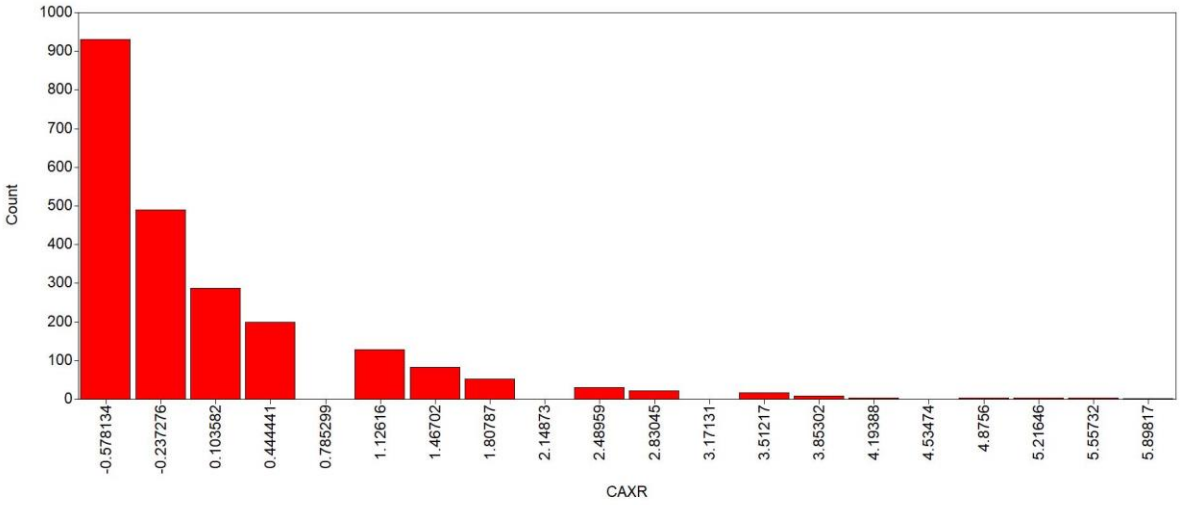
Table IV.1
Descriptive Statistics

Scale	Subscale	n	Mean	SD	Min	Max	Kurtosis	Skew	α
UPPS-P	1. Negative Urgency	2264	8.18	2.48	4.00	16.00	-0.38	0.28	0.64
	2. Positive Urgency	2264	7.48	2.66	4.00	16.00	-0.16	0.53	0.78
	3. Sensation Seeking	2264	9.88	2.56	4.00	16.00	-0.44	-0.04	0.49
	4. Lack of Premeditation	2264	7.52	2.11	4.00	16.00	0.94	0.64	0.74
	5. Lack of Perseverance	2264	6.66	2.00	4.00	16.00	1.03	0.82	0.68
BIS/BAS	6. BIS Punishment Sensitivity	2264	9.44	3.53	0.00	21.00	0.03	0.39	0.67
	7. BAS Reward Responsiveness	2264	8.71	2.21	0.00	15.00	0.01	0.16	0.30
	8. BAS Drive	2264	5.24	2.03	0.00	12.00	0.01	0.48	0.38
	9. BAS Fun Seeking	2264	5.79	2.27	0.00	12.00	-0.24	0.17	0.52
CBCL	10. DSM-Oriented Anxiety	2264	1.41	1.88	0.00	15.00	6.67	2.13	0.77

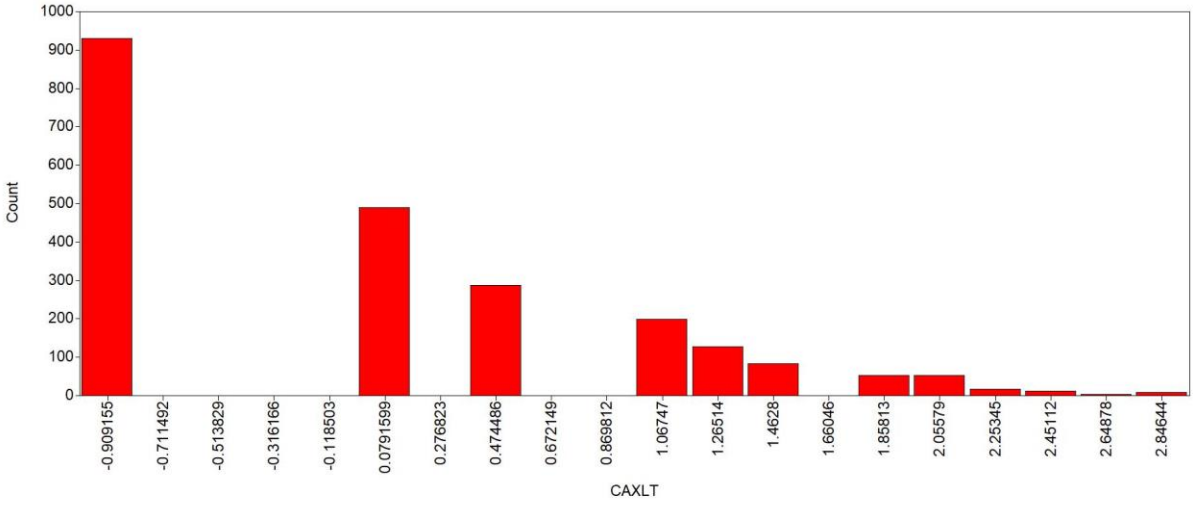
Note. SD = standard deviation.

Figure IV.3
Histogram of responses on the CBCL Anxiety scale

a) Raw data



b) log transformed data



All data were standardized (Z scores computed) to account for impact of differences in scales of psychometric measures and beta weights with respect to the BOLD signal from neuroimaging analyses. Intercorrelations between measures of impulsivity included in the EFA ranged from r = -0.15 (UPPS-P Lack of Perseverance and BIS Punishment Sensitivity) to r = 0.62 (BIS Punishment Sensitivity and BAS Fun Seeking; Table IV.2). Distributions and correlations were consistent with previous studies conducted using baseline behavioral data from the ABCD sample (Davis et al., 2019; Watts et al., 2021a).

Table IV.2
Zero-order correlation matrix

Scale	Subscale	1	2	3	4	5	6	7	8	9
UPPS-P	1. Negative Urgency	-								
	2. Positive Urgency	0.48	-							
	3. Sensation Seeking	0.11	0.14	-						
	4. Lack of Premeditation	0.19	0.20	0.08	-					
	5. Lack of Perseverance	0.16	0.18	-0.11	0.41	-				
BIS/BAS	6. BIS Punishment Sensitivity	0.23	0.19	0.27	-0.04	-0.15	-			
	7. BAS Reward Responsiveness	0.22	0.15	0.15	-0.07	-0.12	0.56	-		
	8. BAS Drive	0.23	0.21	0.11	-0.08	-0.05	0.60	0.45	-	
	9. BAS Fun Seeking	0.25	0.21	0.10	-0.02	-0.04	0.62	0.57	0.47	-
CBCL	10. DSM-Oriented Anxiety	0.03	-0.01	-0.04	-0.02	0.06	0.06	0.07	0.05	0.05

Exploratory Factor Analysis. In keeping with the analytic strategy for Chapter 2, we first conducted an EFA to determine the structure of a latent factor of impulsivity for inclusion in the subsequent CFA. A parallel analysis indicated a two-factor solution for the EFA above the

95th percentile estimates based on 100 simulated datasets. An EFA with maximum likelihood extraction with an oblique rotation revealed that indicators from the BIS/BAS and UPPS-P Sensation Seeking loaded robustly (i.e. greater than 0.35; Hair Jr et al., 2009) on one factor; while the other subscales of the UPPS-P loaded on a second factor. The model met conventional standards for good fit (χ^2 = 489.10, df = 19, p < 0.001; RMSEA = 0.11; SRMR = 0.05; CFI = 0.91; TLI = 0.83). Factors were significantly correlated (0.37) and were likely attributable to measure-specific effects (For factor loadings in the EFA, see Table IV.3).

Table IV.3
Factor loadings from exploratory factor analysis

	EFA	
	Factor 1	Factor 2
BIS Punishment Sensitivity	0.85*	-0.02
BAS Reward Responsiveness	0.69*	0.00
BAS Drive	0.65*	0.06*
BAS Fun Seeking	0.70*	0.09*
Negative Urgency	0.07*	0.70*
Positive Urgency	0.01	0.63*
Sensation Seeking	0.22*	0.07*
Premeditation	-0.28*	0.50*
Perseverance	-0.35*	0.50*

Note. EFA = exploratory factor analysis; STD = standardized factor loadings. Oblique, geomin rotation used in EFA; factor correlation *r* = 0.37.

Informed by results of this EFA, we sought to improve model fit of subsequent hypotheses-driven models. We conducted analyses including a) CFAs with a latent factor of impulsivity without specifying covariances between subscales, and b) CFAs with a latent bifactor model of impulsivity specifying covariances between subscales from each measure and sensation seeking with BIS/BAS subscales, per the EFA. Differences in fit were negligible and

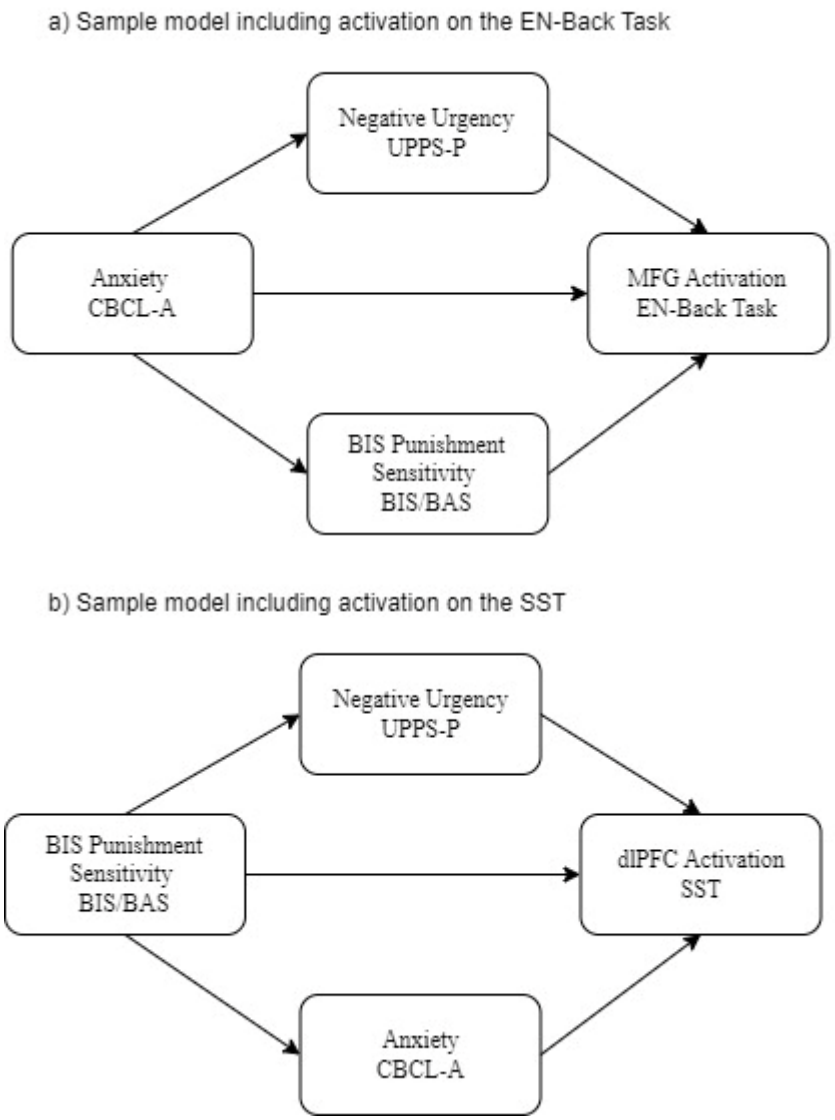
no models met conventional standards for good fit (Table IV.4). Subsequently, we report the results from our second approach including latent bifactor models of impulsivity in our primary CFAs.

In order to address possible low power for the proposed models, we followed up primary analyses with parsimonious analyses isolating dimensions of impulsivity (See Figure IV.4 for sample models). Model fit did not improve with more parsimonious models and results did not differ from those of the models originally proposed. Thus, we ascertained that issues of power or latent factor structure were not driving our results.

Table IV.4
Goodness of fit of confirmatory factor models

Model with task-based activation as outcome variable	Structure of Impulsivity Factor	$\chi2$(df)	p-value	RMSEA (CI)	CFI	TLI	SRMR
MFG	No Covariances Specified	1636.53 (42)	<0.001	0.13 [0.12, 0.14]	0.69	0.60	0.11
	Covariances Specified	1348.60 (36)	<0.001	0.13 [0.12, 0.13]	0.75	0.61	0.10
ACC	No Covariances Specified	1444.80 (42)	<0.001	0.12 [0.12, 0.13]	0.73	0.64	0.09
	Covariances Specified	1130.52 (36)	<0.001	0.13 [0.11, 0.12]	0.79	0.68	0.08
dlPFC	No Covariances Specified	1436.08 (42)	<0.001	0.12 [0.12, 0.13]	0.73	0.65	0.08
	Covariances Specified	1123.37 (36)	<0.001	0.12 [0.11, 0.12]	0.79	0.68	0.08

Figure IV.4
Samples of parsimonious confirmatory factor models



Relationships Between Anxiety, Impulsivity, and Emotional Interference.

Our first hypothesis posited that the indirect effect of anxiety on activation in the MFG during the EN-Back task would be stronger through negative urgency than global impulsivity. Therefore, a CFA was conducted using CBCL Anxiety scores as the primary predictor variable, the global impulsivity factor and negative urgency as mediators, and activation in the MFG

during the EN-Back Task as the outcome variable. Model fit did not meet conventional standards of good fit (χ^2 = 1348.60, df = 36, p < 0.001; RMSEA = 0.13; SRMR = 0.10; CFI = 0.75; TLI = 0.61). Results revealed a significant relationship between anxiety and global impulsivity (*b* = 0.06, *p* = 0.01). Indirect effects of global impulsivity and negative urgency were not significant; however, the direct effect of anxiety on MFG activation (*b* = 0.07, *p* = 0.001) and total effect (*b* = 0.07, *p* = 0.001) were significant (See Table IV.5 for full results).

Table IV.5
Results of the confirmatory factor model for emotional interference

	STD	95% CI
Impulsivity Factor (IMP)		
ANX → IMP	0.06	[0.01,0.11]*
IMP → MFG	0.03	[-0.02, 0.08]
Indirect Effect	0.002	[-0.002, 0.01]
Negative Urgency (NU)		
ANX → NU	0.02	[-0.02, 0.06]
NU → MFG	-0.02	[-0.06, 0.03]
Indirect Effect	0.00	[-0.001, 0.001]
Direct & Total Effects		
Direct Effect	0.07	[0.03, 0.11]*
Total Indirect Effect	0.001	[-0.002, 0.004]
Total Effect	0.07	[0.02, 0.11]*

Note: ANX= CBCL Anxiety; NU = Negative Urgency; MFG = Activation in middle frontal gyrus during the EN-Back Task; 95% CI = 95% confidence intervals for standardized coefficients; STD = standardized coefficients; * = p < 0.05. 95% confidence intervals derived via bias corrected bootstrap procedure with 10,000 random draws.

Relationships Between Anxiety, Impulsivity, and Non-Emotional Inhibition.

Similarly, we hypothesized that the indirect effect of global impulsivity on both dACC and dlPFC activation during the SST would be stronger through negative urgency than anxiety. In these models, the impulsivity factor was the primary predictor, the UPPS-P negative urgency subscale and CBCL anxiety were included as mediators, and dACC activation and dlPFC activation as outcome variables in each respective model.

dACC. Model fit did not meet conventional standards of good fit (χ^2 = 1130.52, df = 36, p < 0.001; RMSEA = 0.12; SRMR = 0.08; CFI = 0.79; TLI = 0.68). Results revealed significant positive relationships between global impulsivity and both anxiety (*b* = 0.06; *p* = 0.02) and negative urgency (*b* = 0.36; *p* < 0.001). Additionally, there was a significant inverse relationship between anxiety and ACC activation during failed inhibition (*b* = -0.05, *p* = 0.01). There were no significant total, direct, or indirect effects (Table IV.6).

Table IV.6
Results of the confirmatory factor model for ACC activation during motor inhibition

	STD	95% CI
Anxiety (ANX)		
IMP → ANX	0.06	[0.01, 0.10]*
ANX → ACC	-0.05	[-0.09, -0.01]*
Indirect Effect	-0.003	[-0.01, 0.00]
Negative Urgency (NU)		
IMP → NU	0.36	[0.32, 0.41]*
NU → ACC	0.02	[-0.02, 0.07]
Indirect Effect	0.01	[-0.01, 0.03]
Direct & Total Effects		
Direct Effect	-0.04	[-0.09, 0.01]
Total Indirect Effect	0.01	[-0.01, 0.02]
Total Effect	-0.03	[-0.08, 0.01]

Note: ANX= CBCL Anxiety; IMP = latent factor of impulsivity; NU = Negative Urgency; ACC = Activation in the ACC during the Stop Signal Task; 95% CI = 95% confidence intervals for standardized coefficients; STD = standardized coefficients; * = p < 0.05. 95% confidence intervals derived via bias corrected bootstrap procedure with 10,000 random draws.

dlPFC. Model fit did not meet conventional standards of good fit (χ^2 = 1123.37, df = 36, p < 0.001; RMSEA = 0.12; SRMR = 0.08; CFI = 0.79; TLI = 0.68). Results revealed significant positive relationships between global impulsivity and both anxiety (*b* = 0.06; *p* = 0.02) and negative urgency (*b* = 0.36; *p* < 0.001). There were no significant total, direct, or indirect effects (Table IV.7).

In a final effort to understand whether lack of findings may be driven by low reliability of child self-reported impulsivity, we conducted an exploratory analysis including parent-reported ADHD symptoms (i.e., CBCL ADHD syndrome subscale) as an indicator of impulsivity. These models demonstrated poorer fit than models including child-reported impulsivity.

Table IV.7
Results of the confirmatory factor model for dlPFC activation during motor inhibition

	STD	95% CI
Anxiety (ANX)		
IMP → ANX	0.06	[0.01, 0.10]*
ANX → dlPFC	-0.004	[-0.05, 0.04]
Indirect Effect	0.00	[-0.003, 0.002]
Negative Urgency (NU)		
IMP → NU	0.36	[0.32, 0.41]*
NU → dlPFC	-0.02	[-0.07, 0.02]
Indirect Effect	-0.01	[-0.03, 0.01]
Direct & Total Effects		
Direct Effect	0.02	[-0.04, 0.07]
Total Indirect Effect	-0.01	[-0.03, 0.01]
Total Effect	0.01	[-0.04,0.05]

Note: ANX= CBCL Anxiety; IMP = latent factor of impulsivity; NU = Negative Urgency; dlPFC = Activation in the dlPFC during the Stop Signal Task; 95% CI = 95% confidence intervals for standardized coefficients; STD = standardized coefficients; * = $p < 0.05$. 95% confidence intervals derived via bias corrected bootstrap procedure with 10,000 random draws.

Discussion

The present study capitalizes on multiple indicators of psychopathology and neural function from early adolescents in the longitudinal ABCD dataset to examine associations between anxiety, impulsivity, and PFC activation during emotional interference and motor inhibition. Notably, these results did not support our hypotheses that negative urgency would mediate links between 1) anxiety and neural function during emotional interference and 2) global impulsivity and neural function associated with non-emotional inhibition. However, our results demonstrated a significant positive relationship between anxiety and global impulsivity, consistent with prior findings linking anxiety and impulsivity (Elsey et al., 2016; Merz et al.,

2018). Additionally, we found a significant direct effect of anxiety on MFG responsivity to emotional interference, consistent with prior findings suggesting that anxiety is associated with greater activation in PFC regions during emotional interference (Li et al., 2020b; Veerapa et al., 2020). While anxiety was associated with both impulsivity and MFG activation, neither negative urgency nor global impulsivity mediated the relationship between anxiety and MFG activation. Neither dimensions of impulsivity, nor anxiety were linked to PFC activation associated with motor inhibition. This might suggest that anxiety and impulsivity play less of a role in non-emotional inhibitory control processes.

Examining relationships between neural function and symptoms of psychopathology in childhood is critical to inform efforts for early detection and intervention. Onset of both anxiety and externalizing disorders marked by high impulsivity (e.g., ADHD) often occur in early childhood and are highly comorbid (Friesen & Markowsky, 2021; Gordon-Lipkin et al., 2018). Our findings aligned with these previous findings, suggesting that anxiety and global impulsivity were positively associated. By contrast, the relationship between anxiety and negative urgency was not significant despite previous findings linking these constructs (Malivoire et al., 2019; Pawluk & Koerner, 2016). Previous literature has largely examined associations between anxiety and negative urgency in adult populations in the context of substance use and risky behaviors (Guillot et al., 2014; Menary et al., 2015). Therefore, it is possible that relationships between anxiety and the specific dimension of negative urgency may develop as this cohort ages.

Despite limitations outlined in Chapter 3 surrounding the utility of the EN-Back Task in probing emotional interference (e.g., limited salience of emotional stimuli, lack of instruction to attend to emotion), our findings revealed a significant direct effect of anxiety on MFG function during this task. This finding aligns with previous studies linking pediatric anxiety with

functional differences in the PFC prior to full maturation. Differences have been demonstrated between anxious and healthy pediatric samples in prefrontal regions such as the ACC and vlPFC, particularly during appraisal of threat or emotion information, although directionality of findings have been mixed (For review, see Strawn et al., 2021). Our finding corroborates previous evidence of greater PFC engagement in individuals with anxiety, perhaps as a compensatory function to manage emotional interference (Ladouceur et al., 2005b). Contrary to our predictions, global impulsivity and negative urgency did not mediate the relationship between anxiety and MFG activation.

Similarly, ADHD in early childhood has been linked to hypoactivity in frontoparietal and ventral attention networks during inhibition-related tasks (including ACC and dlPFC; for meta-analysis, see Cortese et al., 2012). Therefore, we would have anticipated that global impulsivity and negative urgency would have been associated with PFC function and mediated relationships between anxiety and PFC function. Lack of findings surrounding impulsivity may suggest that links between anxiety, impulsivity, and brain function during emotion-related inhibitory control may become stronger over the course of development. The lack of impulsivity-related findings may also reflect previous criticisms of correlational analyses between self-report data and neural indices of behavioral performance. Previous studies have argued that low reliability and discrepant factors associated with measurement of self-report and behavioral response data contribute to weak correlations between these metrics (Dang et al., 2020; Hedge et al., 2018). Child and parent reported measures assess average levels of constructs uniformly to capture between-subjects variability. Behavioral tasks capture “best” performance and within-subjects variability through contrasts between trial types. This methodological issue is common across studies examining relationships between psychological symptoms and task-based neural function

(Dang et al., 2020); however, it poses a particular obstacle when examining the multifaceted and ill-defined construct of impulsivity. Well-established fMRI tasks (e.g. SST) have frequently been employed as behavioral proxies for symptom or trait based indicators such as impulsivity. However, meta-analyses have demonstrated low correlations of 0.10 between various forms of self-reported and behavioral performance-based impulsivity (Cyders & Coskunpinar, 2012; Duckworth & Kern, 2011). Therefore, in spite of a large sample size and multiple indicators of both child and parent-reported impulsivity, low concordance between these measures likely obfuscated nuanced relationships between these constructs and contributed to lack of findings.

Limitations. Several factors should be considered when interpreting these findings. These analyses are limited by floor effects of psychometric data. In particular, CBCL scores across subscales, including anxiety, were skewed due to floor effects as many participants reported sum scores of 0. These floor effects are likely attributable to the age of participants at baseline data collection (Bevans et al., 2020). Although both anxiety and externalizing disorders associated with impulsivity have onsets in early adolescence, symptoms may be underreported by both children and parents due to limited insight or lack of consequences associated with symptoms. Therefore, the limited range of scores and non-normal distribution on a self-report measure of anxiety made it difficult to detect relationships between phenotypic and neural measures.

Additionally, it is possible that lack of findings surrounding impulsivity and PFC function in the present analyses may reflect challenges in capturing impulsive traits through self and parent report measures. As previously discussed, numerous measures of self and parent-reported impulsivity have been developed to capture nuances in dimensions of impulsivity (Carver & Johnson, 2018). Both the BIS/BAS and UPPS-P have been widely used as probes of

impulsivity; however, few studies have employed them in tandem. Similar to our findings, the previous studies that have used both of these measures have reported low to moderate correlations between their subscales (Threadgill & Gable, 2018; Watts et al., 2021b). Additionally, one study reported differences between subscales on the UPPS-P and BIS/BAS in relationships with the related construct of aggression (Miller et al., 2012). It is possible then that these results further illustrate possible differences in underlying constructs measured by these scales of impulsivity.

Future Directions. These analyses leverage a number of strengths that may lay the foundation for future longitudinal analyses using data from this sample. Future analyses including baseline data from the ABCD sample may leverage items from parent reported measures of conduct, behavioral problems, or social interaction as more meaningful indicators of early adolescent impulsivity or psychopathology. As the ABCD cohort ages, future analyses may also examine relationships between baseline PFC function established in these analyses and symptom development over time. Longitudinal analyses allow for further understanding of early childhood neural function as a prospective index of psychopathology. Analyses spanning development may also provide further insight into the age when child self-reported symptoms serve as more reliable indicators of psychopathology than parent report measures.

Conclusions. The present study did not demonstrate significant indirect effects of anxiety on MFG activation during emotional interference through impulsive dimensions. We also did not demonstrate indirect effects of global impulsivity on ACC or dlPFC activation during motor inhibition through emotional constructs of negative urgency and anxiety. These results provide insight into data and sample characteristics and may lay the foundation to explore relationships between neural function and psychopathology at subsequent time points. Future studies may

further examine relationships between PFC function and psychopathology as the ABCD cohort develops. Continued research in this domain will shed light on the role that negative urgency may play at the intersection of anxiety and impulsivity and its relationship to neural mechanisms underlying inhibitory control. This work has important implications for better defining a construct (i.e., impulsivity) that has relevance to both internalizing and externalizing pathology. Better characterizing relationships between anxiety and impulsivity may also move diagnostic and treatment efforts beyond the current limitations of categorical classifications.

CHAPTER V

Integration and Conclusions

Anxiety and impulsivity have been diagnostically siloed despite significant evidence of comorbidity and similarities in underlying neural mechanisms. Efforts to uncover behavioral and mechanistic similarities across different forms of psychopathology may provide a data-driven foundation for the reclassification of clinical conditions. However, these efforts have been complicated by broad and inconsistent definitions of impulsivity and inhibitory control (Strickland & Johnson, 2021). This dissertation project aimed to elucidate links between anxiety and impulsivity through the trait of negative urgency using both behavioral and neuroimaging data from adult and early adolescent samples.

The first study examined how experiential avoidance varies in association with both negative urgency and global impulsivity, using self-report data collected from adult participants. We replicated links between anxiety and experiential avoidance, as well as anxiety and both global impulsivity and negative urgency. Our results did not support initial hypotheses that high negative urgency in combination with high anxiety would be associated with greater experiential avoidance than high global impulsivity in combination with high anxiety. However, our follow up analyses including data-driven factors of impulsivity revealed that high anxiety and both emotion-based impulsivity and hyperactivity were associated with greater experiential avoidance, while high anxiety and less planning were linked to less experiential avoidance. The second study sought to reveal overlapping and distinct patterns of PFC-based inhibitory control

associated with management of emotional interference and motor inhibition in a sample of pre-adolescents from the ABCD Study. We demonstrated that emotional and non-emotional inhibitory control had both shared and distinct patterns of activation in the PFC. Specifically, emotional interference was associated with activation in the right MFG; motor inhibition was associated with activation in the bilateral dACC and dlPFC; and there was shared activation across both behavioral tasks in the IFG. The third study examined the role of negative urgency in relationships between the distinct patterns of activation identified in the second study, and child and parent reported anxiety and global impulsivity. We found a positive relationship between anxiety and global impulsivity and a significant relationship between anxiety and MFG responsivity to emotional interference. We found no significant interactions between anxiety, impulsivity, negative urgency, and PFC function on either task, which could be due to limited endorsement of anxiety and impulsivity symptoms, or lack of involvement of negative urgency in these relationships in pre-adolescents.

Results from both the first and second studies revealed differences in behavioral relationships and PFC function associated with distinct dimensions of impulsivity and inhibitory control. Study 1 showed significant indirect effects of anxiety on experiential avoidance through the dimensions of emotion-based impulsivity, hyperactivity, and (lack of) planning. Study 2 showed unique activation in MFG specific to management of emotional interference and activation in dlPFC and dACC specific to motor inhibition. These differences between distinct dimensions of impulsivity and inhibitory control in relationships with anxiety symptoms and in underlying PFC function underscore the importance of greater specificity in definitions of impulsivity. This is also consistent with prior literature suggesting that anxiety is associated with both emotional dimensions of impulsivity such as negative urgency (Altan-Atalay et al., 2020;

Pawluk & Koerner, 2016b), and non-emotional dimensions such as hyperactivity (Jacob et al., 2014; Reimherr et al., 2017) and motor inhibition (Grillon, Robinson, Krimsky, et al., 2017). Generalization of terminology across previous literature has likely contributed to mixed findings in relationships between impulsive dimensions and both psychological symptoms and patterns of neural function. Previous studies have asserted that impulsivity may serve as a broad term for related but distinct constructs (i.e., the jingle fallacy; Strickland & Johnson, 2021), while other studies have examined whether distinct impulsive dimensions such as negative and positive urgency may be measuring the same underlying construct (i.e., the jangle fallacy; Peterson & Smith, 2019). Our findings provide support for further research interrogating nuances in distinct dimensions of impulsivity and their relationships with internalizing pathology, namely anxiety. We hope that findings from this dissertation will provide potential targets (i.e., urgency, non-planning, hyperactivity) for continued research to hone in on distinct impulsive dimensions.

Given previous evidence of relationships between anxiety and negative urgency, we had hypothesized that emotion-based dimensions of impulsivity and inhibitory control would show stronger relationships with anxiety than non-emotional dimensions. However, Study 1 revealed a similar indirect effect of anxiety on experiential avoidance through both emotion-based impulsivity (i.e., negative and positive urgency), and the non-emotional dimension of hyperactivity. Additionally, Study 3 found that only relationships between anxiety and global impulsivity, but not negative urgency, were significant, although neither impulsive facet mediated the relationship between anxiety and MFG activation. Furthermore, Study 2 demonstrated that the ACC and dlPFC, regions previously implicated in management of emotional interference (Kohn & Fernández, 2020; Stollstorff et al., 2013), were uniquely involved in non-emotional motor inhibition. These findings suggest that failed inhibition engages

both cognitive and emotional functions associated with dlPFC and ACC, or that over the course of development, differences in activation associated with failed inhibition and emotional interference may emerge (Larsen & Luna, 2018). These findings lay the foundation for future research to explore common factors (e.g., suppression of distractors, conflict management) that may underlie emotional and non-emotional facets of inhibitory control.

The current dissertation also illustrates how relationships between anxiety and impulsivity and neural indices of inhibitory control may differ across the lifetime, underscoring the need for continued longitudinal research in this area. In our first study, we found that anxiety was positively related to multiple dimensions of impulsivity in an adult sample. Our third study corroborated the link between anxiety and global impulsivity, not negative urgency in pre-adolescents. However, impulsive facets were not related to PFC function on either task and indirect effects of anxiety, global impulsivity, and negative urgency on PFC function were not significant. Previous literature has demonstrated that onset of anxiety and externalizing disorders marked by high impulsivity (e.g., ADHD) typically occurs in early adolescence, as young as 6-8 years of age (O'Neill et al., 2017; Ströhle et al., 2018b). These symptoms have been associated with differences in PFC activation and functional connectivity between PFC and salience processing regions (e.g., amygdala, striatum; (Gold et al., 2016; Noordermeer et al., 2016; Wu et al., 2016b). Thus, findings from our second and third studies may serve as a foundation for future studies following the ABCD sample over time to investigate how baseline PFC activation may be associated with trajectories of anxiety and impulsivity symptoms and interactions between these symptoms over time. Furthermore, developmental studies often capture combinations of child, teacher, and parent reported symptoms; at young ages, child reported measures may be susceptible to low reliability due to lack of insight or psychological mindedness (Bevans et al.,

2020). Future studies may examine the age at which parent and child-report measures reliably track symptoms and when these symptoms may align with PFC development.

In summary, the findings from this dissertation provided further insight into nuanced relationships between anxiety and impulsivity and potential neural and behavioral targets for further study. In addition to setting the stage for future research into the complex interactions between these clinical presentations, this work has important implications for the future of behavioral treatments. Avoidance behavior is a key target of behavioral exposure treatments for anxiety disorders. Continued research examining the role of negative urgency and additional dimensions of impulsivity in inciting avoidance behavior may allow for greater specificity in targets of exposure treatments for anxiety. Negative urgency in particular has been shown to predict poorer outcomes for patients with depression and anxiety who underwent brief partial hospitalization (Peckham, Forgeard, Hsu, Beard, & Björgvinsson, 2019). Uncovering neural and behavioral relationships between negative urgency and anxiety-based avoidance behavior may allow for more targeted forms of exposure treatment that better account for the role of negative urgency. It is hoped that this dissertation project provides rationale for continued work in understanding these relationships, thus paving the way for greater efficacy in behavioral treatments and nuance in diagnostic approaches.

APPENDIX I

Supplemental Methods

FMRIPrep Methods

Results included in this manuscript come from preprocessing performed using *fMRIPrep* 1.5.0 (Esteban, Markiewicz, et al. (2018); Esteban, Blair, et al. (2018); RRID:SCR_016216), which is based on *Nipype* 1.2.2 (Gorgolewski et al. (2011); Gorgolewski et al. (2018); RRID:SCR_002502).

Anatomical data preprocessing

> The T1-weighted (T1w) image was corrected for intensity non-uniformity (INU) with `N4BiasFieldCorrection` (Tustison et al. 2010), distributed with ANTs 2.2.0 (Avants et al. 2008, RRID:SCR_004757), and used as T1w-reference throughout the workflow. The T1w-reference was then skull-stripped with a *Nipype* implementation of the `antsBrainExtraction.sh` workflow (from ANTs), using OASIS30ANTs as target template. Brain tissue segmentation of cerebrospinal fluid (CSF), white-matter (WM) and gray-matter (GM) was performed on the brain-extracted T1w using `fast` (FSL 5.0.9, RRID:SCR_002823, Zhang, Brady, and Smith 2001). Brain surfaces were reconstructed using `recon-all` (FreeSurfer 6.0.1, RRID:SCR_001847, Dale, Fischl, and Sereno 1999), and the brain mask estimated previously was refined with a custom variation of the method to reconcile ANTs-derived and FreeSurfer-derived segmentations of the cortical gray-matter of Mindboggle (RRID:SCR_002438, Klein et al. 2017). Volume-based spatial normalization to one standard space (MNI152NLin6Asym) was performed through nonlinear registration with `antsRegistration` (ANTs 2.2.0), using brain-extracted versions of both T1w reference and the T1w template. The following template was selected for spatial normalization: *FSL's MNI ICBM 152 non-linear 6th Generation Asymmetric Average Brain Stereotaxic Registration Model* [Evans et al. (2012), RRID:SCR_002823; TemplateFlow ID: MNI152NLin6Asym].

Functional data preprocessing

> For each of the 10 BOLD runs found per subject (across all tasks and sessions), the following preprocessing was performed. First, a reference volume and its skull-stripped version were generated using a custom methodology of *fMRIPrep*. A deformation field to correct for susceptibility distortions was estimated based on two echo-planar imaging (EPI) references with opposing phase-encoding directions, using `3dQwarp` Cox and Hyde (1997) (AFNI 20160207). Based on the estimated susceptibility distortion, an unwarped BOLD reference was calculated for a more accurate co-registration with the anatomical reference. The BOLD reference was then co-registered to the T1w reference using `bbregister` (FreeSurfer) which implements boundary-

based registration (Greve and Fischl 2009). Co-registration was configured with six degrees of freedom. Head-motion parameters with respect to the BOLD reference (transformation matrices, and six corresponding rotation and translation parameters) are estimated before any spatiotemporal filtering using `mcflirt` (FSL 5.0.9, Jenkinson et al. 2002). The BOLD time-series, were resampled to surfaces on the following spaces: *fsaverage5*. The BOLD time-series (including slice-timing correction when applied) were resampled onto their original, native space by applying a single, composite transform to correct for head-motion and susceptibility distortions. These resampled BOLD time-series will be referred to as *preprocessed BOLD in original space*, or just *preprocessed BOLD*. The BOLD time-series were resampled into standard space, generating a *preprocessed BOLD run in ['MNI152NLin6Asym'] space*. First, a reference volume and its skull-stripped version were generated using a custom methodology of *fMRIPrep*. Several confounding time-series were calculated based on the *preprocessed BOLD*: framewise displacement (FD), DVARS and three region-wise global signals. FD and DVARS are calculated for each functional run, both using their implementations in *Nipype* (following the definitions by Power et al. 2014). The three global signals are extracted within the CSF, the WM, and the whole-brain masks. Additionally, a set of physiological regressors were extracted to allow for component-based noise correction (*CompCor*, Behzadi et al. 2007). Principal components are estimated after high-pass filtering the *preprocessed BOLD* time-series (using a discrete cosine filter with 128s cut-off) for the two *CompCor* variants: temporal (tCompCor) and anatomical (aCompCor). tCompCor components are then calculated from the top 5% variable voxels within a mask covering the subcortical regions. This subcortical mask is obtained by heavily eroding the brain mask, which ensures it does not include cortical GM regions. For aCompCor, components are calculated within the intersection of the aforementioned mask and the union of CSF and WM masks calculated in T1w space, after their projection to the native space of each functional run (using the inverse BOLD-to-T1w transformation). Components are also calculated separately within the WM and CSF masks. For each CompCor decomposition, the *k* components with the largest singular values are retained, such that the retained components' time series are sufficient to explain 50 percent of variance across the nuisance mask (CSF, WM, combined, or temporal). The remaining components are dropped from consideration. The head-motion estimates calculated in the correction step were also placed within the corresponding confounds file. The confound time series derived from head motion estimates and global signals were expanded with the inclusion of temporal derivatives and quadratic terms for each (Satterthwaite et al. 2013). Frames that exceeded a threshold of 0.5 mm FD or 1.5 standardised DVARS were annotated as motion outliers. All resamplings can be performed with *a single interpolation step* by composing all the pertinent transformations (i.e. head-motion transform matrices, susceptibility distortion correction when available, and co-registrations to anatomical and output spaces). Gridded (volumetric) resamplings were performed using `antsApplyTransforms` (ANTs), configured with Lanczos interpolation to minimize the smoothing effects of other kernels (Lanczos 1964). Non-gridded (surface) resamplings were performed using `mri_vol2surf` (FreeSurfer).

Many internal operations of *fMRIPrep* use *Nilearn* 0.5.2 (Abraham et al. 2014, RRID:SCR_001362), mostly within the functional processing workflow. For more details of the pipeline, see the section corresponding to workflows in *fMRIPrep*'s documentation.

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APPENDIX II

Supplementary Table

Hemisphere	Area ID	Peak T	Vertex	DKT	DKT % Overlap	MMP	MMP % Overlap	Yeo7	Yeo7 % Overlap
Stop Signal Task- Failed Stop vs. Go Trials									
L	1	5.962	2389	Posterior Cingulate	59%	RSC	100%	7Networks_6	69%
L	3	4.898	4127	Posterior Cingulate	100%	23c	98%	7Networks_4	93%
L	4	8.027	2233	Superior Frontal	30%	p32pROI	5%	7Networks_4	52%
L	4	7.19	2767	Caudal Anterior Cingulate	6%	p32pROI	5%	7Networks_4	52%
L	4	7.226	3467	Superior Frontal	30%	6ma	6%	7Networks_4	52%
L	4	5.585	5769	Precentral	22%	FEF	4%	7Networks_3	21%
L	4	6.255	6231	Superior Frontal	30%	6a	8%	7Networks_3	21%
L	4	8.077	11054	Insula	14%	FOP4	7%	7Networks_4	52%
L	4	8.428	18473	Pars Opercularis	14%	FOP4	7%	7Networks_4	52%
L	4	6.787	19163	Precentral	22%	6ROI	10%	7Networks_4	52%
L	4	6.655	19601	Precentral	22%	IFJp	3%	7Networks_3	21%
L	4	5.118	30977	Superior Temporal	2%	PI	2%	7Networks_4	52%
L	5	3.509	4030	Paracentral	100%	5m	26%	7Networks_2	100%
L	8	7.06	7150	Supramarginal	45%	IP2	8%	7Networks_6	25%
L	8	8.421	9412	Supramarginal	45%	PSROI	7%	7Networks_4	21%

L	8	4.563	12289	Superior Parietal	31%	7PROI	5%	7Networks_3	39%
L	8	4.103	12906	Precuneus	7%	POS2	6%	7Networks_1	5%
L	8	5.751	13780	Inferior Parietal	10%	IP0	3%	7Networks_3	39%
L	8	7.828	14129	Supramarginal	45%	IP2	8%	7Networks_6	25%
L	8	6.57	14546	Superior Parietal	31%	LIPv	4%	7Networks_3	39%
L	9	5.599	8533	Superior Temporal Sulcus	11%	TPOJ1	7%	7Networks_4	9%
L	9	6.678	15442	Lateral Occipital	49%	MST	6%	7Networks_1	50%
L	9	6.956	23445	Lateral Occipital	49%	V3	7%	7Networks_1	50%
L	9	6.335	23486	Lateral Occipital	49%	LO2	5%	7Networks_1	50%
L	9	5.643	24141	Fusiform	13%	PH	9%	7Networks_3	30%
L	10	4.401	9440	Superior Temporal	100%	STSdp	100%	7Networks_7	100%
L	16	4.731	27861	Rostral Middle Frontal	100%	9-46d	41%	7Networks_6	65%
L	16	4.75	29779	Rostral Middle Frontal	100%	46	48%	7Networks_6	65%
L	17	2.527	20586	Lateral Orbitofrontal	100%	11ROI	100%	7Networks_6	100%
L	18	2.389	22114	Fusiform	100%	TGv	100%	7Networks_5	100%
L	19	2.621	22251	Inferior Temporal	100%	TF	100%	7Networks_5	100%
L	21	3.586	23785	Lateral Occipital	100%	V1	63%	7Networks_1	100%
L	23	5.732	24165	Lateral Occipital	86%	V2	59%	7Networks_1	100%
L	25	3.059	25541	Pericalcarine	47%	V1	100%	7Networks_1	100%
L	31	2.988	31103	Insula	39%	52	100%	7Networks_2	100%
R	33	6.899	9001	Supramarginal	18%	PF	6%	7Networks_4	16%

R	33	6.625	9223	Supramarginal	18%	PSROI	4%	7Networks_4	16%
R	33	4.028	11975	Superior Parietal	13%	V7	0%	7Networks_1	31%
R	33	4.897	12520	Precuneus	5%	7Am	1%	7Networks_3	26%
R	33	5.507	12704	Superior Parietal	13%	POS2	5%	7Networks_1	31%
R	33	5.001	13683	Inferior Parietal	16%	IP1	4%	7Networks_3	26%
R	33	4.907	14093	Superior Parietal	13%	IPS1	1%	7Networks_3	26%
R	33	6.565	14355	Superior Parietal	13%	AIP	3%	7Networks_3	26%
R	33	7.161	15077	Lateral Occipital	24%	V4t	2%	7Networks_1	31%
R	33	6.934	15348	Middle Temporal	6%	FST	2%	7Networks_3	26%
R	33	4.559	22471	Inferior Temporal	3%	TE2p	3%	7Networks_3	26%
R	33	7.177	23342	Lateral Occipital	24%	V4	5%	7Networks_1	31%
R	33	7.875	23585	Lateral Occipital	24%	V4	5%	7Networks_1	31%
R	33	5.442	23961	Fusiform	5%	PH	3%	7Networks_1	31%
R	33	7.786	24234	Lateral Occipital	24%	V1	2%	7Networks_1	31%
R	33	7.398	32280	Superior Temporal	3%	STSdp	3%	7Networks_7	9%
R	34	3.638	2214	Posterior Cingulate	88%	p24pROI	13%	7Networks_4	40%
R	34	5.432	2298	Posterior Cingulate	88%	RSC	36%	7Networks_6	44%
R	34	4.315	4164	Posterior Cingulate	88%	23c	29%	7Networks_4	40%
R	35	3.784	1511	Lingual	96%	V1	92%	7Networks_1	100%
R	36	8.005	2282	Superior Frontal	77%	p32pROI	11%	7Networks_4	51%
R	36	7.296	2903	Caudal Anterior Cingulate	15%	a32pROI	11%	7Networks_6	26%
R	36	6.119	4942	Superior Frontal	77%	SCEF	16%	7Networks_4	51%

R	36	5.592	6268	Caudal Middle Frontal	6%	6a	12%	7Networks_3	14%
R	36	6.3	6529	Superior Frontal	77%	6ma	17%	7Networks_4	51%
R	37	4.41	30912	Precentral	53%	55b	47%	7Networks_4	50%
R	39	5.278	10290	Insula	80%	PoI1	63%	7Networks_4	100%
R	40	7.608	10645	Insula	25%	MI	9%	7Networks_4	61%
R	40	8.679	10981	Insula	25%	FOP4	11%	7Networks_4	61%
R	40	8.719	18574	Pars Opercularis	34%	6ROI	22%	7Networks_4	61%
R	40	4.971	19492	Precentral	19%	6ROI	22%	7Networks_3	16%
R	42	2.786	15042	Middle Temporal	50%	TE1p	100%	7Networks_6	100%
R	45	5.679	17541	Supramarginal	60%	PFop	100%	7Networks_4	73%
R	47	3.341	18346	Rostral Middle Frontal	100%	IFSa	50%	7Networks_6	100%
R	48	3.813	19311	Rostral Middle Frontal	100%	8C	41%	7Networks_6	100%
R	49	2.278	21000	Lateral Orbitofrontal	100%	10pp	100%	7Networks_5	100%
R	50	3.881	21888	Inferior Temporal	83%	TE2a	92%	7Networks_5	94%
R	54	3.323	22165	Inferior Temporal	100%	TGv	100%	7Networks_5	100%
R	58	3.72	28746	Rostral Middle Frontal	100%	9-46d	92%	7Networks_4	66%
R	60	3.687	29923	Rostral Middle Frontal	100%	46	63%	7Networks_6	100%
R	60	3.596	30218	Rostral Middle Frontal	100%	p9-46v	26%	7Networks_6	100%
R	62	3.287	31726	Superior Temporal	100%	STSda	100%	7Networks_7	100%

L	64	-4.707	12450	Superior Parietal	54%	V3A	41%	7Networks_1	100%
L	64	-4.465	24837	Lateral Occipital	39%	V2	20%	7Networks_1	100%
L	65	-7.938	26124	Precuneus	75%	POS1	52%	7Networks_7	66%
L	66	-3.805	112	Lingual	100%	V2	22%	7Networks_1	100%
L	67	-6.951	21503	Parahippocampal	43%	PHA2	14%	7Networks_7	20%
L	67	-6.172	23114	Fusiform	36%	PHA3	32%	7Networks_1	68%
L	69	-4.667	3802	Posterior Cingulate	37%	31a	57%	7Networks_7	97%
L	71	-3.734	4450	Paracentral	100%	4	100%	7Networks_2	100%
L	74	-2.717	5505	Postcentral	100%	3b	85%	7Networks_2	100%
L	75	-3.43	6685	Superior Parietal	100%	2	100%	7Networks_2	100%
L	77	-3.117	7996	Postcentral	100%	1	100%	7Networks_2	100%
L	78	-3.798	8226	Precentral	100%	4	74%	7Networks_2	100%
L	79	-3.236	8382	Middle Temporal	100%	TE1p	100%	7Networks_7	89%
L	80	-3.063	9702	Superior Temporal	100%	A4	100%	7Networks_2	100%
L	81	-3.205	9909	Superior Temporal	100%	LBelt	60%	7Networks_2	100%

L	82	-4.134	18279	Pars Triangularis	100%	IFSa	25%	7Networks_7	100%
L	84	-4.876	11674	Pars Triangularis	30%	p47ROI	42%	7Networks_6	43%
L	85	-6.601	20026	Lateral Orbitofrontal	79%	47m	31%	7Networks_7	57%
L	86	-3.147	10266	Insula	100%	OP2-3	60%	7Networks_2	100%
L	88	-2.879	12372	Superior Parietal	100%	V6A	100%	7Networks_1	100%
L	92	-5.164	15894	Inferior Parietal	100%	PGp	20%	7Networks_3	20%
L	95	-4.1	18635	Pars Opercularis	97%	IFSp	37%	7Networks_7	85%
L	96	-6.555	27909	Medial Orbitofrontal	79%	10ROI	29%	7Networks_7	85%
L	98	-2.768	20339	Lateral Orbitofrontal	100%	47s	100%	7Networks_7	51%
L	100	-3.895	21068	Medial Orbitofrontal	72%	OFC	100%	7Networks_5	100%
L	102	-2.846	21238	Medial Orbitofrontal	100%	OFC	100%	7Networks_5	100%
L	103	-2.64	32471	Middle Temporal	100%	TE1p	100%	7Networks_7	100%
L	106	-4.669	24762	Lingual	86%	V1	100%	7Networks_1	100%
L	107	-7.568	24537	Lingual	97%	V3	35%	7Networks_1	100%
L	108	-4.16	24030	Lateral Occipital	95%	V1	100%	7Networks_1	100%
L	113	-4.819	25731	Cuneus	73%	V1	47%	7Networks_1	100%

L	115	-4.049	26755	???	100%	H	90%	FreeSurfer_Defined_Medial_Wall	82%
L	116	-3.726	29165	Superior Frontal	74%	9p	13%	7Networks_7	98%
L	116	-4.767	29860	Superior Frontal	74%	8Ad	57%	7Networks_7	98%
L	116	-4.155	30272	Caudal Middle Frontal	26%	8Ad	57%	7Networks_7	98%
L	118	-4.167	28346	Superior Frontal	92%	10d	80%	7Networks_7	100%
L	119	-3.507	28828	Superior Frontal	100%	9a	40%	7Networks_7	100%
L	124	-3.037	29330	Superior Frontal	100%	8BM	100%	7Networks_7	69%
L	128	-3.368	30755	Caudal Middle Frontal	100%	8Av	100%	7Networks_7	100%
L	129	-2.774	31274	Superior Temporal	100%	TGd	100%	7Networks_5	100%
L	131	-5.919	32010	Superior Temporal	63%	STSva	54%	7Networks_7	87%
R	134	-5.901	24839	Lateral Occipital	37%	V3	41%	7Networks_1	100%
R	134	-5.207	25280	Cuneus	34%	V3	41%	7Networks_1	100%
R	135	-7.973	23127	Lingual	30%	VMV2	13%	7Networks_1	79%
R	135	-6.502	26731	Parahippocampal	43%	PHA1	22%	7Networks_1	79%
R	137	-8.212	26159	Precuneus	74%	POS1	65%	7Networks_7	76%

R	138	-3.162	12692	Precuneus	100%	5mv	68%	7Networks_3	68%
R	139	-3.219	3801	Posterior Cingulate	100%	31a	77%	7Networks_7	74%
R	140	-3.325	4060	Paracentral	100%	5m	58%	7Networks_2	100%
R	141	-3.355	4384	Paracentral	100%	4	100%	7Networks_2	100%
R	142	-2.852	4538	Paracentral	100%	24dd	100%	7Networks_2	100%
R	144	-3.715	5319	Precentral	95%	3a	54%	7Networks_2	100%
R	146	-3.898	5360	Postcentral	90%	3b	86%	7Networks_2	100%
R	147	-3.748	5344	Precentral	100%	FEF	92%	7Networks_4	37%
R	149	-3.683	5755	Precentral	100%	6d	100%	7Networks_2	100%
R	151	-3.423	5872	Precentral	100%	6mp	27%	7Networks_2	100%
R	152	-3.281	5900	Precentral	100%	4	100%	7Networks_2	100%
R	153	-4.405	7020	Superior Parietal	93%	2	52%	7Networks_2	84%
R	153	-4.205	14851	Superior Parietal	93%	7PC	45%	7Networks_3	16%
R	154	-3.628	17658	Postcentral	53%	2	57%	7Networks_2	55%
R	156	-3.557	7513	Postcentral	100%	1	40%	7Networks_2	100%

R	158	-4.905	7968	Postcentral	100%	1	86%	7Networks_2	100%
R	160	-4.758	8163	Precentral	45%	3a	40%	7Networks_2	100%
R	164	-4.014	9802	Superior Temporal	100%	A4	30%	7Networks_2	100%
R	165	-5.905	20130	Lateral Orbitofrontal	93%	47m	40%	7Networks_7	59%
R	166	-3.003	13303	Precuneus	100%	31pd	75%	7Networks_7	100%
R	167	-3.598	15930	Inferior Parietal	100%	PGs	59%	7Networks_7	70%
R	168	-3.052	16896	Supramarginal	100%	PFcm	100%	7Networks_2	100%
R	169	-4.079	21380	Medial Orbitofrontal	81%	s32	14%	7Networks_5	17%
R	169	-6.126	27964	Medial Orbitofrontal	81%	10ROI	28%	7Networks_7	83%
R	170	-3.62	20214	Rostral Middle Frontal	65%	a47ROI	100%	7Networks_6	100%
R	173	-4.179	22522	Fusiform	100%	TF	100%	7Networks_5	100%
R	174	-4.366	24032	Lateral Occipital	19%	V1	100%	7Networks_1	100%
R	176	-3.906	24818	Pericalcarine	39%	V1	100%	7Networks_1	100%
R	177	-6.635	24807	Lingual	100%	V2	71%	7Networks_1	100%
R	180	-3.943	25129	Pericalcarine	90%	V1	100%	7Networks_1	100%

R	182	-4.308	25247	Lingual	100%	V1	86%	7Networks_1	100%
R	184	-5.228	25731	Pericalcarine	37%	V1	42%	7Networks_1	100%
R	185	-5.072	26615	???	100%	H	100%	7Networks_5	47%
R	187	-2.859	28106	Rostral Middle Frontal	100%	10pp	100%	7Networks_5	100%
R	188	-2.572	28761	Superior Frontal	100%	9m	100%	7Networks_7	100%
R	189	-3.218	29204	Superior Frontal	100%	8BROI	100%	7Networks_7	100%
R	194	-3.611	30073	Rostral Middle Frontal	22%	8Ad	93%	7Networks_7	100%
R	197	-3.36	31370	Superior Temporal	100%	TGd	100%	7Networks_5	88%
R	199	-5.25	31779	Superior Temporal	100%	A4	60%	7Networks_2	100%
R	200	-4.732	32197	Middle Temporal	93%	TE1a	72%	7Networks_7	100%
EN-Back Task- Negative vs. Neutral Face Trials									
L	1	4.931	22610	Fusiform	20%	TE2p	100%	7Networks_3	100%
R	6	5.855	9080	Superior Temporal Sulcus	95%	STSdp	33%	7Networks_7	54%
R	7	3.738	11029	Insula	100%	AVI	100%	7Networks_4	100%
R	9	4.266	18816	Pars Opercularis	100%	44	100%	7Networks_6	100%
R	10	4.236	19070	Rostral Middle Frontal	100%	IFSp	100%	7Networks_6	100%
R	12	4.569	22496	Inferior Temporal	100%	TF	100%	7Networks_5	100%
R	13	3.857	28796	Superior Frontal	100%	9a	100%	7Networks_7	100%

R	14	5.163	31950	Superior Temporal	100%	STSda	62%	7Networks_7	80%
R	17	5.359	32288	Middle Temporal	100%	STSva	73%	7Networks_7	100%
Conjunction									
R	6	4	11029	Insula	100%	AVI	100%	7Networks_4	100%
R	7	3.046	19174	Pars Opercularis	100%	IFJa	100%	7Networks_6	100%

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