# NEURAL CORRELATES UNDERLYING THE INTERACTIONS BETWEEN ANXIETY AND CANNABIS USE IN PREDICTING MOTOR RESPONSE INHIBITION

by

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ABSTRACT

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The ability to effectively withhold an inappropriate response is a critical feature of cognitive control. Prior research indicates alterations in neural processes required for motor response inhibition in anxious individuals, including those with posttraumatic stress disorder (PTSD), and those who engage in regular cannabis use. However, thus far most research has examined how anxiety-related symptoms and cannabis use influence response inhibition in isolation of one another. The current study examined the interactions between anxious symptomology and recent cannabis use in a sample that recently experienced a traumatic event using functional magnetic resonance imaging (fMRI) during the completion of a Stop-Signal task. We identified an underlying component reflective of anxious symptomology and PTSD, and examined how this factor interacted with recent cannabis use to predict behavioral performance and neural activity during completion of this task. We found no evidence for impaired behavioral performance, or alterations in underlying brain regions between those who did and did not recently engage in cannabis use, across levels of anxiety and PTSD, or interactions between these variables. These results are discussed in relation to the current literature surrounding the relationship between motor response inhibition, anxiety, cannabis use, and PTSD.

To

my parents,

my brother,

and my partner

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**AUDIT –** Alcohol Use Disorders Identification Test

**dACC –** Dorsal Anterior Cingulate Cortex

**DASS-21 –** Depression, Anxiety, and Stress Scale

**Δ-9-THC –** Delta-9-Tetrahydrocannabinol

**dlPFC –** Dorsolateral Prefrontal Cortex

**EEG –** Electroencephalography

**ERP –** Event-Related Potential

**fMRI –** Functional Magnetic Resonance Imaging

**PCA –** Principal Component Analysis

**PCL-5 –** PTSD Checklist for the DSM-5

**PTSD –** Posttraumatic Stress Disorder

**rIFG –** Right Inferior Frontal Gyrus

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**Neural Correlates Underlying the Interactions Between Anxiety and Cannabis Use in Predicting Response Inhibition**

Individuals engage in a variety of tasks that require the continuous utilization of cognitive processes (e.g., counting numbers in a list) and behavioral actions (e.g., walking) throughout their daily lives. However, we are often presented with both internal and external changes in our environment that require us to withhold such mental processes (e.g., ignore some numbers presented) and behavioral engagement (e.g., stop walking). This ability to effectively inhibit task-irrelevant information and withhold behavioral responses is considered a hallmark feature of cognitive control (Diamond, 2013; Gratton et al., 2018; Lehto et al., 2003; Lenartowicz et al., 2010; Miyake et al., 2000; Sabb et al., 2008; Xu et al., 2017), and allows individuals to flexibly adapt in order to meet current environmental demands to complete goal-directed tasks (Burgess & Simons, 2005; Mesulam, 1986; Miller & Cohen, 2001; Shallice & Burgess, 1996). As such, inhibitory control allows individuals to control their attention, thoughts, and behaviors to overcome specific prepotent processes when they are disadvantageous based on current task demands (Lehto et al., 2003).

Inhibitory control can be further separated into motor and attentional domains, with the motor construct reflecting the ability to suppress a prepotent motor response and the cognitive aspect involving the ability to ignore distracting stimuli (Diamond, 2013; Friedman & Miyake, 2004; Gandolfi et al., 2014; Kane et al., 2016; Nigg, 2000, 2017; Stahl et al., 2014; Tiego et al., 2018). Although these domains of inhibitory control are strongly related (Friedman & Miyake, 2004), previous work has demonstrated their independence from one another as separate constructs (Bender et al., 2016; Gandolfi et al., 2014; Kane et al., 2016; Stahl et al., 2014; Tiego et al., 2018). In support of this view, others have found that these two facets of inhibitory control

rely on separate brain regions, although they also share overlap by relying on prefrontal regions (Bunge et al., 2002; Diamond, 2013; Hung et al., 2018; Munakata et al., 2011). Specifically, a recent meta-analysis indicated that attentional inhibition is associated with the recruitment of regions composing of the dorsal frontal inhibitory system (e.g., dorsal anterior cingulate cortex, dorsolateral prefrontal cortex, parietal regions, etc.), while motor inhibition was related to activity in fronto-striatal regions, such as the supplementary motor area, basal ganglia, and both dorsal and ventral lateral prefrontal cortex (Hung et al., 2018). This suggests that despite some overlap in neural networks necessary to engage in both forms of inhibitory control, the motor and attentional factors also rely on distinct structures.

**Motor Response Inhibition**

The motor component of inhibitory control is often referred to as motor response inhibition, and pertains to the ability to effectively inhibit a prepotent motor response (Chevrier et al., 2007; Congdon et al., 2012; Diamond, 2013; Friedman & Miyake, 2004; Gandolfi et al., 2014; Li et al., 2009; Neo et al., 2011; Nigg, 2000; Nigg, 2017; Sharp et al., 2010; Stahl et al., 2014; Tiego et al., 2018; Verbruggen & Logan, 2010), allowing one to effectively overcome habitual responses when they are no longer relevant or even detrimental to a current task. This ability is often measured through tasks that require individuals to withhold a motor response, such as the Go/No-Go and Stop-Signal task (Bender et al., 2016; Diamond, 2013; Gratton et al., 2018; Raud et al., 2020). In a Go/No-Go task, participants are shown a stimulus that requires a response (e.g., Go trials) or to withhold a response (e.g., NoGo trials), with the frequency of Go trials far exceeding that of NoGo trials in typical designs (Cragg & Nation, 2008; Diamond et al., 2013; Gratton et al., 2018). In contrast, the Stop-Signal task uses a stimulus cue as a “Go” cue on all trials, but also includes an additional “Stop” cue appearing with this Go cue on a subset of

trials, indicating to participants that they must inhibit their response following these "Stop" cues (Diamond et al., 2013; Gratton et al., 2018; Logan et al., 1984; Neo et al., 2011; Verbruggen & Logan, 2008). Thus, although both tasks require participants to engage in motor response inhibition, they differ slightly due to the inclusion of "Go" cues on all trials and the addition of an interval delay between the "Go" and "Stop" cues present in the Stop-Signal task. These tasks, particularly the Stop-Signal task, have shown strong ecological validity based on their association with motor impulsivity traits and observations (Congdon et al., 2012; Lijffijt et al., 2004, 2005; Logan et al., 1997; Schachar et al., 1993; Solanto et al., 2001), and are highly reliabile over the developmental lifespan (Logan et al., 1997; Williams et al., 1999), lending support for their use as an assessment of motor response inhibition.

Despite these slight variations in design between the Go/No-Go and Stop-Signal task, functional magnetic resonance imaging (fMRI) comparing these two tasks often demonstrate converging brain network activity (Cai et al., 2014; Dambacher et al., 2014; Nee et al., 2007; Rubia et al., 2001; Sebastian et al., 2013; Swick et al., 2011; Zheng et al., 2008), which is believed to contribute to motor response inhibition (although see Raud et al., 2020 who found differences in topographical scalp activity between the two tasks using electroencephalography). Current neuroimaging evidence suggests that motor response inhibition recruits various frontal brain regions (Aron & Poldrack, 2005; Boehler et al., 2010; Chevrier et al., 2004; Floden & Stuss, 2006; Kelly et al., 2004; Li et al., 2008; Neo et al., 2011; Rubia et al., 2003; Wager et al., 2005), including the right inferior frontal gyrus (rIFG, Aron et al., 2003, 2004, 2007; Aron & Poldrack, 2006; Chambers et al., 2006, 2007; Chevrier et al., 2007; Erika-Florence et al., 2014; Garavan et al., 1999; Li et al., 2006a; Matthews et al., 2005; Rubia et al., 2001, 2003, 2007; Xu et al., 2017) and the pre-supplementary motor area (Aron et al., 2007; Aron & Poldrack, 2006;

Coxon et al., 2009; Duann et al., 2008; Garavan et al., 2002; Li et al., 2006a; Mostofsky & Simmonds, 2008; Sharp et al., 2010). Additional work indicates the involvement of structures within the striatum (Aron et al., 2003; Boehler et al., 2010; Chevrier et al., 2007; Vink et al., 2005), such as the subthalamic nucleus of the basal ganglia (Aron & Poldrack, 2005; Chevrier et al., 2007; Rieger et al., 2003; Rubia et al., 2007). Taken together, these structures form the fronto-basal-ganglia inhibition network that allows for successful motor response inhibitory behaviors (Aron et al., 2007; Chambers et al., 2009; Jahanshahi et al., 2015; Sandrini et al., 2020; Verbruggen & Logan, 2008; Zhang & Iwaki, 2020).

The fronto-basal-ganglia inhibition network facilitates response inhibition through projections from prefrontal regions to the basal ganglia (Aron et al., 2007; Aron & Poldrack, 2006; Chambers et al., 2009; Mostofsky & Simmonds, 2008; Nambu et al., 2002; Verbruggen & Logan, 2008; Voytek, 2006; Zhang & Iwaki, 2020). Specifically, the inferior frontal gyrus and pre-supplementary motor area send excitatory signals to the subthalamic nucleus of the basal ganglia. This in turn activates the globus pallidus and substantia nigra, resulting in the inhibition of the thalamus. Consequently, the inhibition of the thalamus leads to suppression of downstream structures necessary for motor output, such as the primary motor cortex, premotor cortex, and supplementary motor area (Aron & Verbruggen, 2008; Coxon et al., 2006). However, the specific contributions of these prefrontal structures in this circuit have been debated, with some arguing that the inferior frontal gyrus is more involved with attentional response inhibition when viewing stimuli, while the pre-supplementary motor area is primarily involved with the suppression of motor output through direct and indirect communication with the basal ganglia, allowing for successful motor response inhibition (Duan et al., 2008; Criaud & Boulinguez, 2013; Erika-Florence et al., 2014; Hampshire et al., 2010; Munakata et al., 2011; Sharp et al.,

2010; Walther et al., 2011; Xu et al., 2017). Regardless of these differing views, it is clear that structures within this fronto-basal-ganglia network are critical in contributing to the ability to successfully inhibit a motor response.

**Common Associates of Response Inhibition**

Successfully inhibiting a response is necessary to promote healthy functioning, and provides individuals with the opportunity to choose how to react instead of simply succumbing to habitual responses. As such, successful response inhibition has been associated with various processes supporting adaptive functioning, including greater working memory capacity (Tiego et al., 2018), verbal intelligence (Lee et al., 2015), socioemotional functioning (Sahdra et al., 2011), and emotional regulation (King, 2020). Therefore, individuals displaying a greater degree of response inhibition also demonstrate characteristics that promote healthy day-to-day functioning.

In contrast, failure to appropriately engage in response inhibition has been associated with developmental disorders (Geurts et al., 2004; Schmitt et al., 2018; Uzefovsky et al., 2016), impulsivity (Jasinska et al., 2012; Leshem, 2016; Sellaro & Colzato, 2017), neurological disorders (Enticott et al., 2008; Hughes et al., 2012; Manza et al., 2017; Matzke et al., 2017; Verbruggen & Logan, 2008), alcohol and drug seeking behaviors (Czermainski et al., 2017; Fillmore & Rush, 2002; Li & Sinha, 2008; Li et al., 2006b; López-Caneda et al., 2012; Molnar et al., 2018; Monterosso et al., 2005; Poulton et al., 2016), and various forms of psychopathology (Alderson et al., 2007; Booth et al., 2005; Crosbie & Schachar, 2001; Chamberlain et al., 2006; Geurts et al., 2006; Lijffijt et al., 2005; Kertz et al., 2016; Paulus, 2015; Roth et al., 2007; Rubia et al., 1999; Zeier Baskin-Sommers et al., 2012). In accordance with these findings, many consider deficits in response inhibition as a risk-factor for the development and maintenance of various psychopathological (Carver et al., 2017; Friedman et al., 2020; Kagan, 2008) and

substance use disorders (Everitt, 2014; Everitt & Robbins, 2005; Smith et al., 2014; Tervo-Clemmens et al., 2017), such that individuals dcharacterized by poor response inhibition are likely to develop these disorders.

Given the abundant societal costs associated with psychopathological (Kessler et al., 2012; Trautmann et al., 2016; Whiteford et al., 2013; Wittchen et al., 2011) and substance use (Trautmann et al., 2016; Whiteford et al., 2012; Wittchen et al., 2011) disorders, it is crucial to further our understanding of response inhibition and the underlying neural mechanisms among individuals suffering from these disorders. Such investigation is warranted in more commonly observed forms of psychopathology and substance use in the general population, such as anxiety (Bandelow & Michaelis, 2015; Baxter et al., 2013; Remes et al., 2016) and cannabis use (Hasin et al., 2016; Rotermann & Macdonald, 2018; Trivers et al., 2018), respectively. This line of work will aid in providing knowledge of the neurobiological markers of response inhibition in anxious and cannabis using individuals, and may provide potential clinical treatment targets for intervention.

**Anxiety and Response Inhibition**

Anxiety disorders are some of the most prevalent mental health disorders (Bandelow & Michaelis, 2015; Kessler et al., 2012) with a growing body of literature indicating that many of the societal costs associated with anxiety (Collins et al., 2011; Johnston et al., 2009; Lépine, 2002) are due to alterations in cognitive functioning (Beck & Clark, 1997; Hamm, 2020; Heeren et al., 2013; Mathews & MacLeod, 2005; Ouimet et al., 2009; Robinson et al., 2013b; Vytal et al., 2012). Specifically, current theoretical models propose broad deficits in inhibitory cognitive control in clinical and sub-clinical anxious populations (Ansari & Derakshan, 2011; Basten et al., 2011; Cavanagh & Shackman, 2015; Derakshan & Eysenck, 2009; Eysenck & Derakshan, 2011;

Eysenck et al., 2007; Liao et al., 2019; Paulus, 2015; White et al., 2011; Wood et al., 2001). However, many of these models are based work that focused exclusively on attentional inhibition of task-irrelevant stimuli (Bishop, 2009; Chen et al., 2015; Liao et al., 2019; Pacheco-Unguetti et al., 2010; Ward et al., 2018; Yu et al., 2018). Although attentional and motor response inhibition are strongly related sub-constructs of inhibitory cognitive control (Friedman & Miyake 2004), recent work has demonstrated their independence (Stahl et al., 2014; Tiego et al., 2018), supporting the notion that these factors should be examined separately.

Given the link between attentional and motor response inhibition (Friedman & Miyake 2004), and current theories suggesting that anxious individuals experience an overall deficit in attentional processes (Derakshan & Eysenck, 2009; Eysenck & Derakshan, 2011; Eysenck et al., 2007), researchers have proposed that anxiety is related to response inhibition deficits. Evidence supporting these accounts can be found in behavioral work demonstrating that anxious individuals commit more errors on Stroop tasks (Hallion et al., 2017; Pallak et al., 1975; Wieser et al., 2009) and display greater antisaccade latencies on antisaccade tasks (i.e., increased time to fixate away from a visual stimulus, Ansari & Derakshan, 2011; Ansari et al., 2008; Derakshan et al., 2009), with both tasks being designed to create a tendency to respond in a given way. However, others have found no differences in accuracy accompanied by longer response times in anxious individuals on response inhibition tasks, such as the antisaccade (Ansari & Derakshan, 2011; Ansari et al., 2008), leading to the proposal that anxiety is more likely to influence the overall processing efficiency, and has little impact on specific performance outcomes on antisaccade and Stroop tasks (Eysenck et al., 2007). Despite these findings, additional contradictions are reported in the field, with some showing enhanced performance in anxious individuals (Hardin et al., 2009; Schmid et al., 2015; Yang et al., 2018), and others identifying

no differences in behavioral outcomes for anxious individuals (Ng et al., 2012; Osinsky et al., 2012; Price & Mohlman, 2007) in response inhibition. Therefore, the current results in this area provide an inconsistent picture on the effects of anxiety on motor response inhibition, at least when measured using the Stroop and antisaccade tasks.

One potential confound of the previously reported studies may be the inclusion of addition task demands outside of simply inhibiting a motor response. While the Stroop and antisaccade tasks are commonly categorized with the Stop-Signal and Go/No-Go tasks (Friedman & Miyake, 2004, 2017; Friedman et al., 2008; Miyake & Friedman, 2012), these tasks include additional elements that are omitted in the Stop-Signal and Go/No-Go tasks. For example, while the antisaccade task involves the inhibition of planned motor responses, it also requires the generation of eye movements to create an antisaccade (Hunt et al., 2004; Massen, 2004). The Stroop also requires a separate response to be made in addition to inhibition. In contrast, the Stop-Signal and Go/No-Go tasks simply require the inhibition of a motor response, without additional task requirements. Therefore, while the Stroop and antisaccade tasks involve the inhibition of a response, they also require the formation of a response that conflicts with a learned habitual response (i.e., Stroop task) or generation of a saccade in the direction opposite of a given cue (i.e., antisaccade task). Therefore, it is possible that these tasks may tap into a different form of motor response inhibition than other tasks, such as the Stop-Signal and Go/No-Go tasks, which more strongly isolate motor response inhibition in their design (Bartholow et al., 2018; Paap et al., 2020; Stahl et al., 2014; Verbruggen & Logan, 2008). In complement to these differences in motor response inhibition tasks, Ansari and Derakshan (2010) found that anxiety had no impact on the generation of antisaccades, suggesting that increased antisaccade latencies observed in high anxious individuals on this task reflects response inhibition deficits.

Furthermore, Hallion and colleagues (2017) identified differences in performance on the Stroop and Go/No-Go tasks between anxious individuals, with clinically anxious participants showing slower responses on incongruent trials in the Stroop task, but showing no behavioral differences in terms of accuracy or response time in the Go/NoGo compared to healthy controls. Therefore, the discrepancies observed in motor response inhibition tasks measured using the Stroop and antisaccade tasks may be due to these additional task elements, making them a less specific index of strictly motor response inhibition. Given that tasks such as the Stop-Signal and Go/No-Go simply require the inhibition of a motor response, it is likely they serve as a more valid measure of motor response inhibition (Bartholow et al., 2018; Hallion et al., 2017; Nee et al., 2007; Paap et al., 2020; Stahl et al., 2014; Verbruggen & Logan, 2008), despite their common categorization in the same factor as the Stroop and antisaccade tasks (Friedman & Miyake, 2004).

The quantity of studies examining the influence of anxiety on motor response inhibition using Stop-Signal and Go/No-Go tasks are limited. Of the few studies that have examined this question, some researchers have found enhanced response inhibition accuracy in individuals placed in an anxious state (Choi & Cho, 2020; Grillon et al., 2017a, 2017b; Robinson et al., 2013a), and those with enhanced test anxiety (Hagopian & Ollendick, 1994) and trait anxiety (Sehlmeyer et al., 2010). This line of work has provided support for Gray's proposal that anxiety yields enhanced activation of the behavioral inhibition system, thus facilitating response inhibition (Gray & McNaughton, 2000; Epstein et al., 2001; Quay, 1997; Sylwan, 2004). In contrast to this theory, others have reported reduced response inhibition accuracy in state anxious individuals (Pacheco-Unguetti et al., 2012; Roxburgh et al., 2020). Further conflicting evidence can be seen by work failing to find motor response inhibition behavioral differences between healthy controls and those with diagnosed anxiety disorders (Grillon et al., 2017b, Hallion et al.,

2017; Herrmann et al., 2003; Kim et al., 2007; Leonard & Abramovitch, 2019), and those high in trait anxiety (Oosterlaan & Sergeant, 1996) and high trait anxiety with alcohol dependence (Karch et al., 2008). These inconsist findings concerning the effects of anxiety on motor response inhibition may reflect the use of a specific task (e.g., Go/No-Go versus Stop-Signal) and the type of anxiety measured (e.g., clinical, sub-clinical, trait, state, etc.). Thus, the impact of anxiety on motor response inhibition on tasks such as the Stop-Signal and Go/No-Go tasks remains unclear, with some proposing that anxiety leads to a broad deficit in inhibitory cognitive control that encompasses motor response inhibition (Eysenck et al., 2007), and others suggesting that anxiety only impairs attentional inhibitory control while enhancing motor response inhibition (Gray & McNaughton, 2000; Grillon et al., 2017a).

The contradictory behavioral findings for the effects of anxiety on motor response inhibition tasks, particularly for studies reporting null behavioral differences (Grillon et al., 2017b, Hallion et al., 2017; Herrmann et al., 2003; Karch et al., 2008; Kim et al., 2007; Leonard & Abramovitch, 2019; Oosterlaan & Sergeant, 199), may be accounted for by neuroimaging and electrophysiological work. For example, prominent theories of anxiety propose that anxious populations engage in compensatory mechanisms to perform a cognitive task to the same degree as their less anxious peers, but at the cost of cognitive processing resources, which can often be identified through neuroimaging methodology (Berggren & Derakshan, 2013; Derakshan & Eysenck, 2009; Eysenck & Derakshan, 2011; Eysenck et al., 2007). Support for this idea can be found in reports demonstrating a lack of behavioral differences between low and high anxious individuals, but alterations in electroencephalography (EEG) markers (Aarts & Pourtois, 2010; Righi et al., 2009; Ruchsow et al., 2007; Savostyanov et al., 2009; Sehlmeyer et al., 2010; although see Herrmann et al., 2003; Kim et al., 2007). For instance, previous work showed that

the anterior N2, an event-related potential (ERP) related to cognitive control, is enhanced in high trait anxious compared to low trait anxious individuals (Righi et al., 2009; Sehlmeyer et al., 2010). In complement to this work, Savostyanov and colleagues (2009) found that anxious individuals exhibit greater desynchronization of alpha, which is believed to reflect greater attentional activation (Klimesch, 2012), on Stop trials compared to their non-anxious peers. They interpreted this finding as reflecting greater arousal and attentional recruitment to engage in successful response inhibition following Stop cues. Taken together, these studies provide evidence for altered neurophysiological activity related to response inhibition in anxious individuals, despite a lack of behavioral effects in terms of response inhibition accuracy.

Additional work using functional magnetic resonance imaging (fMRI) has supported these prior EEG and ERP findings of functional alterations in frontal brain regions in anxious individuals during completion of motor response inhibition tasks. For instance, Forster and colleagues (2015) found reduced dorsolateral prefrontal cortex (dlPFC) and dorsal anterior cingulate cortex (dACC) activity in high trait anxious individuals compared to their less anxious peers during motor response inhibition. In contrast, Torrisi and colleagues (2016) found no response inhibition differences in activation of frontal networks in individuals placed in an anxious state, but observed enhanced putamen activity in these participants. Others examining response inhibition in clinical anxiety disorders, such as posttraumatic stress disorder (PTSD), have found reduced middle frontal cortex activity, encompassing the dlPFC (Carrion et al., 2008), and reduced activity in the rIFG (Falconer et al., 2008) during No-Go trials on a Go/No-Go task. Furthermore, although Carrion and colleagues (2008) did not observe behavioral differences between individuals with PTSD and healthy controls, Falconer and colleagues (2008) found greater commission errors in those with PTSD. Work examining response inhibition with a

Stop-Signal task also found reduced activity in PTSD patients in the ventromedial prefrontal cortex (Jovanovic et al., 2013). Thus, results concerning the specific frontal regions and their activity patterns in anxious versus healthy controls is mixed, with some, but not all, showing reduced recruitment of frontal brain regions, along with enhanced striatal activity necessary to inhibit a response.

The relationship between anxiety and motor response inhibition shows a great deal of variability, even when examining tasks believed to directly measure motor response inhibition, such as the Stop-Signal and Go/No-Go tasks. This is likely due to several factors, including the heterogeneity of anxious symptomology, the specific form of anxiety in question (i.e., clinical anxiety, trait anxiety, state anxiety, etc.), the type of task used to assess response inhibition (i.e., Go/NoGo versus Stop-Signal task), and the nature of the stimuli (i.e., neutral versus affective) used in these tasks. This is reflected by the plethora of differing behavioral (Choi & Cho, 2020; Grillon et al., 2017a, 2017b; Hagopian & Ollendick, 1994; Hallion et al., 2017; Karch et al., 2008; Kim et al., 2007; Leonard & Abramovitch, 2019; Oosterlaan & Sergeant, 1996; Pacheco-Unguetti et al., 2012; Robinson et al., 2013a; Roxburgh et al., 2020; Sehlmeyer et al., 2010) and neuroimaging (Carrion et al., 2008; Falconer et al., 2008; Forster et al., 2015; Jovanovic et al., 2013; Torrisi et al., 2016) results reported. Many of these discrepancies may also be due to other variables that influence response inhibition, such as cannabis use (Behan et al., 2014; Bhattacharyya et al., 2015; Bolla et al., 2002; McDonald et al., 2003; Ramaekers et al., 2006), but were not measured or accounted for in the reviewed studies. Thus, additional research examining the effects of anxiety on response inhibition that accounts for other influencing factors is needed to clarify the mechanisms underlying this phenomenon.

**Cannabis Use and Response Inhibition**

Cannabis is one of the most commonly used drugs (Arterberry et al., 2019; ElSohly et al., 2016; Hasin et al., 2015; Johnston et al., 2011; Johnston et al., 2019; SAMHSA, 2017; UNODC, 2011), with legal restrictions of cannabis becoming less strict and growing positive public opinion of cannabis use (Carliner, Brown, Sarvet, & Hasin, 2017; Johnston et al., 2011) likely to lead to greater prevalence of cannabis use in the general population. Many individuals (~8.9%) who engage in chronic cannabis use go on to develop Cannabis Use Disorder (Budney et al., 2015; Hall & Degenhardt, 2014; Lopez-Quintero et al., 2011), which has been associated with poorer educational outcomes, increased risk of motor vehicle crashes, psychotic symptomology, and cardiovascular and respiratory diseases (Hall, 2009). Others have also proposed that cannabis use contributes to a variety of cognitive deficits (Bhattacharyya et al., 2012; Martin-Santos et al., 2010; Volkow et al., 2014; Volkow et al., 2016; Wrege et al., 2014), including problems related to impulsivity, or problems with self-control (Day et al., 2013; Metrik et al., 2012; Moreno et al., 2012; Ramaekers et al., 2006; Simons & Carey, 2002; van Leeuwen et al., 2011). Given the link between impulsivity and response inhibition problems (Keilp et al., 2005; Marsh et al., 2002; Shen et al., 2014), it is likely that impairments in motor response inhibition may also be associated with cannabis use.

Prior neuropsychological studies highlight deficits in inhibitory cognitive control in cannabis users (Crean et al., 2011; Ganzer et al, 2016; Infante et al., 2020), with others suggesting mixed evidence regarding residual and long-term effects (Crean et al., 2011; Grant et al., 2012; Griffith-Lendering et al., 2012). Regarding motor response inhibition, behavioral work has demonstrated impaired motor response inhibition in individuals given acute administration of delta-9-tetrahydrocannabinol (Δ-9-THC), a primary compound found in cannabis (Bhattacharyya et al., 2015; McDonald et al., 2003; Ramaekers et al., 2006), and individuals who engage in

regular cannabis use (Behan et al., 2014; Bolla et al., 2002). However, others have also reported no differences in response inhibition accuracy among cannabis users (Filbey & Yezhuvath, 2013; Gonzalez et al., 2012; Hester et al., 2009; Smith et al., 2011; Tapert et al., 2007; Wallace et al., 2020) and individuals administered Δ-9-THC (Borgwardt et al., 2008), implying that these individuals may maintain motor response inhibition function. In support of this view, a recent meta-analysis found that behavioral deficits in motor response inhibition in cannabis users are mostly non-existent (Smith et al., 2014). However, it should be noted that many of these studies used task designs that were relatively easy to identify neuronal activity using fMRI (Borgwardt et al., 2008; Filbey & Yezhuvath, 2013; Hester et al., 2009; Smith et al., 2011; Tapert et al., 2007; Wallace et al., 2020). Ultimately, the association between motor response inhibition behavioral performance and cannabis use is inconsistent, with some reporting deficits in cannabis users, and others not observing such effects, albeit in potentially easier versions of tasks used to assess response inhibition.

Although the literature on the effects of cannabis use on motor response inhibition has yielded inconsistent and even null behavioral findings, differences in neural activity between cannabis users and healthy controls is often reported (Borgwardt et al., 2008; Filbey & Yezhuvath, 2013; Hester et al., 2009; Smith et al., 2011; Tapert et al., 2007; Wallace et al., 2020), suggesting that the underlying neural circuitry recruited for successful motor response inhibition may be altered by cannabis use. Supporting this view, various brain regions (Howlett et al., 2002; Smith et al., 2011), including structures constituting the prefrontal cortex (Elphick & Egertova, 2001) that are known to contribute to response inhibition (Aron & Poldrack, 2005; Boehler et al., 2010; Chevrier et al., 2004; Floden & Stuss, 2006; Kelly et al., 2004; Li et al., 2008; Neo et al., 2011; Rubia et al., 2003; Wager et al., 2005), contain a high density of

cannabinoid-1-receptors, which are activated by Δ-9-THC found in cannabis. As such, previous work has identified altered structural (Matochik et al., 2005) and functional activity in frontal brain regions in cannabis users completing motor response inhibition tasks compared to healthy controls (Behan et al., 2014; Hester et al., 2009; Smith et al., 2011; Tapert et al., 2007; Wallace et al., 2020). Specifically, cannabis users displayed greater activity in dlPFC activity, middle frontal gyrus, and premotor cortex (Smith et al., 2011; Tapert et al., 2007). Others have also identified decreased anterior cingulate cortex and right insula activity (Hester et al., 2009), but increased activity in the inferior frontal gyrus (Behan et al., 2014; Wallace et al., 2020), a key region responsible for contributing to response inhibition (Aron et al., 2007; Aron et al., 2003; Aron & Poldrack, 2006; Aron et al., 2004; Chambers et al., 2007; Chambers et al., 2006; Chevrier et al., 2007; Erika-Florence et al., 2014; Garavan et al., 1999; Li et al., 2006a; Matthews et al., 2005; Rubia et al., 2001, 2003, 2007; Xu et al., 2017). In addition, despite not finding regional activity differences, Filbey and Yezhuvath (2013) observed increased functional connectivity between frontal control regions, including the rIFG, and the substantia nigra in cannabis users. Importantly, individuals who reported greater severity of Cannabis Use Disorder demonstrated greater connectivity between these two regions, leading the authors to suggest that this increased functional connectivity was necessary for cannabis users to perform appropriately on response inhibition relative to their healthy control peers. In contrast, the acute administration of Δ-9-THC has been found to reduce activity in the inferior frontal gyrus (Bhattacharyya et al., 2015; Borgwardt et al., 2008), suggesting differential effects on this underlying neural circuitry based on acute administration versus more long-term effects associated with regular cannabis use. Nonetheless, a recent meta-analysis identified functional abnormalities in three key regions in cannabis users: the anterior cingulate cortex, striatum, and dlPFC (Yanes et al., 2018).

Specifically, cannabis use is associated with decreased activity in the anterior cingulate cortex and dlPFC, but increased activity in the striatum. Thus, most neuroimaging work posits functional alterations within frontal regions contributing to the fronto-basal-ganglia inhibition network necessary for motor response inhibition in cannabis users.

Overall, this body of literature indicates that cannabis users are likely to perform to the same degree as their healthy control peers when completing a motor response inhibition task. However, this comes at the cost of alterations in functional activity in frontal regions necessary to contribute to successful response inhibition, indicating an underlying cognitive processing efficiency deficit in these populations. Therefore, despite maintaining intact behavioral motor response inhibition performance, individuals who engage in regular cannabis use require greater recruitment of structures implicated in the fronto-basal-ganglia inhibition network.

**Anxiety and Cannabis Use**

Behavioral performance and the underlying neural circuitry associated with motor response inhibition are influenced by anxiety (Eysenck et al., 2007; Gray & McNaughton, 2000; Grillon et al., 2017a, 2017b) and cannabis use (Smith et al., 2011; Tapert et al., 2007; Wallace et al., 2020; Yanes et al., 2018). However, the specific association between motor response inhibition and cannabis use interacting with anxiety is still unclear. In addition, these constructs are often studied independently in previous work, failing to account for the effects the other might have in influencing motor response inhibition. This is critical, given evidence reporting a high comorbidity rate between anxious symptomology and anxiety disorders, and cannabis use (Buckner & Carroll, 2010; Butler, 2019; Crippa et al., 2009; Dorard et al., 2008; Teesson et al., 2012; Young-Wolff et al., 2020). Specifically, anxious individuals will often use cannabis as an anxiolytic medication (Buckner et al., 2007; Kosiba et al., 2019; Menary et al., 2011; Reinarman

et al., 2011; Sarvet et al., 2018; Wycoff et al., 2018). Contrary to these expected outcomes of using cannabis based on this rationale, many instead report feeling more anxious after use (Bhattacharyya et al., 2017; Crippa et al., 2009; D'Souza et al., 2004; Hall & Solowij, 1998; Kedzior & Laeber, 2014; Mammen et al., 2018; Rusby et al., 2019; Witkin et al., 2005). In line with these findings, others have demonstrated that cannabis use predicts later onset of anxiety (Epstein et al., 2015; Mammen et al., 2018; Wittchen et al., 2007), and that reducing cannabis use yields less anxious symptomatology (Hser et al., 2017), suggesting that cannabis use may serve as a potential risk factor in the development and maintenance of anxiety disorders. However, the effects of cannabis on anxiety-related symptomatology and behaviors remains inconclusive (Andrade et al., 2019), making it difficult to determine whether cannabis use will lead to the development of anxiety disorders or if more anxious individuals tend to engage in cannabis use. Therefore, anxiety and cannabis use commonly co-occur, with the directionality of this relationship currently unclear.

Despite the high comorbidity between anxiety and cannabis use, only two studies have thus far examined how these variables might interact to influence motor response inhibition (Borgwardt et al., 2008; Spechler et al., 2020). Specifically, Borgwardt and colleagues (2008) administered Δ-9-THC to healthy participants prior to completing a Go/No-Go task. They found reduced right anterior cingulate cortex and inferior frontal gyrus activation in individuals administered Δ-9-THC. In addition, despite participants reporting enhanced levels of anxiety following administration compared to baseline, functional activity changes resulting from this acute administration were not associated with changes in self-reported anxiety level. As such, the authors concluded that these underlying neural alterations were directly due to the influence of Δ-9-THC, and not anxiety. Furthermore, work by Spechler and colleagues (2020) also failed to

identify differences in functional activity when comparing three groups: One including individuals who had received a diagnosis of anxiety or depression at least once along with a diagnosis of cannabis dependency or use at least 50 times in the past year; another including the same criteria as the first group, but without cannabis dependency diagnosis or heavy cannabis use; and a final group consisting of healthy controls without anxiety and/or depression diagnoses or cannabis use diagnoses. Specifically, they found a lack of behavioral and functional activation differences in the right opercularis, right orbitalis, and right ventral and dorsal anterior insula between groups. Despite these null effects, their work did observe enhanced self-reported impulsivity in the group diagnosed with anxiety and/or depression with cannabis dependency compared to the healthy control group and the group diagnosed with anxiety and/or depression without cannabis dependency. Thus, these initial results suggest a lack of interaction between anxiety and cannabis use on response inhibition, at least when looking at individuals with comorbid anxiety and depression, and when examining acute administration of cannabis.

Although these two studies (Borgwardt et al., 2008; Spechler et al., 2020) failed to identify interactions between anxiety and cannabis use in predicting response inhibition performance and neural activity, several important limitations pertaining to these studies should be noted. First, Borgwardt and colleagues (2008) gave an acute dose of Δ-9-THC to healthy participants, which may differentially impact response inhibition compared to individuals who regularly engage in cannabis use, as described prior. Second, this work also assessed self-reported changes in anxiety resulting from the administration of Δ-9-THC, and thus may be considered as more of an impact resulting from an anxious *state*. Given that state and trait anxiety have been shown to differentially impact neural systems responsible for various cognitive processes (Bishop et al., 2007; Pacheco-Unguetti et al., 2010), these findings may

instead reflect interactions between an anxious *state* and acute cannabis use. In line with this rationale, it is important to note that the participants in Borgwardt and colleagues' (2008) work were healthy participants, who had not used cannabis within the past month, and were not assessed for current anxious symptomatology or the presence of clinical anxiety disorders. Third, although Spechler and colleagues (2020) included individuals with a current diagnosis or a history of diagnosed anxiety and cannabis use, many of these individuals also had comorbid Major Depressive Disorder, which may have contributed to variability in their results. Finally, the work by Spechler and colleagues (2020) used a group approach, which fails to account for how the variability in individual differences in anxious symptomology interacts with cannabis use to predict response inhibition. As such, while these results suggest that individuals with comorbid anxiety and depressive disorders engaging in cannabis use show little influence on response inhibitory processes, they fail to account for potential individual differences in anxious symptomatology, on a continuous spectrum from low to high anxiety, may interact with cannabis use and impact motor response inhibition.

**Current Study**

Given the inconsistent and often contradictory effects of anxiety and cannabis use on response inhibition, and the lack of studying these variables in conjunction, the current study aimed to address three primary goals. First, we examined how individual differences in anxious symptomatology, which has been shown to serve as a risk factor for the development and maintenance of anxiety disorders (Shackman et al., 2016), affects behavioral and neural outcomes associated with motor response inhibition. Second, we analyzed how motor response inhibition outcomes are impacted in individuals testing positive for recent cannabis use, measured through urine toxicology. Finally, we sought to identify interactions between recent

cannabis use (positive or negative urine toxicology results) and anxious symptomology (reported through the Depression, Anxiety, and Stress Scale; Lovibond & Lovibond, 1995) in predicting behavioral and neural activity differences in motor response inhibition.

We used a modified Stop-Signal task following previous designs (Leibenluft et al., 2007; Jovanovic et al., 2013) to assess motor response inhibition. Importantly, this design omits the typical delay period between the Go and Stop signal, and instead the Stop signal is shown in conjunction with the Go signal on Stop trials. Our participants consisted of individuals recruited from a larger study that had previously experienced a traumatic injury (e.g., motor vehicle injuries, falling, stabbing, domestic violence, etc.). Given that previous work has indicated that motor response inhibition performance is negatively impacted by PTSD diagnosis (Swick et al., 2012; Wu et al., 2010) and recent traumatic experiences (van der Bij et al., 2020; van Rooij et al., 2018), both of which also involve experiencing anxiety, it is possible that some of the variability in our outcomes may not be due entirely to recent cannabis use and its interaction with anxious symptomology alone, and may also reflect the impact of anxious symptomatology and recent cannabis use associated with trauma-related anxiety and PTSD. To account for this, we also examined PTSD symptoms using the PTSD Checklist for the DSM-5 (PCL-5; Blevins et al., 2015) and their interaction with recent cannabis use in predicting motor response inhibition separate from our anxiety analyses.

We hypothesized that behavioral motor response inhibition, measured through Stop trial accuracy, would not be influenced by anxious symptomology (Figure 1a), recent cannabis use urine toxicology outcomes (Figure 1a), or their interaction (Figure 1b). However, we anticipated observing impaired motor response inhibition in individuals with greater PTSD (Figure 1a), and an interaction between PTSD and recent cannabis use (Figure 1c), such that individuals with

greater PTSD, reflected through higher PCL-5 total scores, who also test positive for recent cannabis use perform to the same degree as individuals low in PTSD who also recently used cannabis. These behavioral hypotheses are based on the work of others observing null differences in motor response inhibition in individuals with clinical levels of anxiety (Grillon et al., 2017b, Hallion et al., 2017; Herrmann et al., 2003; Kim et al., 2007; Leonard & Abramovitch, 2019), and because studies that did find effects primarily focused on induced state anxiety (Choi & Cho, 2020; Grillon et al., 2017a; Robinson et al., 2013a; Roxburgh et al., 2020). Regarding individuals testing positive for recent cannabis use, although prior neuropsychological studies highlight deficits in inhibitory cognitive control in cannabis users (Crean et al., 2011; Ganzer et al, 2016; Infante et al., 2020), studies using fMRI have reported null effects on motor response inhibition performance observed in regular cannabis users (Borgwardt et al., 2008; Filbey & Yezhuvath, 2013; Hester et al., 2009; Smith et al., 2011; Tapert et al., 2007; Wallace et al., 2020), supporting our prediction. Our prediction for reduced behavioral performance in those with greater PTSD is primarily based on previous work showing motor response inhibition deficits in individuals diagnosed with PTSD (Swick et al, 2012; Wu et al., 2010; although see Shucard et al., 2008), and those who recently experienced a traumatic event (van der Bij et al., 2020; van Rooij et al., 2018). While PTSD encompasses anxious symptomology (Craske et al., 2009), there are also specific factors present in PTSD that are not expressed in other anxiety-related disorders that may contribute to deficits in response inhibition that have not commonly been observed in anxiety. Current theories suggest that response inhibition deficits in PTSD are primarily associated with the hyperarousal and re-experiencing symptom clusters of PTSD (Aupperle et al., 2012; Swick et al., 2012). Thus, symptomology related to hyperarousal and/or the re-experiencing of a traumatic event may produce deficits in response inhibition, while

anxious symptomology, such as worry and lowered levels of arousal compared to that experienced in PTSD, do not result in such deficits.

Our neuroimaging predictions pertained to four key regions: dlPFC, dACC, rIFG, and the striatum. First, we hypothesized that **(1)** anxious individuals would demonstrate increased activity in the dlPFC, dACC, rIFG, and striatum during trials requiring motor response inhibition (Figure 2). Because of the paucity of work using fMRI to examine motor response inhibition in anxious individuals, our predictions for anxiety are largely based on theoretical accounts that anxious individuals perform to the same degree as their less anxious peers, but experience underlying processing efficiency costs to perform at this equivalent level (Berggren & Derakshan, 2013; Derakshan & Eysenck, 2009; Eysenck & Derakshan, 2011; Eysenck et al., 2007). Supporting this rationale, Karch and colleagues (2008) observed increased activity in the rIFG in high trait anxious individuals, but it should be noted this was in participants with alcohol dependence. Others have also found increased striatal and rIFG activity in anxious individuals (Torrisi et al., 2016). Despite this, one study did identify reduced dlPFC and dACC activity in anxious individuals (Forster et al., 2015), contrary to this theoretical position and our hypotheses.

Regarding urine toxicology for recent cannabis use, we predicted **(2)** that individuals testing positive for recent cannabis use would show increased activity in the dlPFC, rIFG, and striatum, but decreased activity in the dACC (Figure 3) compared to those who tested negative for recent cannabis use. Our predictions concerning the impact of recent cannabis use are more strongly supported by previous work indicating increased dlPFC, rIFG, and striatum activity (Behan et al., 2014; Smith et al., 2011; Tapert et al., 2007; Wallace et al., 2020; Yanes et al., 2018), and reduced dACC activity (Hester et al., 2009; Yanes et al., 2018) during motor response

inhibition in regular cannabis users. However, it should be noted that we may also observe reduced activity in the dlPFC based on recent reports (Yanes et al., 2018).

For PTSD, we anticipated **(3**) decreased activity in the dlPFC, dACC, rIFG, and striatum (Figure 4). Our PTSD predictions were developed based on behavioral deficits observed in this population (Swick et al., 2012; Wu et al., 2010) and among those with recent traumatic experiences (van der Bij et al., 2020; van Rooij et al., 2018), and that this behavioral impairment would be accompanied by reduced activity in these regions known to support effective motor response inhibition. In support of these hypotheses, some have observed reduced anterior cingulate cortex activity in PTSD patients relative to healthy controls (Falconer et al., 2013; Stevens et al., 2016), and reduced dlPFC activity in youth with PTSD (Carrion et al., 2008). In addition, others have found increased striatal activity in those who were successfully treated for PTSD (Falconer et al., 2013), although other work has failed to identify differences in the rIFG in this population (Jovanovic et al., 2013).

Finally, we made **(4)** no *a priori* predictions for interactions between recent cannabis use and anxiety or PTSD given the lack of previous work examining these factors in conjunction, and our predicted main effects of each independent variable (i.e., anxious symptomology and PTSD) with recent cannabis use hypothesized above. Given the strong associations between anxiety and cannabis use (Buckner & Carroll, 2010; Butler, 2019; Crippa et al., 2009; Dorard et al., 2008; Teesson et al., 2012; Young-Wolff et al., 2020) and PTSD and cannabis use (Cornelius et al., 2010; Cougle et al., 2011; Kevorkian et al., 2015), and the lack of studies examining these factors together, this study addresses several novel questions: how is motor response inhibition behavioral performance and neural activity influenced by co-occurring anxiety and recent cannabis use, and PTSD and recent cannabis use. Thus, the results of this study will contribute to

each factor's literature concerning motor response inhibition, while also providing a framework to understand how these variables interact with each other to influence motor response inhibition. Specifically, these findings will allow for the assessment of the overall influence that anxiety and PTSD have on behavioral motor response inhibition performance, and illustrate whether recent cannabis use interacts with these constructs to effect these outcomes. They will also provide biomarkers reflective of compensatory neural activity required for successful completion of motor response inhibition in individuals with elevated anxiety and/or those who engaged in recent cannabis use. Ultimately, these findings will help inform clinical treatment models posed at alleviating potential detrimental effects of anxiety, recent cannabis use, and PTSD on response inhibition.

## Method

### Participants

Initially screened participants ($n$ = 1,026) were recruited from a Level I trauma center emergency department in southeastern Wisconsin between 2016 and 2020 as part of the larger Imaging Study on Trauma & Resilience (iSTAR) project. Participants were included in the iSTAR project if they were: (1) English-speaking, (2) between 18-60 years of age, (3) had scheduled a visit within 30 days post-traumatic injury, (4) had experienced a traumatic event meeting Criterion A of the PTSD diagnosis from the DSM-5 (American Psychiatric Association, 2013), (5) and scored a minimum of three on the Predicting PTSD Questionnaire (Rothbaum et al., 2014) or endorsed a near-death experience from the traumatic event. Participants were excluded from the iSTAR project if they: (1) scored 13 or lower on the Glascow Coma Scale (Sternbach, 2000; Teasdale et al., 2014), (2) had a spinal cord injury accompanied with neurological deficits or were diagnosed with any neurological conditions, (3) a self-inflicted

traumatic injury, (4) severe visual or hearing impairments, (5) a history of psychotic of manic symptoms, (6) current antipsychotic medication use, (7) had a note in the medical record indicating current substance abuse, (8) the inability to verbally communicate, (9) had experienced a sexual assault traumatic event, (10) a non-police hold to be released to jail, (11) displayed moderate to severe cognitive impairment secondary to trauma-related head injury, (12) had a note in the medical record that they tested positive for alcohol ( > 0.08 BAC), illegal drugs, or prescription narcotics at the time of the trauma, (13) had eye conditions that prevented the use of eye tracking assessment, or (14) had severe traumatic brain injury. We did not screen for mild-traumatic brain injury, and instead used scores from the Glasgow Coma Scale as an indicator of no more than mild brain injury. Following the inclusion and exclusion criteria from the iSTAR project, a total 92 participants (54 females; $M_{age}$ = 32.82, $SE_{age}$ = 1.18) completed the Stop-Signal task two weeks post-trauma (Table 1). All participants were financially compensated for participation in the study. Seventy-one (77.17%) participants of this final sample met criteria for mild traumatic brain injury. Of the 92 participants that completed the Stop-Signal task, the following number of individuals tested positive for the presence of cannabis (40, 43.48%), oxycodone (6, 6.52%), methadone (1, 1.09%), cocaine (2, 2.17%), amphetamines (1, 1.09%), opiates (2, 2.17%), methamphetamine (1, 1.09%), and benzodiazepines (6, 6.52%). No participants tested positive for the presence of Barbiturates, PCP, Propoxyphene, and MDMA.

**Behavioral sample.** Of the 92 participants that completed the Stop-Signal task two weeks post-traumatic event, five participants demonstrated outlier values for behavioral performance on the task ($n$ = 1, Stop trial accuracy; $n$ = 3, Go trial accuracy; $n$ = 1 for Go trial RT) and were excluded from further analyses, leaving 87 participants to be used for behavioral analyses (51 females; $M_{age}$ = 32.14, $SE_{age}$ = 1.16; Table 1). Of this total sample ($n$ = 87), 49

participants (30 females; $M_{age}$ = 34.83, $SE_{age}$ = 1.51) tested negative and 38 participants (21 females; $M_{age}$ = 28.82, $SE_{age}$ = 1.67) tested positive for recent cannabis use, assessed via urine toxicology (Table 2). To maintain adequate statistical power without unnecessarily dropping participants and given the quasi-experimental design of the current study, age and gender matching across the recent cannabis use groups was not feasible. Therefore, we included age and gender as covariates in all our behavioral analyses.

**fMRI sample.** Participants from our behavioral sample ($n$ = 87) were further excluded from fMRI data analyses due to technical difficulties during fMRI data acquisition and or missing fMRI data ($n$ = 8) or having more than 20% of volumes censored as being outliers ($n$ = 13), resulting in a total of 66 participants (42 females; $M_{age}$ = 32.16, $SE_{age}$ = 1.25; Table 1) to be used for fMRI analyses. Of these 66 participants, 34 participants (23 females; $M_{age}$ = 36.08, $SE_{age}$ = 1.83) tested negative and 32 participants (19 females; $M_{age}$ = 28.01, $SE_{age}$ = 1.39) tested positive for recent cannabis use based on urine toxicology (Table 2). As in our behavioral sample, we included age and gender as covariates in all our fMRI analyses to control for these variables.

## Materials and Procedures

**Anxious symptomology.** Participants completed the Depression, Anxiety, and Stress Scale (DASS-21; Lovibond & Lovibond, 1995; Table 3) as a measure of our anxious symptomology independent variable. Specifically, scores from the DASS-21's anxiety subscale were used to measure symptoms of anxiety experienced within the past week. The DASS-21 holds relatively high internal reliability for its depressive (α = 0.81), anxiety (α = 0.89), and stress (α = 0.78) subscales, and shows high validity across cultures (Antony et al., 1998; Bottesi

et al., 2015; Coker et al., 2018; Henry & Crawford, 2005; Sinclair, 2012; Tonsing, 2014; Vasconcelos-Raposo et al., 2013).

***Anxious symptomology descriptive statistics for behavioral sample.*** For our behavioral sample ($n$ = 87), scores on the DASS-21 anxiety subscale ranged from 0 to 36 ($M$ = 8.23, $SE$ = 0.87). These scores ranged from 0 to 36 ($M$ = 8.73, $SE$ = 1.29) in the group testing negative for recent cannabis and from 0 to 20 ($M$ = 7.58, $SE$ = 1.09) in the group testing positive for recent cannabis use. Scores on the anxiety subscale did not differ between groups, $t(85) = 0.660$, $p$ = 0.511, $d$ = 0.143, $BF_{10} = 0.273$ (Appendix A, Figure 1A). Internal reliability was high across the whole behavioral sample ($\alpha = 0.84$), and for participants testing negative ($\alpha = 0.87$) and positive ($\alpha = 0.78$) for recent cannabis use.

***Anxious symptomology descriptive statistics for fMRI sample.*** Similar to our behavioral sample, the distribution of scores on the DASS-21 anxiety subscale in the fMRI sample ($n$ = 66) ranged from 0 to 36 ($M$ = 8.18, $SE$ = 0.98). Participants in the group testing negative for recent cannabis use displayed scores from 0 to 36 ($M$ = 8.94, $SE$ = 1.55) and scores in group testing positive for recent cannabis use ranged from 0 to 20 ($M$ = 7.38, $SE$ = 1.18). DASS-21 anxiety scores did not differ between groups in this sample, $t(64) = 0.796$, $p = 0.429$, $d$ = 0.196, $BF_{10}$ = 0.330 (Appendix A, Figure 1B). Internal reliability was also high across the entire fMRI sample ($\alpha = 0.89$), and for participants testing negative ($\alpha = 0.90$) and positive ($\alpha = 0.89$) for recent cannabis use.

**PTSD symptomology.** To account for potential influence on our dependent variables resulting from PTSD symptoms and the trauma experienced in our sample, we used total scores from the PTSD Checklist for the DSM-5 (PCL-5; Blevins et al., 2015; Table 4) to serve as a predictor replacing anxiety in separate models for all our analyses. Higher total scores on this

measure have been used to indicate PTSD symptom severity in individuals experiencing traumatic injuries (Geier et al., 2019). The PCL-5 has demonstrated high internal reliability (α = 0.95; Blevins et al., 2015), and has been adapted across various cultures (Ibrahim et al., 2018; Lima et al., 2016; Van Praag et al., 2020). In the current study, the PCL-5 scores were completed for the index trauma participants had experienced two weeks prior.

***PTSD descriptive statistics for behavioral sample.*** Scores on the PCL-5 ranged from 0 to 73 ($M$ = 27.29, $SE$ = 18.29) for the behavioral sample ($n$ = 87). Participants in the group testing negative for recent cannabis use reported a range from 0 to 73 ($M$ = 27.18, $SE$ = 2.74) and participants in the group testing positive recent cannabis use displayed scores from 0 to 59 ($M$ = 27.42, $SE$ = 2.80). PCL-5 Total scores did not differ between these groups in the behavioral sample, $t(85) = -0.060$, $p = 0.953$, $d = 0.013$, $BF_{10} = 0.226$ (Appendix A, Figure 2A). Internal consistency was high for the full behavioral sample (α = 0.95), and in the negative (α = 0.95) and positive (α = 0.94) recent cannabis use groups.

***PTSD descriptive statistics for fMRI sample.*** The distribution of total scores on the PCL-5 in the fMRI sample ranged from 0 to 71 ($M$ = 27.79, $SE$ = 2.24). In thegroup testing negative for recent cannabis use, PCL-5 scores ranged from 2 to 71 ($M$ = 28.74, $SE$ = 3.28) and from 0 to 59 ($M$ = 26.78, $SE$ = 3.07) in the group testing positive for recent cannabis use. Total scores on the PCL-5 did not significantly differ between these groups, $t(64) = 0.433$, $p = 0.666$, $d = 0.107$, $BF_{10} = 0.273$ (Appendix A, Figure 2B). The fMRI sample showed high internal reliability (α = 0.94), and this was observed for participants in the recent negative (α = 0.95) and positive (α = 0.94) cannabis use groups.

**Recent cannabis use screening.** Urine analyses were conducted on all participants to assess recent cannabis use. Specifically, urine samples (4 oz specimen cups) were collected from

participants and tested for the presence of cannabis, oxycodone, methadone, barbiturates, phencyclidine, propoxyphene, cocaine, amphetamine, opiate, methamphetamine, benzodiazepine and MDMA. Urine analyses were completed at A-TRU and the local Pavilion Scanning Facility Bathroom. Urine analyses were completed using an easy@home drug test kit (Easy Healthcare). Specifically, cutoff levels for screening substances were as follows: THC (52 mg/mL), opiates (2000 ng/mL), cocaine (300 ng/mL), amphetamine (1000ng/mL), barbiturates (300 ng/mL), MDMA (500 ng/mL), methamphetamine (1000 ng/mL), PCP (25 ng/mL), and methadone (300 ng/mL).

It is important to note that this measure of recent cannabis use has several limitations, specifically pertaining to the sensitivity of detecting how recently individuals engaged in cannabis use. This is because the test can detect the presence of cannabis in urine between very recent use (~ 2 hours ago) through use that occurred three weeks prior. In addition, this test does not allow us to disentangle the frequency of recent cannabis use over this duration. This is critical given the frequency of use can impact how sensitive this test is in detecting recent cannabis use from a more distant prior time point. For example, individuals who engaged in chronic cannabis use may test positive even if they haven't used cannabis within the past 3 months. In contrast, individuals who do not regularly engage in cannabis use may test negative even if they had used cannabis several days prior to the test. Thus, this measure of cannabis use is imperfect, but was the only source containing data for this measure from the larger iSTAR sample. Finally, this measure of cannabis only allows for the evaluation of recent cannabis use, and does not allow for the examination of more chronic or acute effects of cannabis use.

Additional screening for self-reported total cannabis use in the past month was administered to approximately half (47.13%; $n$ = 41) of the participants in our behavioral sample

and half (50%, *n* = 33) of the participants in our fMRI sample. Participants were required to indicate on a 7-point Likert scale how often they engaged in cannabis use for within the past month (Appendix B, Table 1). Using this assessment, we cross-examined the self-reported total cannabis use within the past month with the outcomes from urine screening analyses to examine the consistency of the self-reported data. Specifically, participants' self-report data was considered "consistent" if their response matched the outcomes from their urine analyses, and "inconsistent" if their response did not match the urine analyses screening. For example, if a participant reported "Never" to using cannabis within the past month and their urine analysis indicated negative results for the presence of cannabis, then the participant would be categorized as consistent. In contrast, if the participant reported "Never" to using cannabis within the past month, but the urine analysis indicated positive results for the presence of cannabis, then the participant would be categorized as inconsistent.

***Behavioral sample recent cannabis use consistency.*** Only about half (47.13%; *n* = 41) of the participants in the behavioral sample completed the self-reported cannabis use screening. Cross-examination of participants' self-report and urine analyses indicated that a majority of participants answered in a manner consistent with their urine analyses for self-reported cannabis use within the past month (95.12%; *n* = 39; Appendix B, Figure 1A). Two participants' (4.88%) self-reported data contradicted the results from their urine analyses. Specifically, one participant indicated never using cannabis use within the past month while the urine analysis tested positive for the presence of cannabis, and another participant reported using cannabis more than once per day within the past month, but received a negative test result from the urine analysis. Most of the participants' responses were consistent with their urine analyses results within the negative (96.15%; *n* = 25; Appendix B, Figure 1B) and positive (93.33%; *n* = 25; Appendix B, Figure 1C)

recent cannabis use groups. As described above, one participant in each group had inconsistent self-report data with their urine analyses results.

***fMRI sample recent cannabis use consistency.*** Half (50%, *n* = 33) of the participants in the fMRI sample completed the recent cannabis use self-report screening. Most participants (93.94%; *n* = 31; Appendix B, Figure 2A) answered the self-report assessment for total cannabis use within the past month in a manner consistent with their urine analyses. The same two participants (4.88%) that had inconsistent self-report and urine analyses in the behavioral sample were present in the fMRI sample. Participants' self-report responses were mostly consistent with their urine analyses in the negative (95.24%; *n* = 20; Appendix B, Figure 2B) and positive (91.67%; *n* = 11; Appendix B, Figure 2C) recent cannabis use groups. Like with the behavioral sample, one participant in each group was categorized as inconsistent.

**Alcohol use.** Given the high comorbidity between cannabis use and alcohol use (Lee et al., 2019a, 2019b; Yurasek et al., 2017), and that individuals with greater PTSD severity engage in greater alcohol use (Kearns et al., 2019), we assessed the quantity of alcohol consumption within the past year as a covariate for our analyses. Specifically, we used scores from the Alcohol Use Disorders Identification Test (AUDIT; Babor et al., 1992; Saunders et al., 1993; Table 5), in which higher scores reflect greater alcohol consumption in participants. Importantly, scores below 8 indicate a low risk for alcohol consumption (i.e., less than 8, Conigrave et al., 1985).

***Alcohol use descriptive statistics for behavioral sample.*** AUDIT scores for the behavioral sample (*n* = 87) ranged from 0 to 19 (*M* = 3.97, *SE* = 0.46). Participants in the group testing negative for recent cannabis use reported scores from 0 to 18 (*M* = 3.96, *SE* = 0.61), while those in the group testing positive for recent cannabis use had scores from 0 to 19 (*M* =

3.97, *SE* = 0.71). Total scores on the AUDIT were similar across both groups in the behavioral sample, $t(85) = -0.016$, $p = 0.988$, $d = 0.003$, $BF_{10} = 0.226$ (Appendix A, Figure 3A). Internal consistency was high for the full behavioral sample ($\alpha = 0.79$), and in the negative ($\alpha = 0.80$) and positive ($\alpha = 0.78$) recent cannabis use groups.

***Alcohol use descriptive statistics for fMRI sample.*** The fMRI sample's (*n* = 66) AUDIT scores ranged from 0 to 18 (*M* = 3.85, *SE* = 0.51), while scores in the group testing negative for recent cannabis use were between 0 and 18 (*M* = 4.29, *SE* = 0.77) and from 0 to 13 (*M* = 3.38, *SE* = 0.66) in the group testing positive for recent cannabis use. Total scores on the AUDIT did not significantly differ between the groups in the fMRI sample, $t(64) = 0.900$, $p = 0.372$, $d = 0.222$, $BF_{10} = 0.356$ (Appendix A, Figure 3B). Internal consistency in the entire fMRI sample was high ($\alpha = 0.79$), as well as within the negative ($\alpha = 0.81$) and positive ($\alpha = 0.76$) recent cannabis use groups.

**Nicotine use.** Cannabis use has high comorbidity with nicotine use (Lee et al., 2019a, 2019b; Subramaniam et al., 2016), and is prevalent in individuals exhibiting PTSD symptomology (Thorndike et al., 2006). Because of this, we initially aimed to include this construct as a covariate in our analyses using a screening for self-reported nicotine use for the total number of days participants smoked cigarettes within the past month, and the average number of cigarettes smoked each day. However, only 39 participants from the behavioral sample (44.83%) and 31 participants from the fMRI sample (46.97%) completed this screening, leading us to omit including this variable as a covariate in our analyses. Nonetheless, we have included the descriptive statistics for this variable below.

***Nicotine use descriptive statistics for behavioral sample.*** Of the 39 participants (44.83%) completing the nicotine use screening from the behavioral sample (*n* = 87), the number of days

reported smoking within the past month ranged between 0 and 30 (*M* = 5.31, *SE* = 1.76) with participants indicating an an average 6.38 cigarettes smoked per day (*SE* = 2.15). Only 8 participants (20.51%) indicated smoking at least one day within the past month. Reported total number of days cigarettes were smoked ranged between 0 and 30 (*M* = 2.40, *SE* = 1.66) in the group testing negative for recent cannabis use and 0 to 30 (*M* = 10.50, *SE* = 3.57) in the group testing positive recent cannabis use. Only 2 (8.00%) participants in the group testing negative for recent cannabis use (*n* = 25) and 6 (42.86%) participants in the group testing positive for recent cannabis use (*n* = 14) reported smoking at least one day within the past month. The average number of cigarettes smoked in the group testing negative for recent cannabis use was 12 (*SE* = 8.00) and 4.5 (*SE* = 1.26) in the group testing positive for recent cannabis use. There were no significant differences between groups in the total number of days participants smoked cigarettes within the past month, $t(18.75) = -2.055$, $p = 0.054$, $d = 0.728$, $BF_{10} = 1.797$ (Appendix C, Figure 1A), and the average number of cigarettes smoked per day, $t(1.05) = 0.926$, $p = 0.518$, $d = 0.844$, $BF_{10} = 0.856$ (Appendix C, Figure 2A).

***Nicotine use descriptive statistics for fMRI sample.*** Approximately 46.97% (n = 31) of participants completed the nicotine use screening in the fMRI sample (*n* = 66). For the entire fMRI sample, total number of days cigarettes were smoked ranged between 0 and 30 (*M* = 5.71, *SE* = 2.01) and an average of 6.43 cigarettes smoked per day (*SE* = 2.49) were reported. Seven participants (22.58%) reported smoking at least one day within the past month. In the negative recent cannabis use group, number of days cigarettes were smoked within the past month ranged between 0 and 30 (*M* = 3.00, *SE* = 2.06), with an average of 12 cigarettes smoked per day (*SE* = 8.00) in indiviudals who reported they had smoked within the past month. Number of days cigarettes were smoked in the past month in the group testing positive for recent cannabis use

ranged from 0 to 30 ($M$ = 10.64, $SE$ = 3.96), with individuals who reported smoking within the past month in this group reporting an average of 4.20 cigarettes smoked per day ($SE$ = 1.50). Two (10%) participants in the group testing negative for recent cannabis use ($n$ = 20), and 5 (45.45%) participants in the group testing positive for recent cannabis use group indicated smoking at least one day within the past month. No significant differences in the total number of days cigarettes were smoked in the past month, $t(15.57) = -1.710$, $p = 0.107$, $d = 0.673$, $BF_{10} = 0.965$ (Appendix C, Figure 1B), and average number of cigarettes smoked per day, $t(1.07) = 0.958$, $p = 0.505$, $d = 0.935$, $BF_{10} = 1.036$ (Appendix C, Figure 2B), were found between the groups.

**Procedures.** All participants from the iSTAR project were initially screened in the emergency department and contacted via telephone following discharge. The iSTAR project protocol was approved by the Institutional Review Board at the Medical College of Wisconsin. Participants were provided written informed consent prior to participation. Participants completed visits at multiple timepoints for the iSTAR project, but only data from the Stop-Signal task from the first time-point, which was two weeks following the traumatic event, is reported here. During this 2-week timepoint, participants completed multiple self-report measures, including assessment of anxiety and PTSD (e.g., DASS-21, PCL-5), provided urine samples for substance-use (e.g., recent cannabis use) assessment, and completed neurocognitive assessments. Participants completed a series of tasks, including a modified Stop-Signal task (Figure 5) used to assess motor response inhibition.

Given the impact trauma can have on response inhibition, and the strong association between scores on the DASS-21 anxiety subscale and PCL-5 in the behavioral ($n$ = 87; $r(85) = 0.687$, $p < 0.001$) and fMRI ($n$ = 66; $r(64) = 0.713$, $p < 0.001$) samples, we attempted to

disentangle the effects of PTSD and trauma from anxiety by conducting a principal component analysis (PCA) on all item-level questions from the PCL-5 and DASS-21 anxiety items for participants in the behavioral sample (n = 87). We anticipated this would results in at least two separate factors: one reflecting anxiety and another representing PTSD. Scores for these two factors would then be used as independent variables in all our analyses to examine the impact each construct has on motor response inhibition.

**Stop-Signal task.** Participants completed a modified Stop-Signal task (Figure 5) based on prior work (Leibenluft et al., 2007; Jovanovic et al., 2013). Each trial began with a white fixation cross against a black background (500ms). Next, the fixation cross was replaced by either an "X" or "O" (counterbalanced) Go cue (1000ms), which required participants to press "1" or "2" on a response pad placed in their right hand for the "X" and "O", respectively. These cues would then disappear followed by a blank screen (750ms). On a subset of trials (~34%), a Stop cue was presented requiring participants to withhold a response, indicated by the background surrounding the "X" and "O" cue changing to red.

Participants first completed a practice task following this design. The practice task consisted of five trials (two Go trials, two Stop trials, and one blank trial). Next, participants completed the Stop-Signal task consisting of 208 total trials split across two runs (104 trials per run), 152 of which were either Go or Stop trials. The remaining 56 trials were blank trials that did not present a stimulus. Of the 152 task-specific trials, 100 were Go trials and 52 were Stop trials. Each run was divided into two blocks (38 trials per block). Blocks were separated by a 2000ms ITI.

**Behavioral variables.** Behavioral variables consisted of accuracy (% correct) for Stop and Go trials, and response time (RT) in ms for Go trials. No trials in the task were excluded based on RT.

**fMRI data acquisition.** Magnetic resonance images were collected on a General Electric Discovery MR750 3.0 Tesla scanner with a 32-channel head-coil. A T1-weighted high-resolution anatomical scan (FOV = 240mm, matrix = 256x224, slice thickness = 1mm, 150 slices, TR/TE = 8.2/3.2, flip angle = 12o, voxel size = 1 x 0.938 x 0.938mm) was obtained for co-registration with functional scan data. Functional T2*-weighted echoplanar images (EPI) were acquired (FOV = 22.4mm, matrix = 64x64, slice thickness = 3.5mm, 41 sagittal slices, repetition time (TR)/echo time (TE) = 2000/25ms, flip angle = 77°, voxel size = 3.5 x 3.5 x 3.5mm) across both runs (each run approximately 250.21 seconds), and transformational matrices were concatenated and applied to EPI data. The entire task duration was approximately 10 minutes. Participants were instructed with remain still and keep their eyes open throughout the entire scan.

**fMRI pre-processing.** fMRI data was processed using the Analysis of Functional Neuroimages (AFNI, Cox, 1996; Cox & Hyde, 1997; Gold et al., 1998) and the 'afni_proc.py' script. Acquired anatomical and EPI images, and .1D timing files were accessed to complete each processing block. Slice-timing alignment on EPI data was conducted using the default time 0 TR with quintic interpolation for time series resampling to account for non-simultaneous slice acquisition for each volume. Following this, anatomical data was non-linearly warped to standard Montreal Neurological Institute space (MNI152, McGill University, Montreal, Quebec). Next, the first 3 volumes for each run were removed to prevent pre-steady state artifacts that occur from the magnetic field stabilization at the beginning of each scan. Volume co-registration was applied using the functional volume that held the minimum number of voxels with signal

intensity values that were outliers for each participant using AFNI's '3dToutcount'. The first volume (after removal of first 3 TRs) was used as an index TR for alignment of structural and functional data across each run. EPI data was aligned to anatomical data through non-linear warping. EPI to anatomical alignment quality control was visually inspected through AFNI's visualization software.

Initial observations confirmed that EPI and anatomical images were appropriately aligned for 39 participants when running a Local Pearson Absolute (LPA) cost function with ginormous move function, which assumes large movements occurred over the sessions, resulting in mis-aligned EPI and anatomical images. Twenty-four additional participants displayed appropriate alignment after removing the ginormous move function while keeping the LPA cost function. Three remaining participants' alignments were re-evaluated after removing the ginormous move function and running with Local Pearson Correlation (LPCzz), and found to have appropriate alignment. Thus, all 66 participants were retained due to proper alignment.

The EPI to anatomical matrix was then combined with the transformation matrix from the anatomical to MNI space warped data. A spatial smoothing blur (FWHM = 4 mm) was applied to EPI data, and a mask was applied to this data to remove stray voxels at the edges of the brain that may have occurred due to shifting resulting from application of the transformations or movement. Each run's mean was scaled to 100 for each voxel to show percentage of signal change in functional data. AFNI's '3dDeconvolve' was used to model the time series response for each condition (Go, Stop, Rest). The default gamma (GAM) functional deconvolution will be used for these single-subject regressions, and six motion parameters were included as covariates. Flagged signal outliers (threshold > 10% of voxels in a volume designated as outliers) and motion outliers (Euclidian distance threshold > 0.3mm) were censored out of the final

deconvolved time series data. Following removal of participants ($n$ = 13) due to excessive censor fractions (> 20% of TRs censored), remaining participants ($n$ = 66) used for data analyses had a mean of 5.89% of volumes censored, with 3.25% of volumes censored due to motion and 2.42% of volumes censored due to exceeding signal outlier limits. This resulted in a total of 86.48 (86.48%) Go TRs and 43.82 (84.27%) Stop volumes for data analyses. In addition, our average time-series signal to noise ratio (TSNR) was 310.10 for participants used in fMRI analyses.

**Proposed Analyses**

**Data reduction.** Because of the strong correlations between DASS-21 anxiety subscale and PCL-5 scores in the behavioral ($n$ = 87; $r(85) = 0.687$, $p < 0.001$) and fMRI ($n$ = 66; $r(64) = 0.713$, $p < 0.001$) samples, we conducted a PCA using the R package "prcomp" on all item-level questions from the PCL-5 and DASS-21 anxiety items for all behavioral subjects (n = 87) to identify factors related specifically to anxiety and PTSD. The resulting factor(s) would serve as our independent variable for anxious and/or PTSD symptomology across both behavioral and fMRI analyses.

**Behavioral analyses.** Behavioral analyses were conducted using the behavioral sample ($n$ = 87) from the study. All subjects with behavioral data following outlier exclusions were used for behavioral analyses. Accuracy (% correct) on Stop trials served as our dependent variable for our behavioral analyses. All continuous variables were mean centered, and the covariates of gender, age, and AUDIT scores were included in these models.

***Primary behavioral analyses.*** Our primary behavioral analyses used the factor(s) identified by our PCA and recent cannabis use group as independent variables. We first conducted Pearson's $r$ correlational analyses between Stop trial accuracy, and the anxiety and PTSD factor(s) resulting from our PCA. Next, we conducted an independent samples $t$-test to

explore whether Stop trial accuracy differences between the group testing positive for recent cannabis use and the group testing negative for recent cannabis use were present. Following this analysis, we employed a frequentist equivalence independent samples *t*-test, based on the two one-sided hypothesis tests from the TOSTER R-package (Lakens, 2017). This was done to examine whether behavioral performance between the two recent cannabis use groups was similar on Stop trials. Specifically, we assumed a small effect size for our equivalence region's lower and upper bound (i.e., Cohen's *d* of -0.2 for lower bound to 0.2 for upper bound) for this test (Lakens et al., 2018). Following these initial examinations, we conducted a multiple linear regression analysis using the factor(s) resulting from our PCA reflecting anxiety and PTSD, recent cannabis use, and the interaction between these factor(s) and recent cannabis use as our independent variables to predict Stop trial accuracy. We included the covariates of gender, age, and AUDIT in this model. All continuous variables were mean centered in our regression models.

***Secondary behavioral analyses***. We performed secondary behavioral analyses using the independent variables of anxiety (i.e., DASS-21 anxiety subscale score) and PTSD (i.e., PCL-5 total score) in combination with recent cannabis use and their interactions in separate analyses. Specifically, we conducted Pearson's *r* correlational analyses examining the association between DASS-21 anxiety subscale scores and PCL-5 total scores with Stop trial accuracy. Two additional multiple linear regression analyses were run to predict Stop trial accuracy with the same covariates mentioned above: one included the DASS-21 anxiety subscale scores and the other included PCL-5 total scores as independent variables and their interactions with recent cannabis use in their respective models.

**fMRI analyses.** All fMRI data were analyzed using a whole-brain approach. First, we computed Go versus Stop trial difference score measures of BOLD activity (% signal change). Specifically, we subtracted Stop trial activity from Go trial activity for correct response trials. Thus, increased activity difference scores would demonstrate greater activity for Go compared to Stop trials, while decreased activity difference scores would reflect greater activity for Stop compared to Go trials in each region. This analysis was conducted across all participants, regardless of recent cannabis use group. As in our behavioral analyses, all continuous variables were mean centered, and the covariates of gender, age, and AUDIT scores were included in these models.

We used the average blur estimates (ACF) across all participants in the fMRI sample ($n$ = 66) to compute cluster thresholds using 3dClustSim. Specifically, this allowed us to control for the probability of false positive clusters. Our results from 3dClustSim when using an alpha of 0.05, a cluster threshold of 0.001, and a False Discovery Rate (FDR) of 0.05 indicated a minimum cluster size of 14 (value of 13.1) for our analyses.

Because we conducted a PCA on the DASS-21 anxiety subscale and PCL-5 total score items to differentiate PTSD and anxious symptomology, we expected that the factor extracted from our PCA reflecting anxiety would positively predict dlPFC, dACC, rIFG, and striatal activity, indicating that individuals reporting greater anxious symptomology engage in greater activity in these regions on Stop compared to Go trials. In contrast, we anticipated that the factor related to PTSD would negatively predict dlPFC, dACC, rIFG, and striatal activity, suggesting that those with greater PTSD recruit these regions to a lesser degree compared to those with lower PTSD on Stop compared to Go trials. In addition, we predicted that individuals categorized as testing positive for recent cannabis use would show increased dlPFC, rIFG, and

striatal activity, but reduced dACC activity compared to those testing negative for recent cannabis user. We did not anticipate observing interactions between recent cannabis use and the factors reflecting anxious symptomology and PTSD.

Given the lack of work examining anxiety and PTSD in combination with cannabis use, it is difficult to make any *a priori* predictions regarding interactions between these independent variables. However, it is because of this paucity of work examining potential interactions in these constructs that this research is necessary to directly test whether such interactions exist in influencing the underlying neural correlates related to motor response inhibition. Such work will inform future research, and provide novel insights into whether anxiety and PTSD interact with recent cannabis use to influence motor response inhibition. Furthermore, due to our primarily main effect hypotheses, it is important to note that we may observe additive effects from the addition of recent cannabis use to our anxiety and PTSD predictions. However, it is possible that additive effects resulting from PTSD and anxiety may be observed in our model containing factor(s) reflecting these constructs.

***Primary fMRI analyses.*** Our primary fMRI analyses were conducted using 3dMVM to predict the dependent variable of BOLD activity (% signal change) between Go and Stop trials. Specifically, we examined the magnitude of activity differences between Stop and Go trials, or the percent BOLD signal change between these conditions, as our dependent variable. Our independent variables in this model included the factor(s) resulting from our PCA reflecting anxiety and PTSD as an independent variable, recent cannabis use, and the interaction(s) between this factor and recent cannabis use to predict percent in BOLD signal change between Stop and Go trials. We included the covariates of gender, age, and AUDIT in this model. All continuous variables were mean centered in our regression models.

***Secondary fMRI analyses.*** We conducted a secondary series of fMRI analyses following the same 3dMVM approach described above. However, in these analyses we replaced the independent variable of the PCA factor(s) with the DASS-21 anxiety subscale and PCL-5 total scores in separate analyses. As in our primary fMRI analyses, all continuous variables were mean centered and the covariates of gender, age, and AUDIT scores were included in these models.

**Exploratory analyses.** Further exploratory behavioral analyses were conducted to examine the influence of our PCA-derived factor(s), anxious symptomology, PTSD, and recent cannabis use on Go trial accuracy and RT. Additional exploratory fMRI analyses were performed to examine how these independent variables influenced BOLD activity on Go and Stop trials independently.

***Primary behavioral exploratory analyses*.** Further exploratory behavioral analyses were performed to examine the associations between anxiety and PTSD factor(s) resulting from our PCA with Go trial accuracy and RT using separate Pearson's *r* correlational analyses. We also conducted two independent samples *t*-tests to explore whether participants testing positive and negative for recent cannabis use scored differently on Go trial accuracy and RT. Next, we conducted two frequentist equivalence independent samples *t*-tests, as described prior, to examine whether scores on these outcomes were similar in both recent cannabis use groups. Following this, two additional multiple linear regression analyses were performed including the factor(s) resulting from our PCA reflecting anxiety and PTSD as an independent variable and the interaction(s) with recent cannabis use to predict Go trial accuracy and RT, respectively. Gender, age, and AUDIT scores were included as covariates in these models.

***Secondary behavioral exploratory analyses.*** A secondary series of exploratory behavioral analyses were conducted to examine the associations between DASS-21 anxiety subscale scores and PCL-5 total scores with Go trial accuracy and RT. Following this, we conducted four multiple linear regression analyses: two examining the dependent variable of Go trial accuracy from the predictors of DASS-21 anxiety subscale scores and PCL-5 total scores and their interactions with recent cannabis use, respectively, and two examining the dependent variable of Go trial RT with these same independent variables.

***Primary fMRI exploratory analyses.*** Our primary fMRI exploratory analyses were conducted to examine the influence of our PCA-derived factor(s) in combination with recent cannabis use in predicting Go and Stop trial signal activity. Specifically, we conducted two 3dMVM models, one for Go trials and one for Stop trials, including the independent variables of the factor(s) resulting from our PCA for anxiety and PTSD, recent cannabis use, the interaction(s) between recent cannabis use and our PCA factor(s), and the covariates of gender, age, and AUDIT scores. All continuous variables were mean centered in our regression models.

***Secondary fMRI exploratory analyses.*** A secondary series of exploratory fMRI analyses were run to examine the influence of the DASS-21 anxiety subscale and PCL-5 total scores in predicting Go and Stop trial activity. Specifically, we conducted four 3dMVM models, two for Go trials and two for Stop trials. In each of these trial types, one model included the DASS-21 anxiety subscale in place of the PCA-derived factor(s), and the other model replaced the PCA factor(s) score with PCL-5 total scores.

## Results

### Data Reduction

Our PCA using the R package "paran" on all item-level questions from the PCL-5 and DASS-21 anxiety items from the full behavioral sample ($n$ = 87) revealed a single retained component with an adjusted eigenvalue of 10.719. Further examination of our PCA using the R package "prcomp" revealed that 27 factors were identified (Table 6), and the retained component accounted for the majority of variance (44.16%). All items from the DASS-21 anxiety subscale and PCL-5 loaded onto this primary component.

We conducted a second series of analyses using the subset of participants to be used for fMRI data ($n$ = 66) to verify if this pattern was consistent across our samples. These results also resulted in one retained component with an adjusted eigenvalue of 10.667 that accounted for approximately 44.79% of variability (Appendix D, Table 1). As in the behavioral sample, all items from our measures loaded onto this primary factor.

Given these results, we chose to create a composite score from the DASS-21 anxiety subscale and PCL-5 items reflecting this component, which we have named general distress. This composite score was created using a simple averaging approach (Song et al., 2013), in which we created individual z scores for each participant's DASS-21 anxiety subscale and PCL-5 total scores, and added these scores together for each participant. Importantly, general distress scores did not differ between the recent negative and positive cannabis use groups in the behavioral sample, $t(85) = 0.326$, $p = 0.745$, $d = 0.070$, $BF_{10} = 0.236$ (Appendix A, Figure 4A), or fMRI sample, $t(64) = 0.663$, $p = 0.510$, $d = 0.163$, $BF_{10} = 0.304$ (Appendix A, Figure 4B). Thus, all primary analyses included this general distress measure as an independent variable for our PCA-derived component.

**Behavioral Outcomes**

**Primary behavioral outcomes.** Overall performance on Stop trials was high (*M* = 99.47%, *SE* = 0.13), with the lowest accuracy being 94.23%. Results from our Pearson's *r* correlation revealed a non-significant association between Stop trial accuracy and general distress, $r(85) = -0.007$, $p = 0.948$, $BF_{10} = 0.134$ (Figure 6A). Our independent samples *t*-test also found non-significant differences in Stop trial accuracy between the recent cannabis use groups, $t(63.29) = 1.469$, $p = 0.147$, $d = 0.070$, $BF_{10} = 0.332$ (Figure 6B). However, our frequentist equivalence independent samples *t*-test for Stop trial accuracy yielded a non-significant result, $t(85) = 1.535$, $p = 0.128$. In addition, our 90% confidence interval (CI = -0.027 to 0.689) extended beyond the upper bound of our equivalence region (Cohen's $d = 0.2$), suggesting that there may be important differences between the recent cannabis use groups, such that the group testing negative for recent cannabis use performed better compared to the group testing positive for recent cannabis use. However, there is insufficient data to draw strong conclusions at this point.

Our multiple linear regression analysis predicting Stop trial accuracy with our predictor variables (i.e., general distress, recent cannabis use, their interaction) and our covariate variables (i.e., gender, age, and AUDIT scores) was non-significant, $F(6, 80) = 0.963$, $p = 0.456$, $R^2 = 0.067$ $BF_{10} = 0.058$ (Table 7). The interaction between general distress and recent cannabis use was also non-significant, $F(1, 80) < 0.001$, $p = 0.979$, $R^2$ change $< 0.001$, $BF_{10} = 0.476$ (Figure 7). These results suggest that motor response inhibition behavioral performance was not influenced by the degree of general distress, recent cannabis use, and their interaction.

**Secondary behavioral outcomes.** Similar to the correlation results from general distress, our secondary analyses also indicated non-significant associations between Stop trial accuracy and DASS-21 anxiety subscale, $r(85) = -0.046$, $p = 0.673$, $BF_{10} = 0.146$ (Figure 8A), and PCL-5

total, $r(85) = 0.033$, $p = 0.764$, $BF_{10} = 0.140$ (Figure 8B) scores. The multiple linear regression model using scores from the DASS-21 anxiety subscale did not significantly predict Stop trial accuracy, $F(6, 80) = 1.073$, $p = 0.386$, $R^2 = 0.075$, $BF_{10} = 0.073$ (Table 8), and the interaction between this measure and recent cannabis use was null, $F(1, 80) = 0.006$, $p = 0.940$, $R^2$ change < 0.001, $BF_{10} = 0.473$ (Figure 9A).

Our second regression model using PCL-5 total scores in place of DASS-21 anxiety subscale scores was also null, $F(6, 80) = 0.916$, $p = 0.488$, $R^2 = 0.064$, $BF_{10} = 0.052$ (Table 9), and the interaction between the PCL-5 total and recent cannabis use was also non-significant, $F(1, 80) = 0.001$, $p = 0.971$, $R^2$ change < 0.001, $BF_{10} = 0.478$ (Figure 9B). Taking the primary and secondary results together, motor response inhibition behavioral performance was not associated with anxious symptomology, PTSD, their common factor of general distress, recent cannabis use, and the interactions between recent cannabis use and these independent variables.

Our behavioral results suggest that motor response inhibitory performance is not influenced by our common PCA-derived factor of general distress, anxious symptomology, PTSD, or recent cannabis use. In addition, recent cannabis use did not interact with general distress, anxiety, or PTSD to predict behavioral estimates of motor response inhibition.

**fMRI Outcomes**

**Primary fMRI outcomes.** Results from our 3dMVM analysis predicting BOLD activation (% change) between Go and Stop trials using the predictor variables (i.e., general distress, recent cannabis use, their interaction) and our covariate variables (i.e., gender, age, and AUDIT scores) was non-significant. For exploratory purposes, we applied a more liberal whole-brain voxel wise threshold of 0.01 (Appendix E). However, we did not report statistical outcomes when using this threshold due to these analyses being for more exploratory purposes.

**Secondary fMRI outcomes.** We conducted a secondary analysis using 3dMVM, but replaced the general distress factor with DASS-21 anxiety subscale scores. The outcome of this analyses also showed no significant differences in BOLD activation (% change) between Go and Stop trials. Similar to our primary analyses, we also analyzed these models using a liberal whole-brain voxel wise threshold of 0.01 (Appendix F).

Finally, we conducted a third 3dMVM, this time replacing the general distress factor with PCL-5 total scores, which resulted in non-significant effects in predicting BOLD activation (% change) between Go and Stop trials. An additional analysis with a liberal whole-brain voxel wise threshold of 0.01 was also examined for using this model (Appendix G).

Based on these outcomes, neural recruitment necessary for the successful completion of motor response inhibition was not associated with general distress, anxiety, PTSD, or recent cannabis use. Furthermore, recent cannabis use did not interact with general distress, anxious symptomology, or PTSD to influence neural activity necessary for motor response inhibition.

## Exploratory Outcomes

**Behavioral exploratory outcomes.** In addition to examining behavioral indicators of motor response inhibition through Stop trial accuracy, we also conducted analyses concerning the dependent variables of Go trial accuracy and RT.

***Primary behavioral exploratory outcomes.*** Go trial accuracy was also relatively high in our sample ($M$ = 95.16%, $SE$ = 0.78). Our Pearson's $r$ correlation analyses indicated null associations between general distress and Go trial accuracy, $r(85) = -0.001$, $p = 0.991$, $BF_{10} = 0.134$ (Figure 10A), and Go trial RT, $r(85) = -0.048$, $p = 0.656$, $BF_{10} = 0.148$ (Figure 10B). Results from our independent samples $t$-tests also indicated no significant differences in Go trial accuracy, $t(85) = 0.986$, $p = 0.327$, $d = 0.213$, $BF_{10} = 0.345$ (Figure 11A) and Go trial RT, $t(85) =$

0.039, $p = 0.969$, $d = 0.009$, $BF_{10} = 0.226$ (Figure 11B) between negative and positive recent cannabis use groups. Our frequentist equivalence independent samples *t*-test for Go trial accuracy also yielded a non-significant result, $t(85) = 0.986$, $p = 0.327$. Our 90% confidence interval (CI = -0.144 to 0.569) extended beyond the upper bound of our equivalence region (Cohen's $d = 0.2$). This indicates that, although we cannot reject the non-equivalence hypothesis, there may be potential differences between the recent cannabis groups in terms of Go trial accuracy, with individuals in the negative recent cannabis use group potentially performing better than those in the positive recent cannabis use group. However, there is insufficient data to draw strong conclusions regarding the possibility, or lack of, group differences in this data. Similar outcomes from the frequentist equivalence independent samples *t*-test for Go trial RT were observed, $t(85) = 0.039$, $p = 0.969$, with our 90% confidence interval (CI = -0.347 to 0.364) extending beyond the both the lower and upper bound of our equivalence region (Cohen's $d$ = +/- 0.2). This suggests that there may be differences between recent cannabis use groups in terms of RT, but the potential directionality of these differences is unable to be speculated upon given that both the upper and lower equivalence region bounds were passed by our confidence intervals. Ultimately, we do not have sufficient data to make strong conclusions regarding group differences in our data.

Our multiple linear regression predicting Go trial accuracy from the predictor variables of general distress, recent cannabis use, their interaction, and the covariates of gender, age, and AUDIT scores was non-significant, $F(6, 80) = 0.539$, $p = 0.777$, $R^2 = 0.039$, $BF_{10} = 0.021$ (Table 10), and the interaction between general distress and recent cannabis use was null, $F(1, 80) = 0.074$, $p = 0.787$, $R^2$ change = 0.001, $BF_{10} = 0.507$ (Figure 12A). Similar null outcomes were observed for our regression using the same predictor and covariate variables to predict Go trial

RT, $F(6, 80) = 1.885$, $p = 0.094$, $R^2 = 0.124$, $BF_{10} = 0.023$ (Table 11), and the interaction between general distress and recent cannabis use was non-significant, $F(1, 80) = 0.144$, $p = 0.705$, $R^2$ change = 0.002, $BF_{10} = 0.470$ (Figure 12B). These results suggest that Go trial performance was not influenced by general distress, recent cannabis use, or their interaction.

***Secondary behavioral exploratory outcomes.*** There were no significant associations between Go accuracy and the DASS-21 anxiety subscale, $r(85) = -0.018$, $p = 0.868$, $BF_{10} = 0.136$ (Figure 13A), and PCL-5 total, $r(85) = 0.016$, $p = 0.884$, $BF_{10} = 0.135$ (Figure 13B) scores, indicated by our Pearson's *r* correlations. Similar null associations between Go trial RT and the DASS-21 anxiety subscale, $r(85) = -0.014$, $p = 0.898$, $BF_{10} = 0.135$ (Figure 14A), and PCL-5 total, $r(85) = -0.075$, $p = 0.490$, $BF_{10} = 0.169$ (Figure 14B) scores were observed.

We conducted a multiple linear regression predicting Go trial accuracy using the same model described above, only replacing general distress scores with scores on the DASS-21 anxiety subscale. Results from this model indicated non-significant outcomes, $F(6, 80) = 0.573$, $p = 0.751$, $R^2 = 0.041$, $BF_{10} = 0.023$ (Table 12). The interaction between DASS-21 anxiety subscale score and recent cannabis use was also null, $F(1, 80) = 0.103$, $p = 0.749$, $R^2$ change = 0.001, $BF_{10} = 0.512$ (Figure 15A). Next, we conducted a multiple regression model including the same predictors and covariates as above, but replaced DASS-21 anxiety subscale scores with PCL-5 total scores, to predict Go trial accuracy. Our regression results for this model were also null, $F(6, 80) = 0.510$, $p = 0.799$, $R^2 = 0.037$, $BF_{10} = 0.020$ (Table 13), and the interaction between recent cannabis use and PCL-5 total score was non-significant, $F(1, 80) = 0.024$, $p = 0.877$, $R^2$ change < 0.001, $BF_{10} = 0.498$ (Figure 15A).

Go trial RT was not significantly predicted by our multiple regression model including the predictor variables of DASS-21 anxiety subscale scores, recent cannabis use, their

interaction, and the covariates of gender, age and ADUIT scores, $F(6, 80) = 2.009$, $p = 0.074$, $R^2 = 0.131$, $BF_{10} = 0.024$ (Table 14). The interaction between DASS-21 anxiety subscale score and recent cannabis use was also null, $F(1, 80) = 0.735$, $p = 0.394$, $R^2$ change = 0.008, $BF_{10} = 0.594$ (Figure 16A). We conducted a second multiple regression model including the same covariates and predictor variables, only we used PCL-5 total scores instead of DASS-21 anxiety subscale scores, to predict Go trial RT. The results of this model were non-significant, $F(6, 80) = 1.866$, $p = 0.097$, $R^2 = 0.123$, $BF_{10} = 0.023$ (Table 15), including the interaction between PCL-5 total score and recent cannabis use, $F(1, 80) = 0.009$, $p = 0.925$, $R^2$ change < 0.001, $BF_{10} = 0.445$ (Figure 16B).

These exploratory behavioral outcomes indicate that Go trial behavioral performance was not influenced by individual differences in general distress, anxious symptomology, PTSD, or recent cannabis use. In addition, recent cannabis use does not interact with general distress, anxiety, or PTSD in predicting Go trial performance.

**fMRI exploratory outcomes.** We conducted additional 3dMVMs to examine signal BOLD activity (% change) during both Go and Stop trials in isolation. Similar to our primary analyses, these models included the predictor variables of either general distress, DASS-21 anxiety subscale scores, or PCL-5 total scores, along with recent cannabis use, and the respective interaction terms with the first predictor variable (i.e., general distress, DASS-21 anxiety subscale scores, or PCL-5 total scores). The covariates of gender, age, and AUDIT scores were also included in each of these models.

***Primary fMRI exploratory outcomes.*** Our 3dMVM examining Stop trial BOLD activity (% change) from the predictors of general distress, recent cannabis use, their interaction, and the covariates of gender, age, and AUDIT scores revealed a non-significant model. Similar null

outcomes were observed when using these predictors for BOLD activity (% change) on Go trials. Because these analyses were exploratory, we did not examine Stop trial BOLD activity (% change) using a more liberal threshold ($p < 0.01$).

***Secondary fMRI exploratory outcomes.*** Similar to our 3dMVM predicting Stop trial BOLD (% change) activity, our model using the same predictor variables and covariates, but replacing general distress with DASS-21 anxiety subscale scores, was also non-significant. Similar null outcomes were observed in our 3dMVM when we replaced DASS-21 anxiety subscale scores with PCL-5 total scores to predict Stop trial BOLD activity (% change). Stop trial BOLD activity (% change) was not examined at a liberal threshold ($p < 0.01$) as was done in our primary analyses.

We conducted a 3dMVM using the predictor variables of DASS-21 anxiety subscale scores, recent cannabis use, their interaction, and the covariates of age, gender, and AUDIT scores to predict Go trial BOLD activity (% change). These results were non-significant, similar to the outcomes observed when using general distress as a predictor variable. Finally, we conducted a 3dMVM using the same predictor variables, but replacing DASS-21 anxiety subscale scores with PCL-5 total scores, and covariates to predict Go trial BOLD activity (% change). The outcome of this analysis was also null. Given that these fMRI analyses were purely exploratory, we did not conduct these analyses using a liberal threshold ($p < 0.01$) as we had in our primary fMRI analyses.

These exploratory analyses indicated that BOLD activity (% change) for both Stop and Go trials in isolation was not influenced by our PCA-derived factor of general distress, anxiety, PTSD, or recent cannabis use. There was also a non-significant interaction between recent

cannabis use and general distress, anxious symptomology, and PTSD in predicting BOLD activity (% change) for these trials.

## Discussion

Given the high comorbidity rate between anxiety disorders and cannabis use (Buckner & Carroll, 2010; Butler, 2019; Crippa et al., 2009; Dorard et al., 2008; Teesson et al., 2012; Young-Wolff et al., 2020), the current study aimed to examine how recent cannabis use and dispositional anxiety interact to influence behavioral performance on motor response inhibition tasks, and the underlying neural activity reflecting this interaction. In addition, because the sample used for the current study recently experienced a traumatic injury, which has been shown to negatively influence motor response inhibition behavioral performance (van der Bij et al., 2020; van Rooij et al., 2018) and we also examined how PTSD following this traumatic event interacts with recent cannabis use to predict motor response inhibition. Given the overlap between anxious symptomology and PTSD, we created a composite PCA-derived factor encompassing both of these measures, which we coined general distress.

### Behavioral Findings

We found no association between Stop trial accuracy and general distress. To our knowledge, no other study has examined the relationship between motor response inhibition and a single factor encompassing both anxiety and PTSD measures. Given our results, and the novelty of this approach, this suggests that individuals endorsing symptomology reflective of both anxiety and PTSD perform to the same degree as their peers scoring lower in this composite construct. It is possible that we obtained a non-significant association between these constructs due to the overlap of anxiety and PTSD in this general distress variable. For example, although prior work has identified behavioral deficits in individuals with PTSD (Swick et al, 2012; Wu et

al., 2010) and those who have experienced a traumatic event (van der Bij et al., 2020), others have found that individuals with elevated trait anxiety (Karch et al., 2008; Oosterlaan & Sergeant, 1996) and those with clinical anxiety disorders (Grillon et al., 2017b, Hallion et al., 2017; Herrmann et al., 2003; Kim et al., 2007; Leonard & Abramovitch, 2019) perform to the same degree as healthy controls and less anxious peers on tasks assessing motor response inhibition. Because our approach for examining anxious symptomology and PTSD under one common factor is novel, additional replication for these results is warranted. Our results indicate that motor response inhibition is not related to the combination of PTSD and anxiety.

To further explore the influence of anxious symptomology and PTSD independently, we examined Stop trial accuracy associations with anxiety and PTSD separately. Similar to our non-significant outcomes for general distress, we also observed non-significant correlations between Stop trial accuracy and anxious symptomology and PTSD. Although our anxiety findings are consistent with previous literature examining motor response inhibition in anxious participants (Grillon et al., 2017b, Hallion et al., 2017; Herrmann et al., 2003; Karch et al., 2008; Kim et al., 2007; Leonard & Abramovitch, 2019; Oosterlaan & Sergeant, 1996), the PTSD outcomes contradict previous reports demonstrating negative behavioral performance in those with PTSD and that had experienced trauma (Swick et al, 2012; Wu et al., 2010; van der Bij et al., 2020; van Rooij et al., 2018). Importantly, much of this previous work specifically examined combat veterans diagnosed with PTSD who had also experienced a mild traumatic brain injury from blast explosions (Swick et al, 2012), and individuals diagnosed with PTSD following a natural disaster (Wu et al., 2010). In contrast, our study used PCL-5 scores as a continuous measure of PTSD symptom severity instead of a clinical diagnosis. Although this approach allows for the assessment of individual variability in PTSD, most of our participants (58.60%) scored below the

standard cut off score of 31 on the PCL-5, which is often used as a criterion for provisional diagnosis of PTSD (Blevins et al., 2015; Bovin et al., 2016). This suggests that many of our participants did not endorse severe levels of PTSD, potentially limiting the variability of this construct and preventing us from observing behavioral deficits in individuals with higher PCL-5 scores. Thus, it may be the case that studies consisting of samples including individuals who meet clinical diagnosis for PTSD or with higher severity PCL-5 scores are more likely to demonstrate behavioral deficits in motor response inhibition. Alternatively, age and the specific type of trauma that this sample experienced may differentially impact motor response inhibition. For instance, the meta-analysis from van der Bij and colleagues (2020) that demonstrated behavioral deficits in motor response inhibition included studies consisting of a variety of forms of trauma, such as sexual abuse, childhood maltreatment, and those who experienced natural disaster events. In contrast, a majority of our participants had experienced a motor vehicle accident (67.80%). Furthermore, the studies selected in van der Bij and colleagues (2020) only included participants ages 25 or below, while our sample mostly consisted of individuals above the age of 25 (64.70%). Thus, it is possible that the specific type of trauma experienced plays a critical role in the association between PTSD and motor response inhibition, with younger individuals being more likely to demonstrate behavioral deficits. Taken together, our results suggest that individuals endorsing greater anxious and posttraumatic stress symptomology perform to the same degree as their peers reporting less anxious symptomology.

We also found non-significant group differences in Stop trial accuracy between those testing positive and those testing negative for recent cannabis use. Further outcomes from our equivalence testing suggested that these groups performed similarly. However, given the examination of confididence intervals from this latter analysis, our confidence for the outcomes

of these equivalence tests is not high. Nonetheless, our finding is consistent with previous reports demonstrating non-significant behavioral differences in motor response inhibition, albeit in regular cannabis users (Filbey & Yezhuvath, 2013; Gonzalez et al., 2012; Hester et al., 2009; Smith et al., 2011; Smith et al., 2014; Tapert et al., 2007; Wallace et al., 2020), but contradicts others who have examined this relationship in regular cannabis users (Behan et al., 2014; Bolla et al., 2002). It should be noted that our measure of recent cannabis use through urine toxicology holds several limitations due to the sensitivity of this test. For instance, although our urine analyses were mostly consistent with participants' self-reported cannabis use, this measure does not allow us to disentangle the frequency of use within the recent time interval (i.e., < 3 weeks). This is critical given that many reports demonstrating behavioral deficits in motor response inhibition involved samples where participants had engaged in chronic or heavy cannabis use (Behan et al., 2014; Bolla et al., 2002). As such, it may be the case that individuals testing positive for recent cannabis use may not have engaged in heavy use, reducing potential deficits in behavioral performance in this group. Overall, our findings suggest that individuals who engaged in recent cannabis use, as determined via urine toxicology, performed to the same degree as those who did not on motor response inhibition, at least when this is measured with a relatively non-difficult Stop-Signal task.

We did not observe any interaction effects between recent cannabis use and general distress. In addition, recent cannabis use did not interact with the specific factors of anxious symptomology or PTSD to predict motor response inhibition behavioral performance. These findings are consistent with others (Spechler et al., 2020) who examined diagnosed comorbid cannabis dependency and anxiety disorders, and found no differences in task performance. In addition, Borgwardt and colleagues (2008) reported non-significant motor response inhibition

behavioral effects in individuals with elevated state anxiety following acute cannabis administration. Although we specifically focused on recent cannabis use and anxious symptomology, the lack of an interaction effect between these variables is consistent with these prior reports. Our results also failed to identify a significant interaction between recent cannabis use and PTSD, measured using PCL-5 scores. While many of the limitations noted above are applicable to our interpretations of these results, these patterns of findings suggest that recent cannabis use does not interact with anxiety, PTSD, or their common factor of general distress to influence motor response inhibition.

Overall, our behavioral results suggest that motor response inhibition performance remains intact regardless of the degree of anxious symptomology and PTSD. In addition, although we did not observe significant behavioral differences between individauls testing positive compared to those testing negative for recent cannabis use, our equivalence test results do not exclude the possibility that there are not meaningful differences between these groups. Others have also reported non-significant behavioral differences in motor response inhibition performance, but observed altered neural activity between regular cannabis users and non-cannabis users (Borgwardt et al., 2008; Filbey & Yezhuvath, 2013; Hester et al., 2009; Smith et al., 2011; Tapert et al., 2007; Wallace et al., 2020) and anxious compared to non-anxious individuals (Aarts & Pourtois, 2010; Berggren & Derakshan, 2013; Derakshan & Eysenck, 2009; Eysenck & Derakshan, 2011; Eysenck et al., 2007; Forster et al., 2015; Righi et al., 2009; Ruchsow et al., 2007; Savostyanov et al., 2009; Sehlmeyer et al., 2010; Torrisi et al., 2016). This suggests that individuals engaging in cannabis use and those with high anxiety may recruit neural networks necessary for the completion of motor response inhibition tasks to a greater degree in order to perform as well as their non-using cannabis and less anxious peers. In addition,

differential neural activity between those with PTSD and healthy controls have also been reported (Carrion et al., 2008; Falconer et al., 2008; Jovanovic et al., 2013). Therefore, we investigated potential differences in neural activity during completion of our Stop-Signal task to identify if these null behavioral results were accounted for by altered underlying brain mechanisms.

**fMRI Findings**

Contrary to our hypotheses, we failed to identify significant changes in BOLD signal between Go and Stop trials in the dlPFC, dACC, rIFG, and striatum. More so, our whole brain analyses did not reveal any significant changes in BOLD activity between these conditions in any brain regions as a function of our PCA-derived factor of general distress. These results suggest that individuals endorsing symptomology common to PTSD and anxiety do not recruit neural regions differently than those with a lower degree of these symptomologies when completing a motor response inhibition task. This non-significant result may be due to the convergence of both PTSD and anxious symptomology present in our general distress factor eliminating differential recruitment of brain regions necessary for the completion of our Stop-Signal task. For example, although prior work has identified enhanced recruitment of the striatum, rIFG (Karch et al., 2008; although see Forster et al., 2015), and putamen (Torrisi et al., 2016) in high trait anxious individuals, those diagnosed with PTSD have demonstrated decreased activity in frontal regions, such as the anterior cingulate cortex and dlPFC (Carrion et al., 2008; Falconer et al., 2013; Stevens et al., 2016), and the striatum (Falconer et al., 2013) compared to healthy controls. Therefore, it is possible that these differences in activity cancelled out due to the inclusion of both anxious symptomology and PTSD, resulting in similar neural activity as that observed in individuals with lesser degrees of anxiety and PTSD.

Similar to our behavioral analyses, we attempted to parse these differential effects by examining BOLD activity change between Go and Stop trials in PTSD and anxious symptomology independently. We failed to identify any significant activity changes in brain regions based on anxious symptomology. Although this finding is inconsistent with previous reports examining trait anxiety (Karch et al., 2008; Forster et al., 2015), several methodological considerations should be considered. First, Karch and colleagues (2008) used a median split to classify individuals differing in high versus low trait anxiety, and found differential neural recruitment in individuals who reported high trait anxiety and were categorized as alcohol-dependent patients. Second, Forster and colleagues (2016) task included a control block consisting of only Go trials along with an experimental condition that included both Go and Stop trials (~7% of trials in this condition were Stop trials), and a mask was inserted between the Go and Stop cues. In addition, their sample was relatively small (i.e., $n = 18$) compared to our current study (i.e., $n = 66$), which may indicate that their study was underpowered and may have committed a Type I error. Third, although trait anxiety measured using the State-Trait Anxiety Inventory (Spielberger & Gorsuch, 1983) was used in these previous reports (Karch et al., 2008; Forster et al., 2015), evidence exists to suggest that this measure may more closely reflect general negative affect than anxiety specifically (Bados et al., 2010; Beiling et al., 1998; Grös et al., 2007). Thus, it may be the case that some of the variance in these previous studies reflect the influence of other negative factors, such as depression, in addition to anxiety. In addition, given that this is the first study to our knowledge that has examined the influence of anxious symptomology on a Stop-Signal task using the DASS-21 anxiety subscale, it may be the case that this measure does not produce the same outcomes as others, such as the State-Trait Anxiety Inventory (Spielbeger & Gorsuch, 1983). Overall, our results indicate that anxious

symptomology, measured using the DASS-21 anxiety subscale and controlling for factors including age, gender, and alcohol use, is not associated with differential neural recruitment between Go and Stop trials on a motor response inhibition task.

Our examination of PTSD independent of anxious symptomology also resulted in non-significant changes in BOLD activity changes between Go and Stop trials, suggesting individuals with elevated PTSD demonstrate similar neural recruitment during completion of a motor response inhibition task as those with a lesser degree of PTSD. Although these outcomes contradict previous studies (Carrion et al., 2008; Falconer et al., 2013; Jovanovic et al., 2013; Stevens et al., 2016), it is important to note that our sample consisted of individuals that had recently experienced a traumatic injury, while much of the current work examining response inhibition and PTSD focused on patients who had been diagnosed with PTSD for a longer interval. To our knowledge, no other study has used fMRI to examine underlying neural recruitment during completion of a Stop-Signal task in a sample that had very recently experienced a traumatic event. It may be the case that behavioral and neural motor response inhibition alterations in individuals with PTSD are not present immediately following a traumatic event, and instead become prevalent at a later timepoint, perhaps even months following this trauma. This idea is supported by prior work demonstrating heterogeneous outcomes for PTSD following an acute traumatic event, in that many of the negative consequences associated with PTSD can become amplified months after this experience occurred (Benyamini & Solomon, 2005; Bliese et al., 2005; Carty et al., 2006; Dekel et al., 2013; deRoon-Cassini et al., 2010; Grieger et al., 2006; Milliken et al., 2007; Orcutt et al., 2004; Solomon & Mikulincer, 2006; Southwick et al., 2000). In many cases, individuals with enhanced PTSD following a traumatic event will often report greater PTSD severity months following this trauma (Orcutt et al., 2004;

Solomon & Mikulincer, 2006), while others have observed a delayed onset for the development of PTSD following an acute traumatic event (Cart et al., 2006; deRoon-Cassini et al., 2010; Grieger et al., 2006; Milliken et al., 2007; Orcutt et al., 2004; Solomon & Mikulincer, 2006). Stress experienced following a traumatic event is known to influence various brain regions necessary for cognitive functioning (e.g., medial prefrontal cortex, hippocampus, etc.), and can dysregulate the endocrine system, specifically involving glucocorticoids (Bremner, 2006; McFarlane, 2015). However, the degree of these alterations can depend on the duration that one experiences stress. For example, animal model research has demonstrated differential effects on gene expression (Datson et al., 2013; Gray et al., 2018; Tsankova et al., 2007), glucocorticoid (Finsterwald & Alberini, 2014; Mizoguchi et al., 2003; McEwen, 2017; Popli et al., 2012; Vyas et al., 2016), and excitatory neurotransmitter activity (Lowy et al., 1993; Peterlik et al., 2016; Popoli et al., 2012) following acute versus chronic periods of stress. Specifically, acute stress yields enhanced glutamatergic transmission within prefrontal brain regions known to contribute to successful response inhibition (Aron & Poldrack, 2005; Boehler et al., 2010; Chevrier et al., 2004; Floden & Stuss, 2006; Kelly et al., 2004; Li et al., 2008; Neo et al., 2011; Rubia et al., 2003; Wager et al., 2005). However, following chronic periods of stress, this amino acid's activity is greatly reduced, and the negative feedback system for glucocorticoids becomes disrupted yielding prolonged enhanced glucocorticoid levels throughout the brain (Finsterwald & Alberini, 2014; Mizoguchi et al., 2003; McEwen, 2017; Popli et al., 2012; Vyas et al., 2016). Evidence has indicated that reduced glutamatergic activity is associated with impairments in cognitive control (Falkenerg et al., 2012; Jett et al., 2017; Naaijen et al., 2018), and prolonged elevation of glucocorticoids is associated with a broad domain of cognitive deficits (Erickson et al., 2003; Paul et al., 2015; Sapolsky, 2000). Thus, individuals who endorse PTSD or stress

during this acute period following their traumatic event may not demonstrate deficits, but may experience impairements at a later time due to the effects of experiencing chronic stress and PTSD. Ultimately, our results indicate that PTSD severity in individuals that had recently experienced a traumatic event is not associated with changes in brain activity between Go and Stop trials on a motor response inhibition task.

Recent cannabis use did not significantly predict differences in BOLD activity change between Go and Stop trials. Although prior work has shown that regular, or chronic, cannabis users demonstrate increased dlPFC, rIFG, and striatum activity (Behan et al., 2014; Smith et al., 2011; Tapert et al., 2007; Wallace et al., 2020; Yanes et al., 2018), but reduced dACC activity (Hester et al., 2009; Yanes et al., 2018) during trials require motor response inhibition, it is important to note that our sample consisted of individuals who classified as recent users or non-users of cannabis. Specifically, we measured recent cannabis use through urine toxicology, which does not allow us to parse the frequency of use during a recent time interval (i.e., < 3 weeks) and when during this interval cannabis was used. In contrast, a majority of neuroimaging work examining the influence of cannabis use on motor response inhibition has consisted of samples that were adolescents being treated for cannabis dependency (Behan et al., 2014), adolescents who regularly engaged in cannabis use but were abstinent for 3-4 weeks prior to fMRI scanning (Tapert et al., 2007; Wallace et al., 2020), and individuals reporting current regular cannabis use (Hester et al., 2009; Smith et al., 2011). Thus, it is possible that the discrepancies in our outcomes compared to these prior reports is the result of our study's sample characteristics (i.e., individuals who recently experienced trauma) and our measure of recent cannabis use though urine toxicology (Yanes et al., 2018). In addition, the positive results from our urine toxicology for measuring recent cannabis users may have reflected residual

intoxication from cannabis use, which would yield differential influences on bran activity compared to those who tested positive but did not experience subacute effects (Balodis & Potenza, 2015; Yanes et al., 2018). Taken together, our results indicate that recent cannabis users who recently experienced a traumatic event do not differentially recruit brain regions compared to those who have not recently engaged in cannabis use when completing a motor response inhibition task.

Our neuroimaging findings revealed non-significant interactions between recent cannabis use and general distress, anxious symptomology, and PTSD. It is important to consider that this is the first study, to our knowledge, that has investigated the interaction between recent cannabis use and a PCA-derived factor of general distress, encompassing anxious symptomology and PTSD, in predicting neural correlates associated with successful motor response inhibition. Our outcomes indicate that general distress does not interact with recent cannabis use to predict differential brain activity in regions necessary for the completion of our Stop-Signal task. Our non-significant interaction effect between anxious symptomology and recent cannabis use replicated a previous report that also observed null neural differences during a Stop-Signal task between individuals with anxiety disorders and comorbid cannabis use problems, those with anxiety disorders without cannabis problems, and healthy controls (Spechler et al., 2020). Finally, we also failed to observe a significant interaction between recent cannabis use and PTSD in predicting neural activity differences in our Stop-Signal task. Although it is important to note that our measure of PTSD consisted of PCL-5 total scores, and our sample had a somewhat restricted range of scores on this measure, with a majority scoring below standard cut off scores used for PTSD (Blevins et al., 2015; Bovin et al., 2016). Nonetheless, these outcomes suggest

that recent cannabis use does not interact with general distress, anxiety, or PTSD to predict differences in neural activity during the completion of a motor response inhibition task.

**Limitations**

Although some limitations in our study design have already been addressed during our discussion of the current study's non-significant outcomes, it is important to consider other potential methodological shortcomings. First, the performance on our task to assess motor response inhibition was high, suggesting that the task was not difficult.. Specifically, the lowest Stop trial accuracy from our sample was 94.24%, with nearly all participant (80.50%) performing perfectly (i.e., 100% accuracy) on these trials. Therefore, it is possible that these ceiling performance effects resulted in reduced variability to detect potential differences in motor response inhibition behavioral performance across our independent variables. In addition, given that such high ceiling effects were observed, it is possible that the task did not require significant engagement to trigger the expected neural responses for Stop compared to Go trials. This may have also resulted in our inability to observe potential differences across our ndependent variables in our neural outcomes.

Second, our sample had previously experienced motor vehicle injuries, which may have resulted in a majority of our participants (77.17%) experiencing mild traumatic brain injury. Given that previous work has found that such injuries influence the neural outcomes associated with motor response inhibition (Dimoska-DiMarco et al., 2011; Fischer et al., 2014; Korgaonkar et al., 2021; Krivitzky et al., 2011; Shen et al., 2020), it is possible that the variability in our sample was influenced by potential mild traumatic brain injury, resulting in non-significant effects across our independent variables of interest.

Third, it is critical to consider the context of our sample in the current study. Specifically, these were individuals who had recently (i.e., two weeks prior to the study) experienced a traumatic event. Given this acute interval, and our measure of recent cannabis use through urine toxicology, it is impossible to ascertain whether individuals who tested positive for cannabis use had regularly engaged in these behaviors prior to the traumatic event, or if they began to use cannabis following this period. As such, the effects of cannabis use following a traumatic event for an acute time period may not influence behavioral or neural activity in a similar manner as reported in previous studies that simply examined these outcomes in regular cannabis users, those who had abstained from such use for several weeks, or adolescent populations. Therefore, our results are unlikely to generalize these prior cannabis use findings given the unique context and characteristics of our sample.

Fourth, although our study included self-reported questionnaires to assess for average cannabis use, we did not assess for when participants had last engaged in cannabis use. This is critical, as participants testing positive for recent cannabis use may have engaged in these behaviors shortly before the session, or maintained residual metabolites upon entering the session that influenced our dependent variables (Balodis & Potenza, 2015; Yanes et al., 2018). In addition, by relying only on urine toxicology as a measure for cannabis use, which has several drawbacks noted prior, we may have falsely grouped participants as recent cannabis users based on potential false positives or vice versa.

Finally, in line with our second limitation, our sample is extremely complex compared to many of the previous work used to inform our hypotheses. Although we attempted to control for the confounding influence of many of the factors present in our sample, such as age, gender, and alcohol use, it is impossible to rule out that our results were not influenced by other individual

variables. For example, while motor vehicle injuries were the common trauma experienced by our sample, other traumatic events were experienced by the remaining participants, such gun shots (*n* = 1, 1.10%), stabbings (*n* = 2, 2.30%), falling (*n* = 2, 2.30%), being struck as a pedestrian (*n* = 4, 4.6%), crash injuries (*n* = 3, 3.40%) domestic violence (*n* = 1, 1.10%), assault (*n* = 10, 11.50%), or other (*n* = 4, 4.60%). In addition, the perceived severity of the trauma experienced varied across our sample. Although these reports were missing for nearly half of our sample (*n* = 38, 43.70%), those who did complete them (*n* = 49) reported their traumatic event has being mild (*n* = 13, 26.50%), moderate (*n* = 20, 40.80%), severe (*n* = 13, 26.50%), or very severe (*n* = 3, 6.10%). Furthermore, some participants also tested positive for other illegal drugs (e.g., amphetamine, cocaine, etc.), which may have influenced our outcomes. Overall, our sample contained several confounding variables that may have altered the variability in our analyses, preventing us from parsing the effects of general distress, anxiety, PTSD, and recent cannabis use on motor response inhibition.

**Future Directions**

Despite these limitations, we believe our study addressed several important questions pertaining to the relationships between motor response inhibition, anxiety, PTSD, and recent cannabis use. Because cannabis use is strongly associated with anxiety (Buckner & Carroll, 2010; Butler, 2019; Crippa et al., 2009; Dorard et al., 2008; Teesson et al., 2012; Young-Wolff et al., 2020) and PTSD (Cornelius et al., 2010; Cougle et al., 2011; Kevorkian et al., 2015), it is important to fully investigate how the interactions between these constructs is associated with motor response inhibition. Importantly, our study contributes to the limited literature examining such interactions, and suggests that anxiety, PTSD, recent cannabis use, and their interactions are not associated with alterations in behavioral or neural activity for motor response inhibition. In

addition, a common factor encompassing anxious symptomology and PTSD did not interact with recent cannabis use in predicting these dependent variables. However, our sample is unique in that participants had recently experienced a traumatic event and that we only measured recent cannabis use through urine toxicology.

Future research should attempt to further isolate the effects of anxious symptomology and trait anxiety, and their interactions with varying degrees of cannabis use, such as chronic and recent users, in predicting motor response inhibition in healthy and clinically diagnoses anxiety disorder populations. Although Spechler and colleagues (2020) partially examined this question, their anxiety disorders group also included individuals diagnosed with mood disorders, such as depression. Therefore, it is necessary to isolate the effects of anxiety and its interaction with cannabis use on motor response inhibition. Such work will allow for a greater conceptual understanding of how anxiety and cannabis use interact to influence motor response inhibition, and help delineate specific conditions in which deficits in motor response inhibition are observed or absent. In addition, this line of work will expand our understanding of whether sub-clinical anxiety interacts with cannabis use to influence this cognitive process at the behavioral and neural level.

In a similar vein, additional work is necessary to examine whether individuals diagnosed with PTSD who also engage in cannabis use experience deficits in motor response inhibition, and the underlying neural correlates associated with these effects. Although our study examined this question under the lens of PCL-5 total scores, there is a paucity of research that has examined the relationship between PTSD, cannabis use, and anxiety with motor response inhibition using continuous measures as our study did. Furthermore, research exploring these effects in clinically diagnosed patients with PTSD will expand our understanding the potential

impact that using cannabis while diagnosed with PTSD has on motor response inhibition. Furthermore, future research should also consider longitudinal designs assessing these independent variables at various time points following traumatic events, such as the acute period, months, and years following trauma. This is because symptoms of PTSD (Benyamini & Solomon, 2005; Bliese et al., 2005; Carty et al., 2006; Derek et al., 2013; Greiger et al., 2006; McFarlane, 1997; Milliken et al., 2007; Orcutt et al., 2004; Solomon & Mikulincer, 2006; Southwick et al., 2000), alterations of endocrine systems (Bremner, 2006; McFarlane, 2010), gene expression (Datson et al., 2013; Gray et al., 2018; Tsankova et al., 2007), neurotransmitter systems (Lowy et al., 1993; Peterlik et al., 2016; Popoli et al., 2012), and endocannabinoid systems (Hill et al., 2011) are differentially impact based on acute versus chronic stress. Such work will ultimately help inform clinical treatment models and creating critical intervention time-windows to alleviate negative symptomology and outcomes experienced by those with PTSD following a traumatic event.

Finally, future research should consider controlling for many of the variables we failed to account for, and examine longitudinal outcomes associated with cannabis use following a traumatic event. Importantly, examining how different forms of trauma experienced are associated with behavioral performance on motor response inhibition tasks, and the neural associates of these effects is pertinent. This will allow for the understanding of whether different forms of traumatic events are more or less likely to impact these cognitive processes, and place an emphasis on treatment based on the type of trauma individuals experienced. Furthermore, examining long-term outcomes from using cannabis following a traumatic event can inform our understanding of whether cannabis use can successfully predict the development of PTSD or greater anxious symptomology at a future time-point. This work can also inform clinical

treatment for individuals who recently experienced trauma, and help alleviate potential long-term negative effects these individuals may experience.

**Conclusions**

In conclusion, our results indicate that motor response inhibition behavioral performance and underlying neural activity did not differ based on the degree of general distress, anxious symptomology, and PTSD in individuals that recently experienced a traumatic event. Also, behavioral performance did not significantly differ between those testing positive and those testing negative for recent cannabis use. In addition, neither general distress, anxiety, or PTSD interacted with recent cannabis use to influence motor response inhibition. Many of our non-significant outcomes were supported by our Bayes Factor analyses, suggesting that such differences are unlikely to be present in our current sample. However, our non-significant group differences for recent cannabis use were not strongly supported from our equivalence testing. It is possible that many of our null results are due to extraneous variables not accounted for in our sample, such as the type of trauma experienced, and the use of urine toxicology to measure recent cannabis use. Nonetheless, these outcomes suggest that motor response inhibition behavioral performance remains intact regardless of anxious symptomology, PTSD, their common factor of general distress, and that individuals testing positive for recent cannabis use may not demonstrate significant differences in performance compared to those testing negative for recent cannabis use. In addition, these constructs do not yield differential neural recruitment of brain regions necessary to successfully engage in motor response inhibition.

## Figures

### Figure Captions

**Figure 1.** Behavioral hypotheses. No main effects predicted for anxiety or cannabis use, but decreased performance for individuals higher in PTSD (**A**). No predicted interactions between anxiety and cannabis use (**B**), but an interaction between PTSD and cannabis use for those higher in PTSD (**C**).

**Figure 2.** Anxious symptomology neuroimaging hypotheses. Individuals with greater anxious symptomology are predicted to have increased activity in the dlPFC, dACC, rIFG, and striatum.

**Figure 3.** Recent cannabis use neuroimaging hypotheses. Recent cannabis users are predicted to have increased activity in the dlPFC, rIFG, and striatum, but reduced activity in the dACC compared to non-recent cannabis users.

**Figure 4.** PTSD neuroimaging hypotheses. Individuals with greater PTSD are predicted to have decreased activity in the dlPFC, dACC, rIFG, and striatum.

**Figure 5.** Stop-Signal task trial procedure. Stop-Signal task requiring participants to respond with either "1" or "2" when presented with an "X" or "O" on Go trials (**A**) or withhold a response on Stop trials (**B**).

**Figure 6.** Stop trial accuracy primary behavioral results. No significant association between Stop trial accuracy and general distress was observed, $r(85) = -0.007$, $p = 0.948$, $BF_{10} = 0.134$ (**A**). There were no differences in Stop trial accuracy between recent negative and positive cannabis use groups, $t(63.29) = 1.469$, $p = 0.147$, $d = 0.070$, $BF_{10} = 0.332$ (**B**).

**Figure 7.** General distress and recent cannabis use interaction in predicting stop trial accuracy Non-significant interaction between general distress and recent cannabis use, $F(1, 80) < 0.001$, $p = 0.979$, $R^2$ change $< 0.001$, $BF_{10} = 0.476$.

**Figure 8.** Stop trial accuracy secondary behavioral results. No significant association between Stop trial accuracy and anxiety $r(85) = -0.046$, $p = 0.673$, $BF_{10} = 0.146$ (**A**), and PTSD, $r(85) = 0.033$, $p = 0.764$, $BF_{10} = 0.140$ (**B**).

**Figure 9.** Recent cannabis use interactions with anxiety and PTSD in predicting stop trial accuracy. Non-significant interaction between recent cannabis use and DASS-21 anxiety subscale scores, $F(1, 80) = 0.006$, $p = 0.940$, $R^2$ change < 0.001, $BF_{10} = 0.473$ (**A**), and PCL-5 total scores, $F(1, 80) = 0.001$, $p = 0.979$, $R^2$ change < 0.001, $BF_{10} = 0.478$ (**B**) in predicting Stop trial accuracy.

**Figure 10**. Go trial accuracy and RT association with general distress. No significant association general distress and Go trial accuracy, $r(85) = -0.001$, $p = 0.991$, $BF_{10} = 0.134$ (**A**), or Go trial RT, $r(85) = -0.048$, $p = 0.656$, $BF_{10} = 0.148$ (**B**).

**Figure 11.** Go trial accuracy and RT between recent cannabis use groups. No significant differences in Go trial accuracy, $t(85) = 0.986$, $p = 0.327$, $d = 0.213$, $BF_{10} = 0.345$ (**A**), or Go trial RT, $t(85) = 0.039$, $p = 0.969$, $d = 0.009$, $BF_{10} = 0.226$ (**B**) between recent cannabis use groups.

**Figure 12.** Recent cannabis use interactions with general distress in predicting Go trial accuracy and RT. Non-significant interactions between recent cannabis use and general distress in predicting Go trial accuracy, $F(1, 80) = 0.074$, $p = 0.787$, $R^2$ change = 0.001, $BF_{10} = 0.507$ (**A**), and Go trial RT, $F(1, 80) = 0.144$, $p = 0.705$, $R^2$ change = 0.002, $BF_{10} = 0.470$ (**B**).

**Figure 13.** Go trial accuracy association with anxiety and PTSD. No significant association between Go trial accuracy and DASS-21 anxiety subscale, r(85) = -0.018, p = 0.868, BF10 = 0.136 (A), or PCL-5 total, r(85) = 0.016, p = 0.884, BF10 = 0.135 (B) scores.

**Figure 14.** Go trial RT association with Anxiety and PTSD. No significant association between Go trial RT and DASS-21 anxiety subscale, $r(85) = -0.014$, $p = 0.898$, $BF_{10} = 0.135$ (**A**), or PCL-5 total, $r(85) = -0.075$, $p = 0.490$, $BF_{10} = 0.169$ (**B**) scores.

**Figure 15.** Recent cannabis use interactions with anxiety and PTSD in predicting Go trial accuracy. Non-significant interactions between recent cannabis use and DASS-21 anxiety subscale scores, $F(1, 80) = 0.103$, $p = 0.749$, $R^2$ change = 0.001, $BF_{10} = 0.512$ (**A**), and PCL-5 total scores, $F(1, 80) = 0.024$, $p = 0.877$, $R^2$ change < 0.001, $BF_{10} = 0.4985$ (**B**) in predicting Go trial accuracy.

**Figure 16.** Recent cannabis use Interactions with anxiety and PTSD in predicting Go trial RT. Non-significant interactions between recent cannabis use and DASS-21 anxiety subscale scores, $F(1, 80) = 0.735$, $p = 0.394$, $R^2$ change = 0.008, $BF_{10} = 0.594$ (**A**), and PCL-5 total scores, $F(1, 80) = 0.009$, $p = 0.925$, $R^2$ change < 0.001, $BF_{10} = 0.445$ (**B**) in predicting Go trial RT.

Figure 1. Behavioral Hypotheses

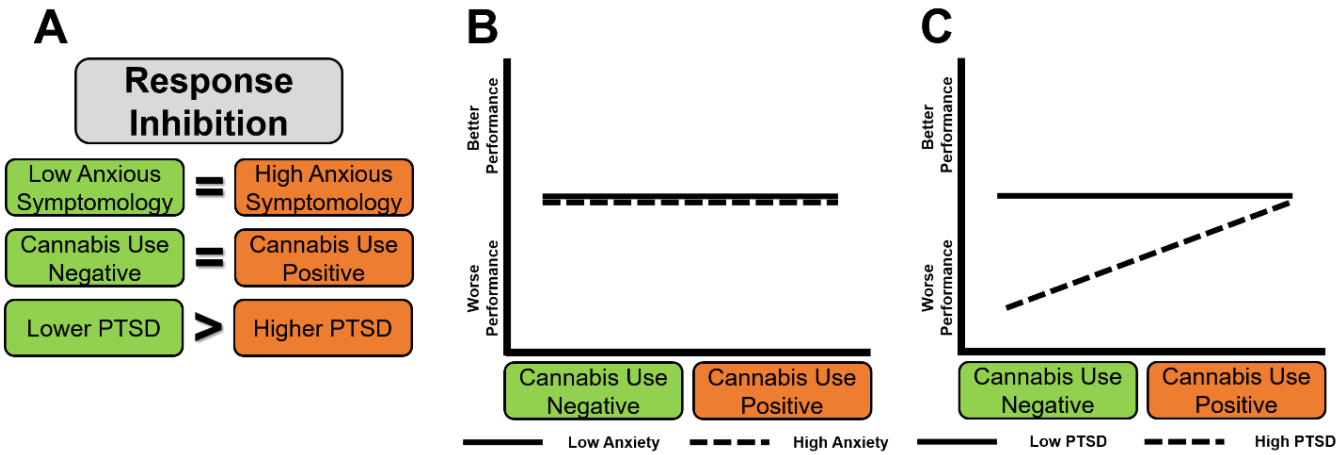


Figure 2. Anxious Symptomology Neuroimaging Hypotheses

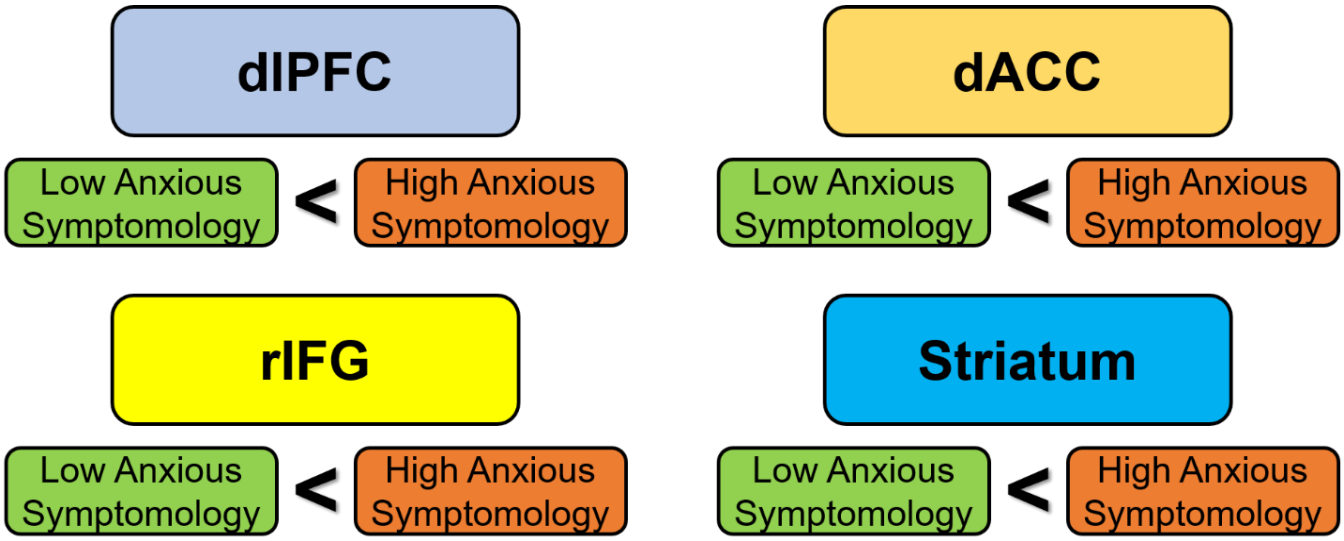


Figure 3. Recent Cannabis Use Neuroimaging Hypotheses

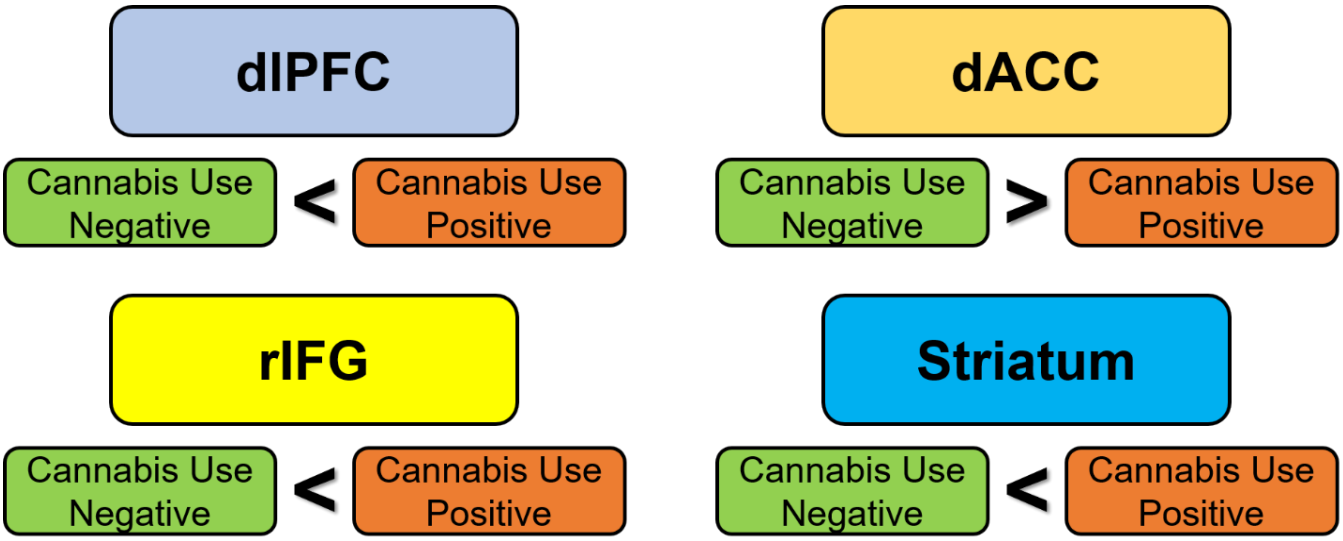


Figure 4. PTSD Neuroimaging Hypotheses

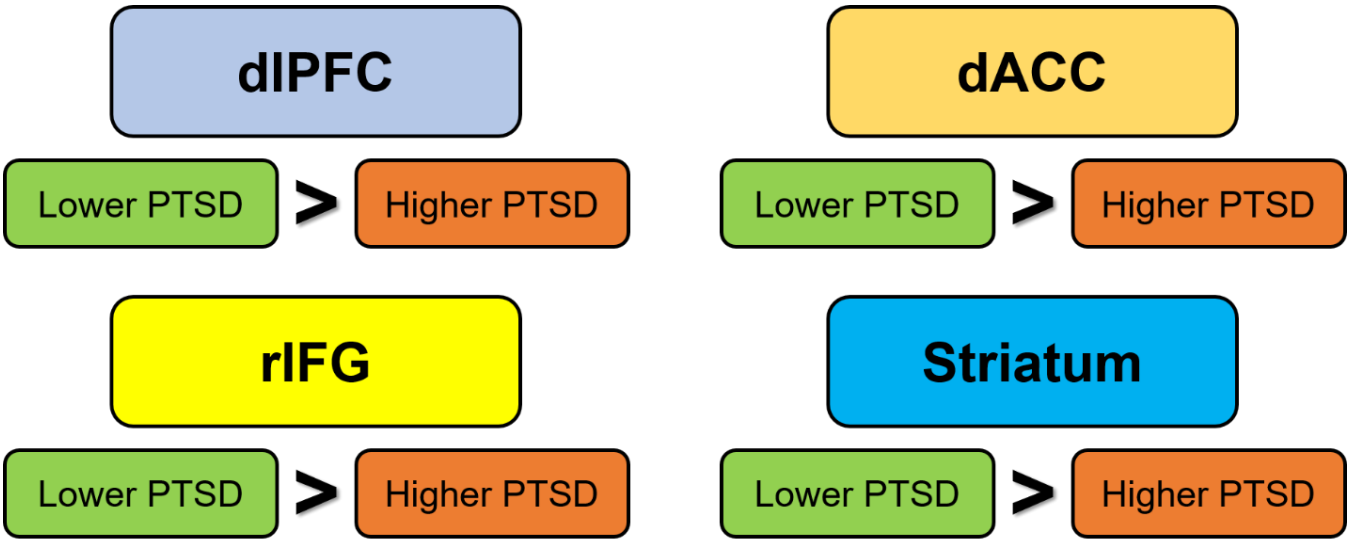


Figure 5. Stop-Signal Task Trial Procedure

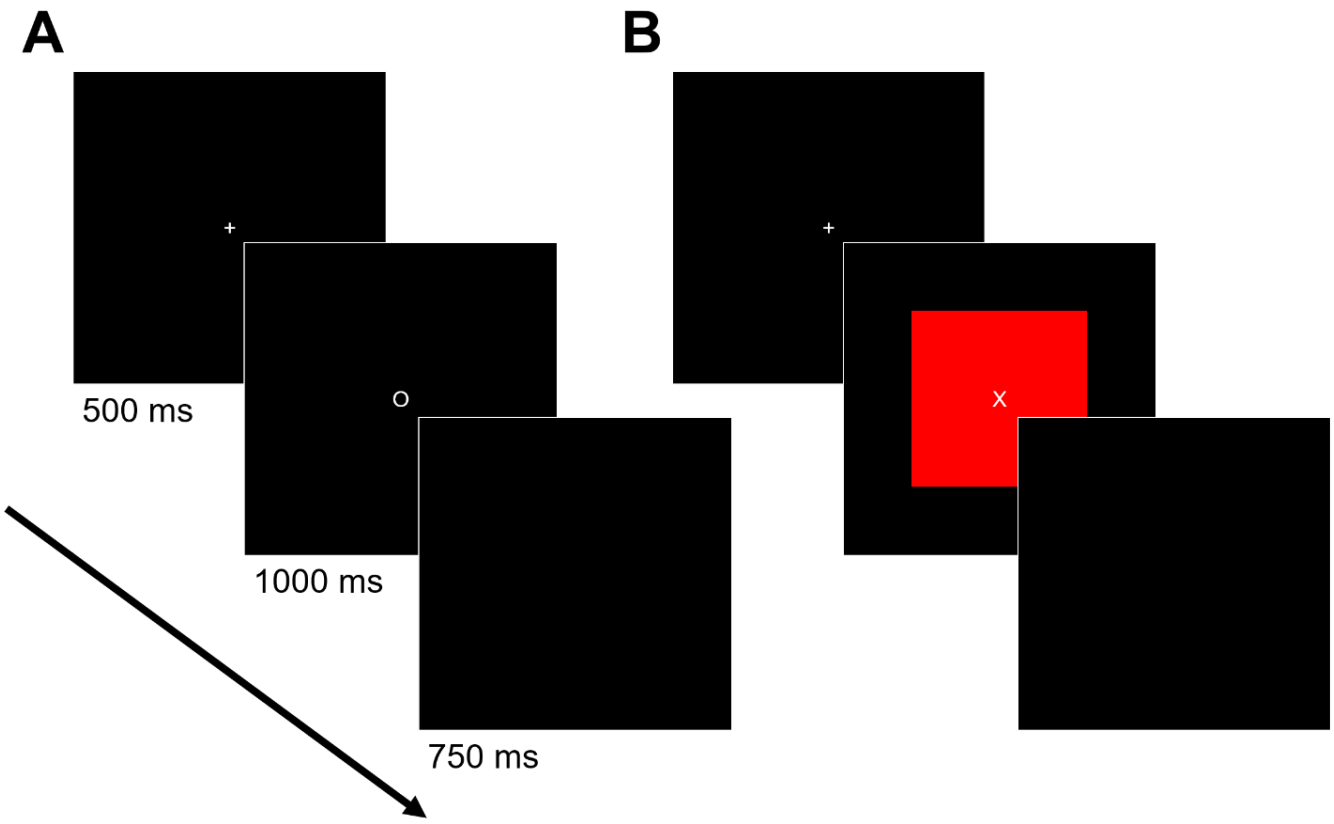


Figure 6. Stop Trial Accuracy Primary Behavioral Results

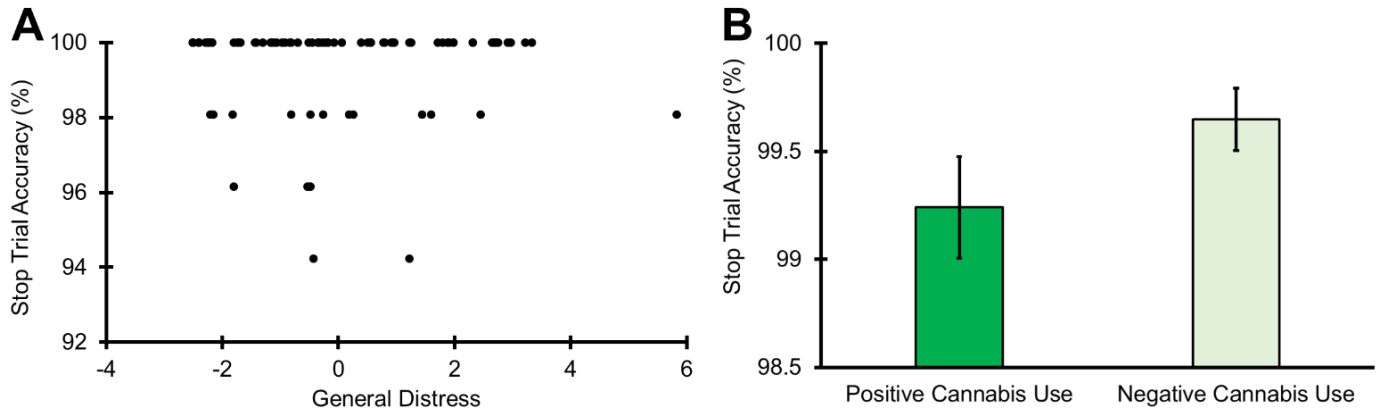


Figure 7. General Distress and Recent Cannabis Use Interaction in Predicting Stop Trial Accuracy

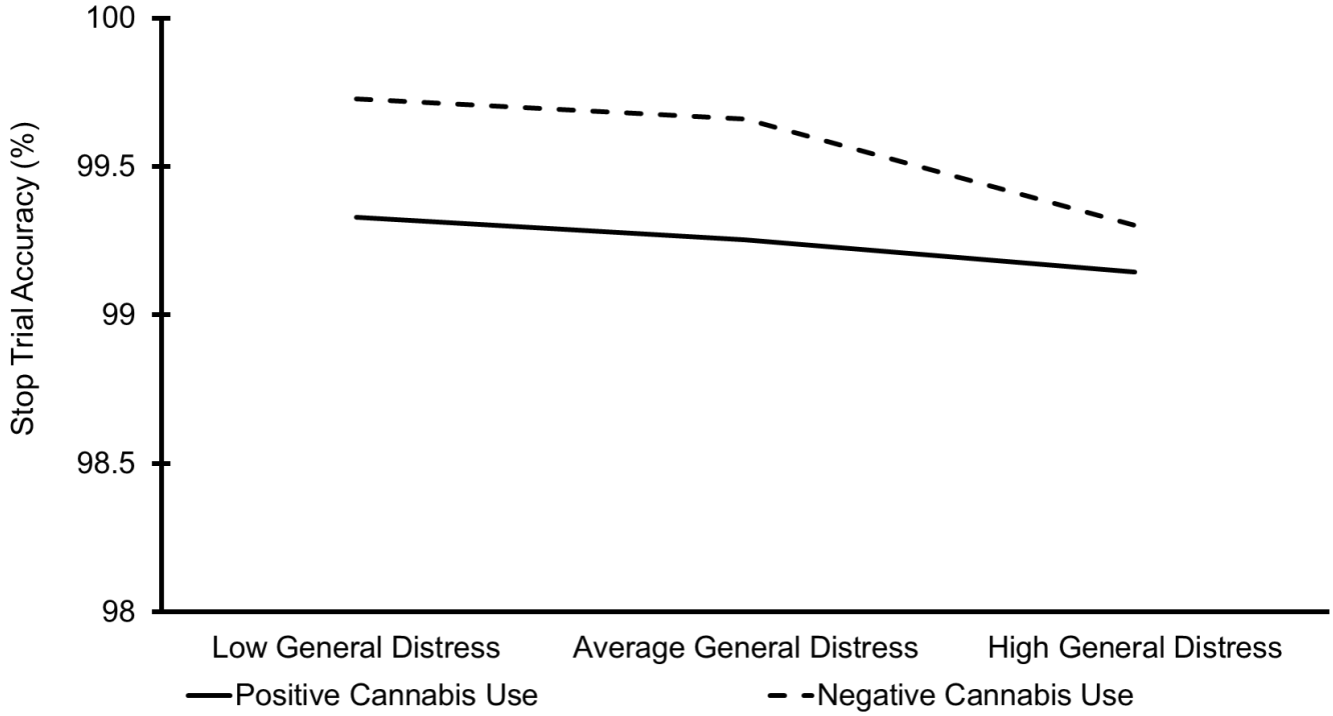


Figure 8. Stop Trial Accuracy Secondary Behavioral Results

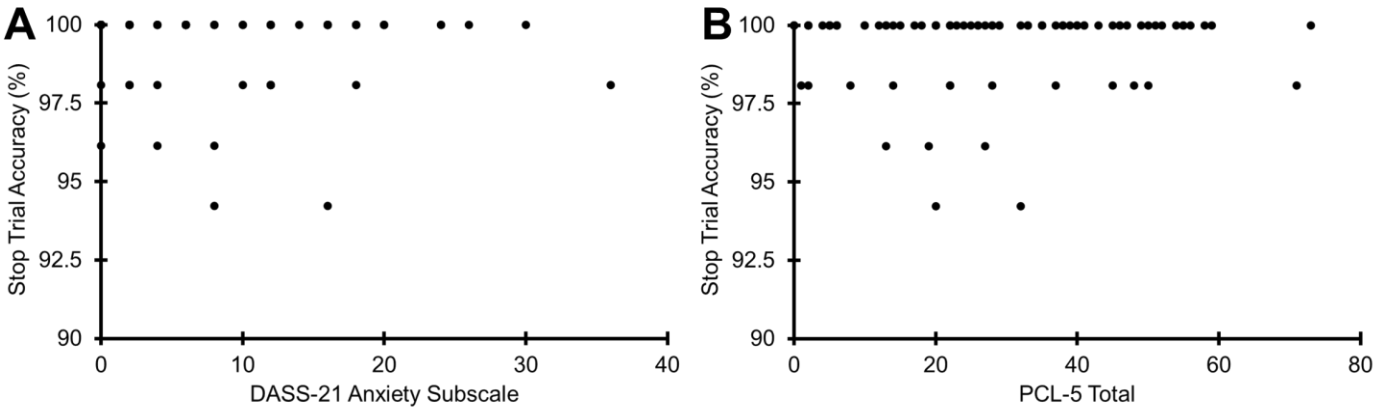


Figure 9. Recent Cannabis Use Interactions with Anxiety and PTSD in Predicting Stop Trial Accuracy

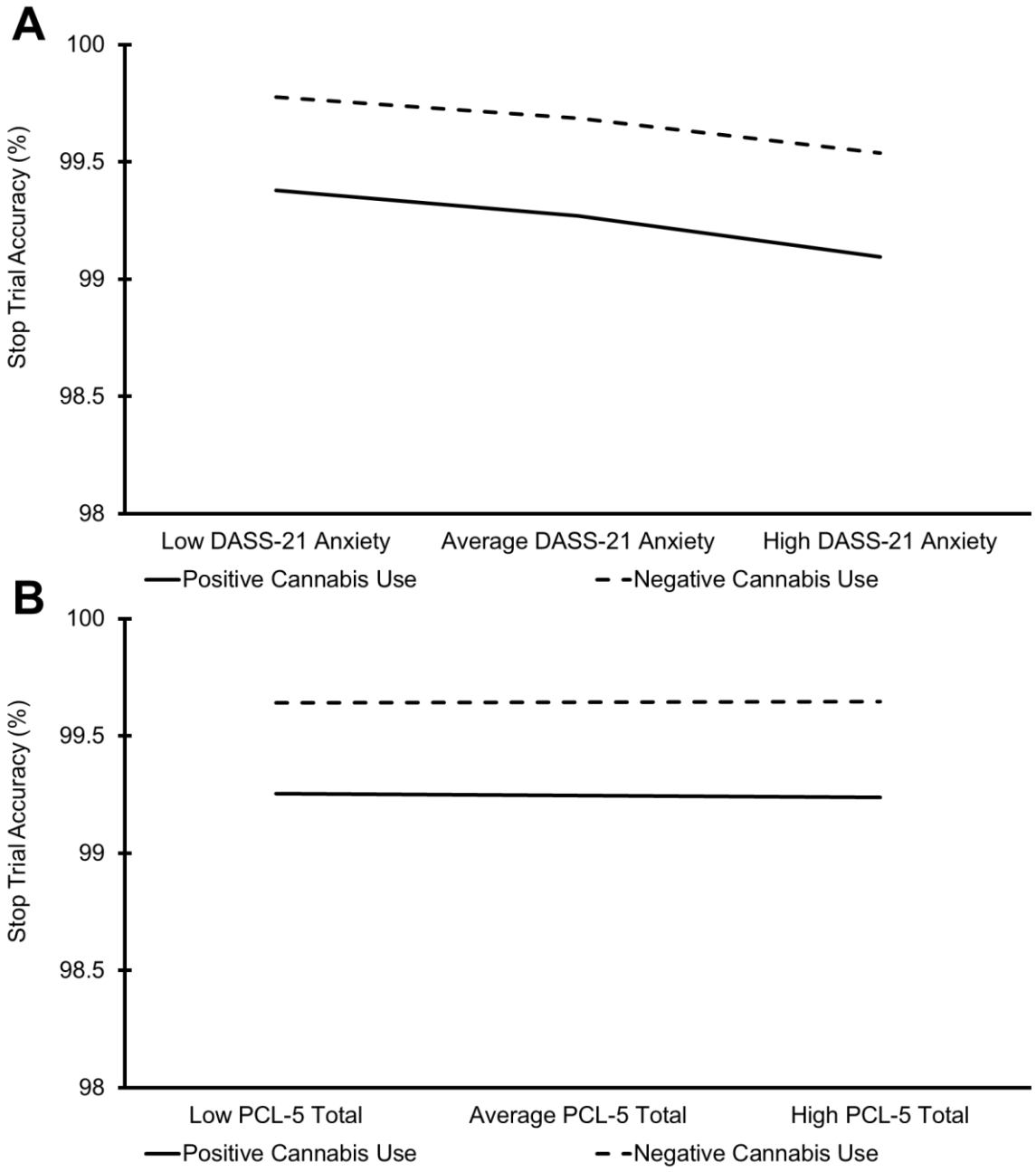


Figure 10. Go Trial Accuracy and RT Association with General Distress

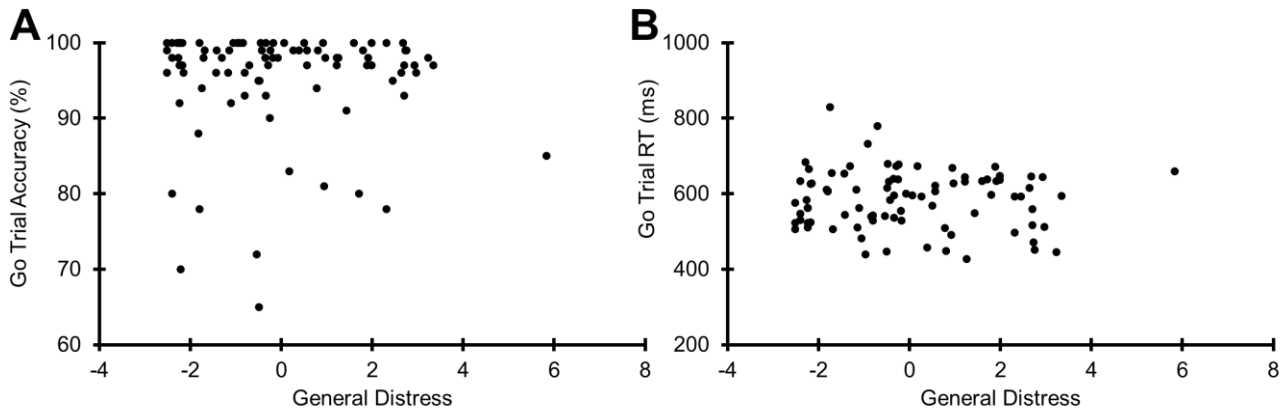


Figure 11. Go Trial Accuracy and RT Between Recent Cannabis Use Groups

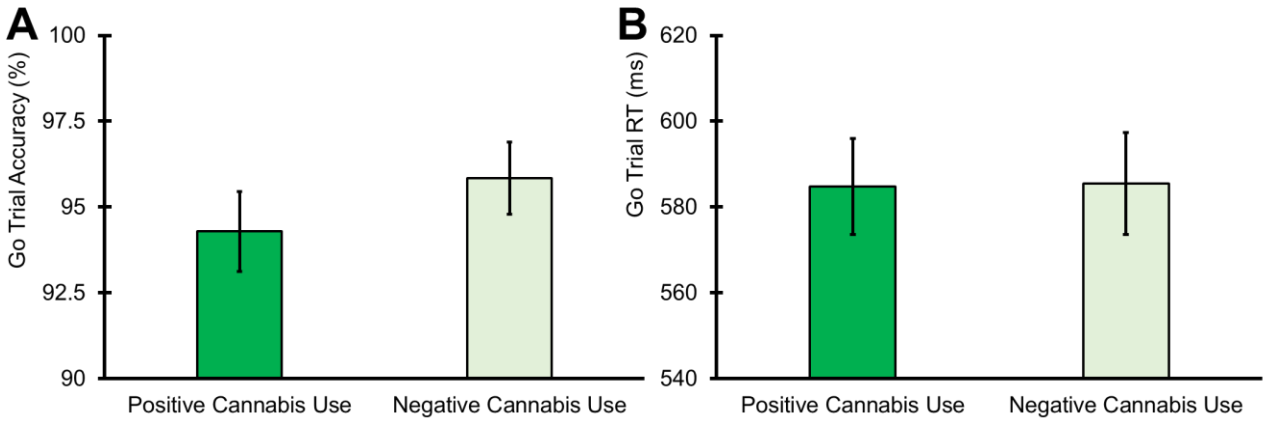


Figure 12. Recent Cannabis Use Interactions with General Distress in Predicting Go Trial Accuracy and RT

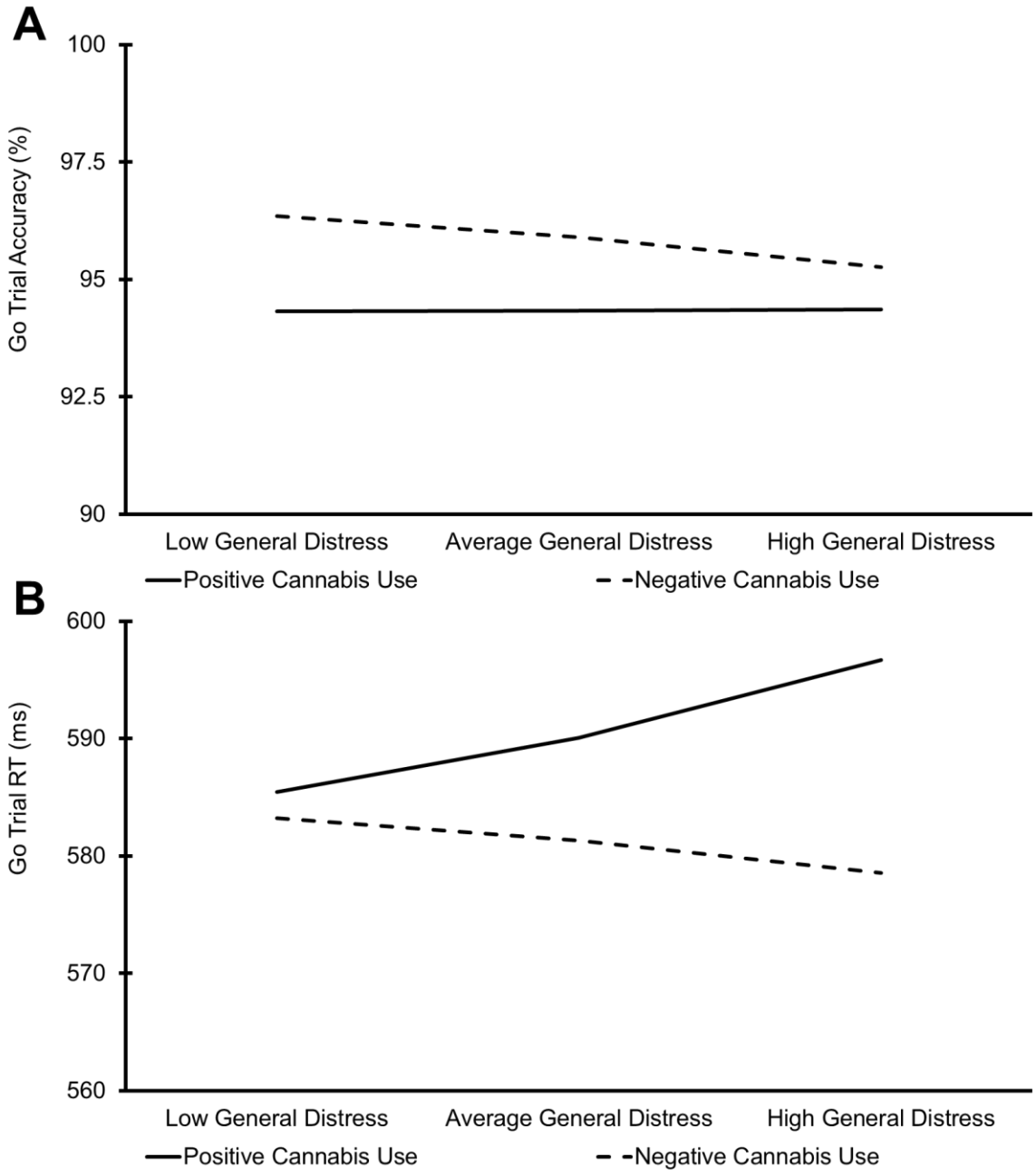


Figure 13. Go Trial Accuracy Association with Anxiety and PTSD

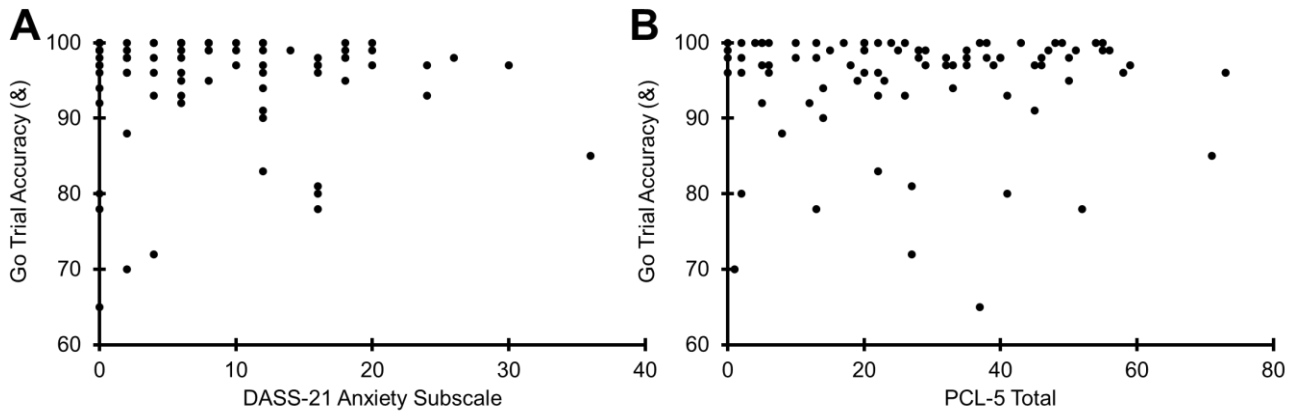


Figure 14. Go Trial RT Association with Anxiety and PTSD

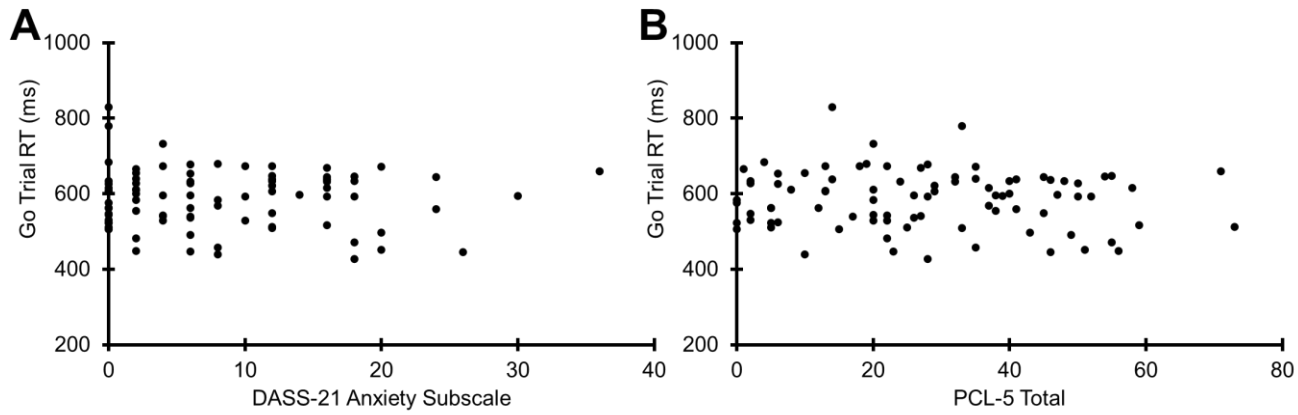


Figure 15. Recent Cannabis Use Interactions with Anxiety and PTSD in Predicting Go Trial Accuracy

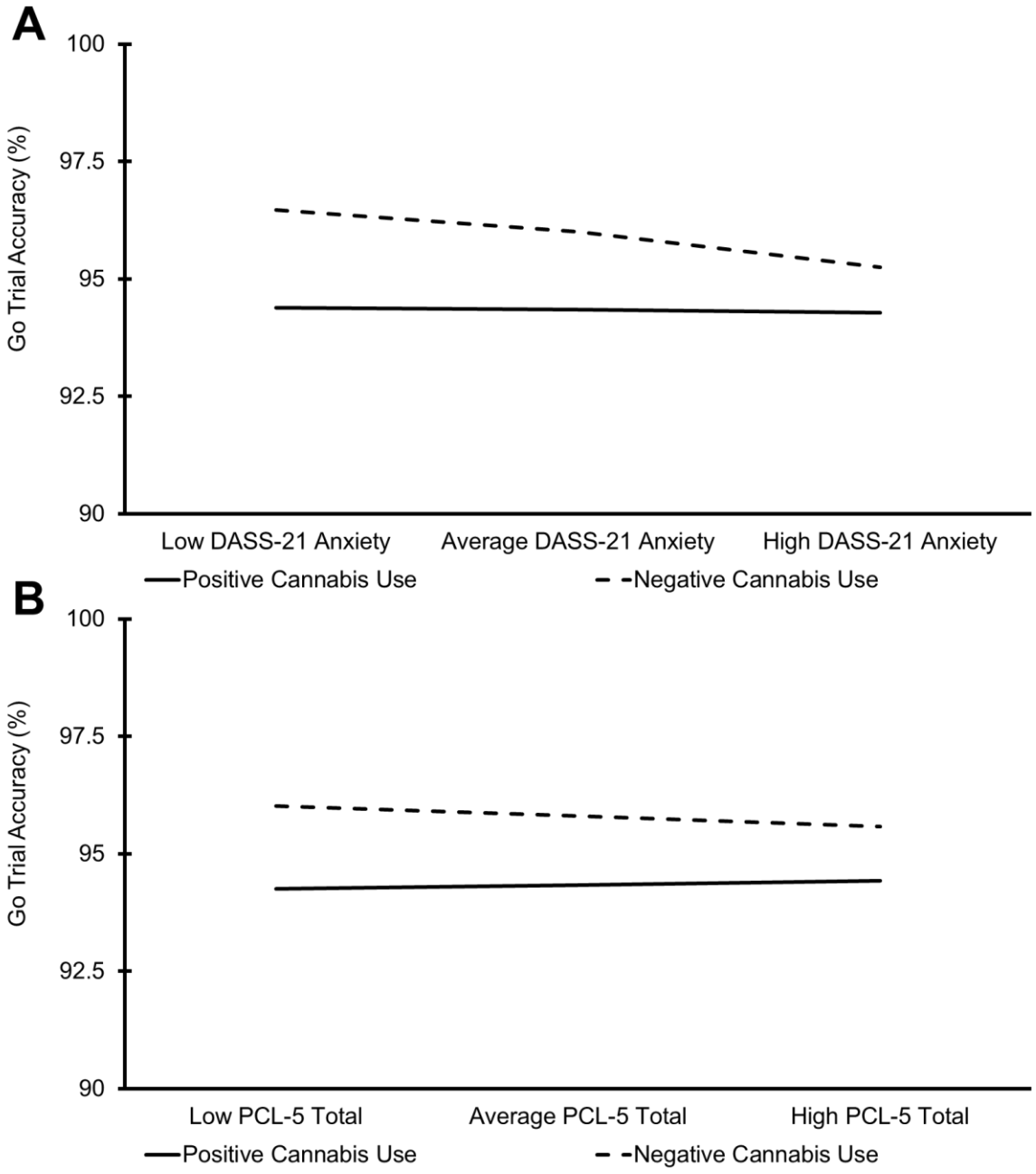
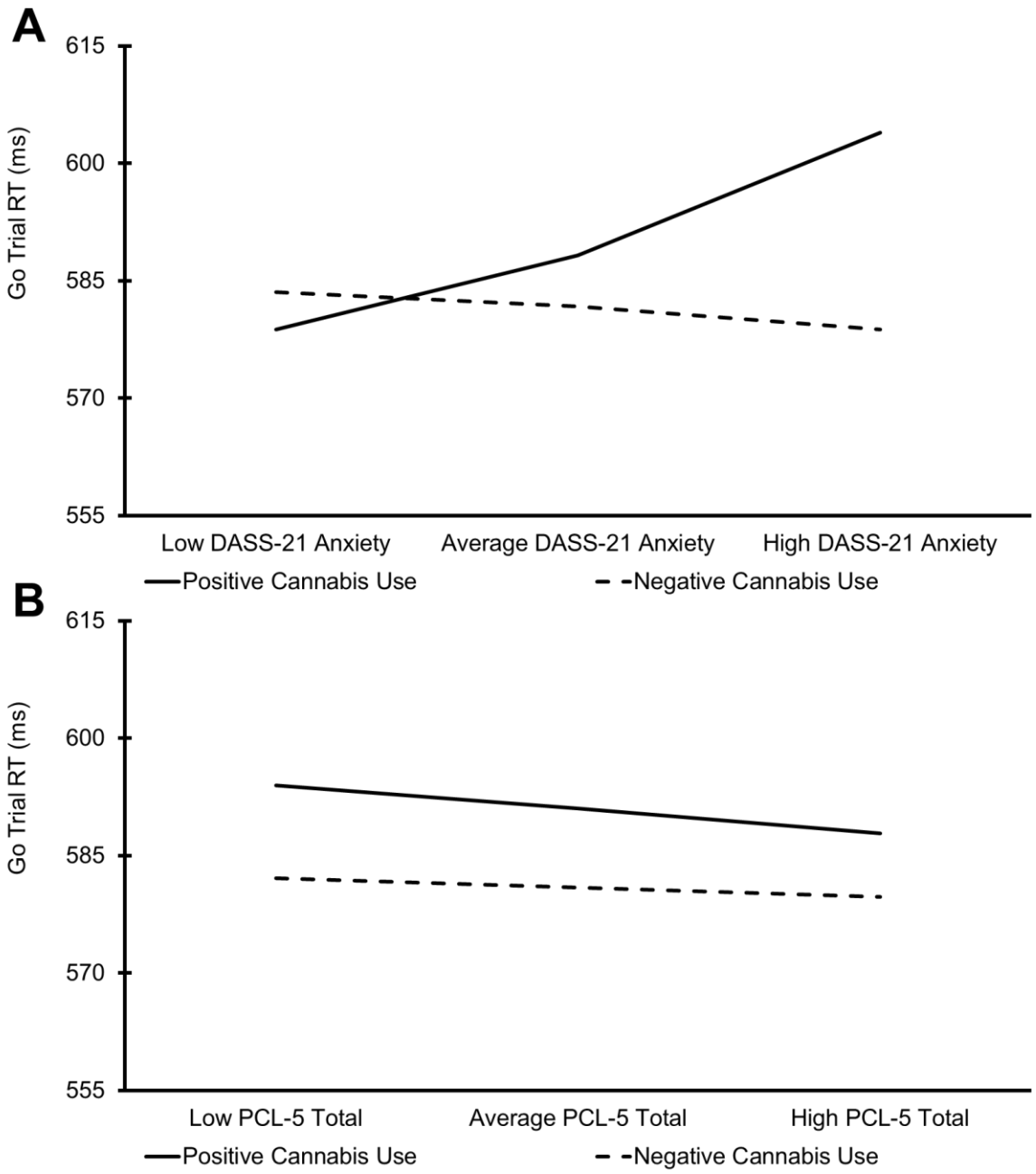


Figure 16. Recent Cannabis Use Interactions with Anxiety and PTSD in Predicting Go Trial RT



## Tables

### Table Captions

**Table 1.** Basic descriptive information for all participants, participants used for behavioral analyses, and participants used for fMRI analyses.

**Table 2.** Recent cannabis use group descriptive information.

**Table 3.** Depression, Stress, and Anxiety Scale (DASS-21) questionnaire.

**Table 4.** PTSD Checklist for DSM-5 (PCL-5) questionnaire.

**Table 5.** Alcohol Use Disorders Identification Test (AUDIT) questionnaire.

**Table 6.** Proportion of variability from components from full behavioral sample.

**Table 7.** Regression results for predicting Stop trial accuracy from general distress, recent cannabis use, and their interaction.

**Table 8.** Regression results for predicting Stop trial accuracy from DASS-21 anxiety subscale scores, recent cannabis use, and their interaction.

**Table 9.** Regression results for predicting Stop trial accuracy from PCL-5 total scores, recent cannabis use, and their interaction.

**Table 10.** Regression results for predicting Go trial accuracy from general distress, recent cannabis use, and their interaction.

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**Table 14.** Regression results for predicting Go trial RT from DASS-21 anxiety subscale Scores, recent cannabis use, and their interaction.

**Table 15.** Regression results for predicting Go trial RT from PCL-5 total scores, recent cannabis use, and their interaction.

Table 1. Basic Descriptive Information for All Participants, Participants Used for Behavioral Analyses, and Participants Used for fMRI Analyses

*Overall Sample Descriptive Information*

| Sample | Total *n* | $M_{age}$ | $SE_{age}$ | Males | Females |
|---|---|---|---|---|---|
| Completed Stop-Signal Task 2 Weeks Post-Trauma | 92 | 32.82 | 1.18 | 38 | 54 |
| Behavioral Analyses | 87 | 32.14 | 1.16 | 36 | 51 |
| fMRI Analyses | 66 | 32.16 | 1.25 | 24 | 42 |

Table 2. Recent Cannabis Use Group Descriptive Information

*Recent Cannabis Groups' Descriptive Information*

| | Positive Recent Cannabis Use | | | | | Negative Recent Cannabis Use | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Sample | Total *n* | $M_{age}$ | $SE_{age}$ | Males | Females | Total *n* | $M_{age}$ | $SE_{age}$ | Males | Females |
| Behavioral Analyses | 38 | 28.82 | 1.67 | 17 | 21 | 49 | 34.83 | 1.51 | 19 | 30 |
| fMRI Analyses | 32 | 28.01 | 1.39 | 13 | 19 | 34 | 36.08 | 1.83 | 11 | 23 |

Table 3. DASS-21 Questionnaire

*Depression, Stress, and Anxiety Scale (DASS-21) Questionnaire Form*

| Question # | Statement | Did not apply to me at all | Applied to me to some degree, or some of the time | Applied to me to a considerable degree or a good part of the time | Applied to me very much or most of the time |
|---|---|---|---|---|---|
| 1 (s) | I found it hard to wind down | 0 | 1 | 2 | 3 |
| 2 (a) | I was aware of dryness of my mouth | 0 | 1 | 2 | 3 |
| 3 (d) | I couldn't seem to experience any positive feeling at all | 0 | 1 | 2 | 3 |
| 4 (a) | I experienced breathing difficulty (e.g. excessively rapid breathing, breathlessness in the absence of physical exertion) | 0 | 1 | 2 | 3 |
| 5 (d) | I found it difficult to work up the initiative to do things | 0 | 1 | 2 | 3 |
| 6 (s) | I tended to over-react to situations | 0 | 1 | 2 | 3 |
| 7 (a) | I experienced trembling (e.g. in the hands) | 0 | 1 | 2 | 3 |
| 8 (s) | I felt that I was using a lot of nervous energy | 0 | 1 | 2 | 3 |
| 9 (a) | I was worried about situations in which I might panic and make a fool of myself | 0 | 1 | 2 | 3 |
| 10 (d) | I felt that I had nothing to look forward to | 0 | 1 | 2 | 3 |
| 11 (s) | I found myself getting agitated | 0 | 1 | 2 | 3 |
| 12 (s) | I found it difficult to relax | 0 | 1 | 2 | 3 |
| 13 (d) | I felt down-hearted and blue | 0 | 1 | 2 | 3 |
| 14 (s) | I was intolerant of anything that kept me from getting on with what I was doing | 0 | 1 | 2 | 3 |
| 15 (a) | I felt I was close to panic | 0 | 1 | 2 | 3 |
| 16 (d) | I was unable to become enthusiastic about anything | 0 | 1 | 2 | 3 |
| 17 (d) | I felt I wasn't worth much as a person | 0 | 1 | 2 | 3 |
| 18 (s) | I felt that I was rather touchy | 0 | 1 | 2 | 3 |
| 19 (a) | I was aware of the action of my heart in the absence of physical exertion (e.g. sense of heart rate increase, heart missing a beat) | 0 | 1 | 2 | 3 |
| 20 (a) | I felt scared without any good reason | 0 | 1 | 2 | 3 |
| 21 (d) | I felt that life was meaningless | 0 | 1 | 2 | 3 |

Table 4. PCL-5 Questionnaire

*PTSD Checklist for DSM-5 (PCL-5) Questionnaire Form*

| Question # | Statement | Not at all | A little bit | Moderately | Quite a bit | Extremely |
|---|---|---|---|---|---|---|
| 1 | Repeated, disturbing, and unwanted memories of the stressful experience? | 0 | 1 | 2 | 3 | 4 |
| 2 | Repeated, disturbing dreams of the stressful experience? | 0 | 1 | 2 | 3 | 4 |
| 3 | Suddenly feeling or acting as if the stressful experience were actually happening again (as if you were actually back there reliving it)? | 0 | 1 | 2 | 3 | 4 |
| 4 | Feeling very upset when something reminded you of the stressful experience? | 0 | 1 | 2 | 3 | 4 |
| 5 | Having strong physical reactions when something reminded you of the stressful experience (for example, heart pounding, trouble breathing, sweating)? | 0 | 1 | 2 | 3 | 4 |
| 6 | Avoiding memories, thoughts, or feelings related to the stressful experience? | 0 | 1 | 2 | 3 | 4 |
| 7 | Avoiding external reminders of the stressful experience (for example, people, places, conversations, activities, objects, or situations)? | 0 | 1 | 2 | 3 | 4 |
| 8 | Trouble remembering important parts of the stressful experience? | 0 | 1 | 2 | 3 | 4 |
| 9 | Having strong negative beliefs about yourself, other people, or the world (for example, having thoughts such as: I am bad, there is something seriously wrong with me, no one can be trusted, the world is completely dangerous)? | 0 | 1 | 2 | 3 | 4 |
| 10 | Blaming yourself or someone else for the stressful experience or what happened after it? | 0 | 1 | 2 | 3 | 4 |
| 11 | Having strong negative feelings such as fear, horror, anger, guilt, or shame? | 0 | 1 | 2 | 3 | 4 |
| 12 | Loss of interest in activities that you used to enjoy? | 0 | 1 | 2 | 3 | 4 |
| 13 | Feeling distant or cut off from other people? | 0 | 1 | 2 | 3 | 4 |
| 14 | Trouble experiencing positive feelings (for example, being unable to feel happiness or have loving feelings for people close to you)? | 0 | 1 | 2 | 3 | 4 |
| 15 | Irritable behavior, angry outbursts, or acting aggressively? | 0 | 1 | 2 | 3 | 4 |
| 16 | Taking too many risks or doing things that could cause you harm? | 0 | 1 | 2 | 3 | 4 |
| 17 | Being “superalert” or watchful or on guard? | 0 | 1 | 2 | 3 | 4 |
| 18 | Feeling jumpy or easily startled? | 0 | 1 | 2 | 3 | 4 |
| 19 | Having difficulty concentrating? | 0 | 1 | 2 | 3 | 4 |
| 20 | Trouble falling or staying asleep? | 0 | 1 | 2 | 3 | 4 |

Table 5. AUDIT Questionnaire

*Alcohol Use Disorders Identification Test (AUDIT) Questionnaire Form*

| Question # | Statement | 0 | 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|---|
| 1 | How often do you have a drink containing alcohol? | Never (Skip to #9-10) | Monthly or less | 2 to 4 times a month | 2 to 3 times a week | 4 or more times a week |
| 2 | How many drinks containing alcohol do you have on a typical day when you are drinking? | 1 or 2 | 3 or 4 | 5 or 6 | 7, 8, or 9 | 10 or more |
| 3 | How often do you have six or more drinks on one occasion? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |
| 4 | How often during the last year have you found that you were not able to stop drinking once you had started? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |
| 5 | How often during the last year have you failed to do what was normally expected from you because of drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |
| 6 | How often during the last year have you been unable to remember what happened the night before because you had been drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |
| 7 | How often during the last year have you needed an alcoholic drink first thing in the morning to get yourself going after a night of heavy drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |
| 8 | How often during the last year have you had a feeling of guilt or remorse after drinking? | Never | Less than monthly | Monthly | Weekly | Daily or almost daily |
| 9 | Have you or someone else been injured as a result of your drinking? | No | | Yes, but not in the last year | | Yes, during the last year |
| 10 | Has a relative, friend, doctor, or another health professional expressed concern about your drinking or suggested you cut down? | No | | Yes, but not in the last year | | Yes, during the last year |

Table 6. Proportion of Variability from Components from Full Behavioral Sample

*Proportion of Variability from Components Extracted from Full Behavioral Sample*

| Components | Proportion of Variance | Cumulative Proportion | Standard Deviation |
|---|---|---|---|
| Principal Component 1***** | 0.442 | 0.442 | 3.453 |
| Principal Component 2 | 0.071 | 0.513 | 1.385 |
| Principal Component 3 | 0.058 | 0.571 | 1.253 |
| Principal Component 4 | 0.056 | 0.627 | 1.230 |
| Principal Component 5 | 0.044 | 0.671 | 1.095 |
| Principal Component 6 | 0.035 | 0.706 | 0.972 |
| Principal Component 7 | 0.032 | 0.738 | 0.926 |
| Principal Component 8 | 0.029 | 0.767 | 0.889 |
| Principal Component 9 | 0.028 | 0.795 | 0.874 |
| Principal Component 10 | 0.026 | 0.821 | 0.839 |
| Principal Component 11 | 0.024 | 0.846 | 0.811 |
| Principal Component 12 | 0.020 | 0.865 | 0.728 |
| Principal Component 13 | 0.018 | 0.883 | 0.696 |
| Principal Component 14 | 0.018 | 0.901 | 0.689 |
| Principal Component 15 | 0.016 | 0.917 | 0.654 |
| Principal Component 16 | 0.013 | 0.930 | 0.598 |
| Principal Component 17 | 0.011 | 0.941 | 0.548 |
| Principal Component 18 | 0.010 | 0.952 | 0.530 |
| Principal Component 19 | 0.008 | 0.960 | 0.472 |
| Principal Component 20 | 0.007 | 0.967 | 0.447 |
| Principal Component 21 | 0.007 | 0.974 | 0.431 |
| Principal Component 22 | 0.006 | 0.980 | 0.406 |
| Principal Component 23 | 0.005 | 0.986 | 0.380 |
| Principal Component 24 | 0.005 | 0.991 | 0.363 |
| Principal Component 25 | 0.004 | 0.994 | 0.337 |
| Principal Component 26 | 0.003 | 0.998 | 0.297 |
| Principal Component 27 | 0.002 | 1.000 | 0.232 |

*****Retained component

Table 7. Regression Results for Predicting Stop Trial Accuracy from General Distress, Recent Cannabis Use, and their Interaction

*Regression Analysis Predicting Stop Trial Accuracy from General Distress, Recent Cannabis Use, and their Interaction.*

| | Model 1 | | | Model 2 | | |
|---|---|---|---|---|---|---|
| Variable | *B* | *SE B* | *b* | *B* | *SE B* | *b* |
| (Constant) | 99.367 | 0.242 | | 99.366 | 0.244 | |
| General Distress | -0.038 | 0.075 | -0.057 | -0.037 | 0.091 | -0.055 |
| Recent Cannabis Use | -0.407 | 0.271 | -0.165 | -0.407 | 0.273 | -0.165 |
| AUDIT | -0.018 | 0.031 | -0.063 | -0.018 | .031 | -0.063 |
| Age | -0.005 | 0.012 | -0.051 | -0.005 | 0.012 | -0.050 |
| Gender | 0.473 | 0.276 | 0.190 | 0.473 | 0.278 | 0.190 |
| General Distress & Recent Cannabis Use Interaction | | | | -0.004 | 0.155 | -0.003 |
| $R^2$ | | 0.067 | | | 0.067 | |
| $R^2$ Change | | 0.067 | | | < 0.001 | |

**$p < 0.05$, **$p < 0.01$

Table 8. Regression Results for Predicting Stop Trial Accuracy from DASS-21 Anxiety Subscale Scores, Recent Cannabis Use, and their Interaction

*Regression Analysis Predicting Stop Trial Accuracy from General Distress, Recent Cannabis Use, and their Interaction.*

| | Model 1 | | | Model 2 | | |
|---|---|---|---|---|---|---|
| Variable | *B* | *SE B* | *b* | *B* | *SE B* | *b* |
| (Constant) | 99.359 | 0.240 | | 99.358 | 0.242 | |
| DASS-21 Anxiety | -0.016 | 0.017 | -0.104 | -0.015 | 0.020 | -0.098 |
| Recent Cannabis Use | -0.421 | 0.031 | -0.170 | -0.422 | 0.273 | -0.171 |
| AUDIT | -0.018 | 0.031 | -0.064 | -0.019 | 0.031 | -0.064 |
| Age | -0.006 | 0.012 | -0.055 | -0.006 | 0.012 | -0.054 |
| Gender | 0.496 | 0.274 | 0.199 | 0.497 | 0.276 | 0.199 |
| DASS-21 Anxiety & Recent Cannabis Use Interaction | | | | -0.003 | 0.036 | -0.010 |
| $R^2$ | | 0.074 | | | 0.074 | |
| $R^2$ Change | | 0.074 | | | < 0.001 | |

**$p < 0.05$, **$p < 0.01$

Table 9. Regression Results for Predicting Stop Trial Accuracy from PCL-5 Total Scores, Recent Cannabis Use, and their Interaction

*Regression Analysis Predicting Stop Trial Accuracy from General Distress, Recent Cannabis Use, and their Interaction.*

| | Model 1 | | | Model 2 | | |
|---|---|---|---|---|---|---|
| Variable | *B* | *SE B* | *b* | *B* | *SE B* | *b* |
| (Constant) | 99.381 | 0.242 | | 99.381 | 0.244 | |
| PCL-5 Total | < -0.001 | 0.007 | -0.001 | < -0.001 | 0.009 | 0.002 |
| Recent Cannabis Use | -0.400 | 0.271 | -0.162 | -0.399 | 0.273 | -0.161 |
| AUDIT | -0.019 | 0.031 | -0.064 | -0.019 | 0.032 | -0.065 |
| Age | -0.004 | 0.012 | -0.041 | -0.004 | 0.012 | -0.041 |
| Gender | 0.443 | 0.274 | 0.178 | 0.444 | 0.277 | 0.178 |
| PCL-5 Total & Recent Cannabis Use Interaction | | | | -0.001 | 0.015 | -0.005 |
| $R^2$ | | 0.064 | | | 0.064 | |
| $R^2$ Change | | 0.064 | | | < 0.001 | |

**$p < 0.05$, ****$p < 0.01$

Table 10. Regression Results for Predicting Go Trial Accuracy from General Distress, Recent Cannabis Use, and their Interaction

*Regression Analysis Predicting Go Trial Accuracy from General Distress, Recent Cannabis Use, and their Interaction.*

| | Model 1 | | | Model 2 | | |
|---|---|---|---|---|---|---|
| Variable | *B* | *SE B* | *b* | *B* | *SE B* | *b* |
| (Constant) | 94.352 | 1.446 | | 94.352 | 1.457 | |
| General Distress | -0.160 | 0.446 | -0.040 | -0.244 | 0.545 | -0.062 |
| Recent Cannabis Use | -1.473 | 1.622 | -0.101 | -1.474 | 1.631 | -0.101 |
| AUDIT | < 0.001 | 0.186 | < 0.001 | 0.004 | 0.188 | 0.002 |
| Age | -0.011 | 0.071 | -0.018 | -0.013 | 0.072 | -0.021 |
| Gender | 2.463 | 1.648 | 0.168 | 2.433 | 1.661 | 0.166 |
| General Distress & Recent Cannabis Use Interaction | | | | 0.251 | 0.926 | 0.037 |
| $R^2$ | | 0.038 | | | 0.039 | |
| $R^2$ Change | | 0.038 | | | 0.001 | |

**$p < 0.05$, **$p < 0.01$

Table 11. Regression Results for Predicting Go Trial RT from General Distress, Recent Cannabis Use, and their Interaction

*Regression Analysis Predicting Go Trial RT from General Distress, Recent Cannabis Use, and their Interaction.*

| | Model 1 | | | Model 2 | | |
|---|---|---|---|---|---|---|
| Variable | *B* | *SE B* | *b* | *B* | *SE B* | *b* |
| (Constant) | 584.286 | 14.647 | | 584.642 | 14.755 | |
| General Distress | 0.149 | 4.514 | 0.004 | -1.045 | 5.523 | -0.025 |
| Recent Cannabis Use | 9.986 | 16.429 | 0.065 | 9.964 | 16.516 | 0.065 |
| AUDIT | -1.769 | 1.888 | -0.098 | -1.706 | 1.905 | -0.095 |
| Age | 2.350 | 0.720 | 0.355 | 2.328 | 0.727 | 0.351 |
| Gender | -2.932 | 0.720 | -0.019 | -3.356 | 16.816 | -0.022 |
| General Distress & Recent Cannabis Use Interaction | | | | 3.560 | 9.379 | 0.049 |
| $R^2$ | | 0.122 | | | 0.124 | |
| $R^2$ Change | | 0.122 | | | 0.002 | |

**$p < 0.05$, **$p < 0.01$

Table 12. Regression Results for Predicting Go Trial Accuracy from DASS-21 Anxiety Subscale Scores, Recent Cannabis Use, and their Interaction.

*Regression Analysis Predicting Go Trial Accuracy from DASS-21 Anxiety Subscale Scores, Recent Cannabis Use, and their Interaction.*

| | Model 1 | | | Model 2 | | |
|---|---|---|---|---|---|---|
| Variable | *B* | *SE B* | *b* | *B* | *SE B* | *b* |
| (Constant) | 94.335 | 1.441 | | 94.354 | 1.451 | |
| DASS-21 Anxiety | -0.055 | 0.101 | -0.062 | -0.076 | 0.121 | -0.085 |
| Recent Cannabis Use | -1.517 | 1.624 | -0.104 | -1.504 | 1.633 | -0.103 |
| AUDIT | -0.001 | 0.186 | -0.001 | 0.002 | 0.187 | 0.001 |
| Age | -0.012 | 0.071 | -0.020 | -0.014 | 0.071 | -0.022 |
| Gender | 2.524 | 1.644 | 0.172 | 2.514 | 1.654 | 0.172 |
| DASS-21 Anxiety & Recent Cannabis Use Interaction | | | | 0.069 | 0.216 | 0.042 |
| $R^2$ | | 0.040 | | | 0.041 | |
| $R^2$ Change | | 0.040 | | | 0.001 | |

**$p < 0.05$, **$p < 0.01$

Table 13. Regression Results for Predicting Go Trial Accuracy from PCL-5 Total Scores, Recent Cannabis Use, and their Interaction

*Regression Analysis Predicting Go Trial Accuracy from PCL-5 Total Scores, Recent Cannabis Use, and their Interaction.*

| | Model 1 | | | Model 2 | | |
|---|---|---|---|---|---|---|
| Variable | *B* | *SE B* | *b* | *B* | *SE B* | *b* |
| (Constant) | 94.396 | 1.447 | | 94.412 | 1.459 | |
| PCL-5 Total | -0.005 | 0.045 | -0.012 | -0.010 | 0.057 | -0.026 |
| Recent Cannabis Use | -1.443 | 1.621 | -0.099 | -1.450 | 1.631 | -0.100 |
| AUDIT | -0.001 | 0.187 | -0.001 | 0.002 | 0.189 | 0.001 |
| Age | -0.008 | 0.071 | -0.013 | -0.009 | 0.072 | -0.015 |
| Gender | 2.369 | 1.639 | 0.162 | 2.344 | 1.657 | 0.160 |
| PCL-5 Total & Recent Cannabis Use Interaction | | | | 0.014 | 0.091 | 0.022 |
| $R^2$ | | 0.037 | | | 0.037 | |
| $R^2$ Change | | 0.037 | | | < 0.001 | |

**$p < 0.05$, **$p < 0.01$

Table 14. Regression Results for Predicting Go Trial RT from DASS-21 Anxiety Subscale Scores, Recent Cannabis Use, and their Interaction

*Regression Analysis Predicting Go Trial RT from DASS-21 Anxiety Subscale Scores, Recent Cannabis Use, and their Interaction.*

| | Model 1 | | | Model 2 | | |
|---|---|---|---|---|---|---|
| Variable | *B* | *SE B* | *b* | *B* | *SE B* | *b* |
| (Constant) | 584.614 | 14.609 | | 585.128 | 14.645 | |
| DASS-21 Anxiety | 0.266 | 1.023 | 0.028 | -0.300 | 1.219 | -0.031 |
| Recent Cannabis Use | 10.324 | 16.457 | 0.067 | 10.660 | 16.489 | 0.069 |
| AUDIT | -1.771 | 1.887 | -0.098 | -1.680 | 1.893 | -0.093 |
| Age | 2.371 | 0.717 | 0.358 | 2.326 | 0.720 | 0.351 |
| Gender | -3.716 | 16.663 | -0.024 | -3.994 | 16.693 | -0.026 |
| DASS-21 Anxiety & Recent Cannabis Use Interaction | | | | 1.869 | 2.181 | 0.108 |
| $R^2$ | | 0.123 | | | 0.131 | |
| $R^2$ Change | | 0.123 | | | 0.008 | |

**$p < 0.05$, **$p < 0.01$

Table 15. Regression Results for Predicting Go Trial RT from PCL-5 Total Scores, Recent Cannabis Use, and their Interaction

*Regression Analysis Predicting Go Trial RT from PCL-5 Total Scores, Recent Cannabis Use, and their Interaction.*

| | Model 1 | | | Model 2 | | |
|---|---|---|---|---|---|---|
| Variable | *B* | *SE B* | *b* | *B* | *SE B* | *b* |
| (Constant) | 583.876 | 14.642 | | 583.778 | 14.769 | |
| PCL-5 Total | -0.090 | 0.451 | -0.021 | -0.056 | 0.574 | -0.013 |
| Recent Cannabis Use | 9.903 | 16.401 | 0.064 | 9.942 | 16.508 | 0.064 |
| AUDIT | -1.755 | 1.888 | -0.097 | -1.774 | 1.911 | -0.098 |
| Age | 2.322 | 0.721 | 0.351 | 2.328 | 0.728 | 0.351 |
| Gender | -2.206 | 16.584 | -0.014 | -2.051 | 16.766 | -0.013 |
| PCL-5 Total & Recent Cannabis Use Interaction | | | | -0.087 | 0.920 | -0.013 |
| $R^2$ | | 0.123 | | | 0.123 | |
| $R^2$ Change | | 0.123 | | | < 0.001 | |

**$p < 0.05$, **$p < 0.01$

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## Appendix A

## Figure Captions

**Figure 1.** DASS-21 anxiety subscale scores for behavioral (**A**) and fMRI (**B**) samples. Scores on the DASS-21 anxiety subscale did not differ between groups for the behavioral sample, t(85) = 0.660, p = 0.511, d = 0.143, BF10 = 0.273 (**A**), or the fMRI sample, t(64) = 0.796, p = 0.429, d = 0.196, BF10 = 0.330 (**B**).

**Figure 2.** PCL-5 total scores for behavioral (**A**) and fMRI (**B**) samples. Scores on the PCL-5 did not differ between groups for the behavioral sample, $t(85) = -0.060$, $p = 0.953$, $d = 0.013$, $BF_{10} = 0.226$ (**A**), or the fMRI sample, $t(64) = 0.433$, $p = 0.666$, $d = 0.107$, $BF_{10} = 0.273$ (**B**).

**Figure 3.** AUDIT total scores for behavioral (**A**) and fMRI (**B**) samples. Scores on the AUDIT did differ between groups for the behavioral sample, $t(85) = -0.016$, $p = 0.988$, $d = 0.003$, $BF_{10} = 0.226$ (**A**), or the fMRI sample, $t(64) = 0.900$, $p = 0.372$, $d = 0.222$, $BF_{10} = 0.356$ (**B**).

**Figure 4.** General distress scores for behavioral (**A**) and fMRI (**B**) samples. Scores on the AUDIT did differ between groups for the behavioral sample, $t(85) = 0.326$, $p = 0.745$, $d = 0.070$, $BF_{10} = 0.236$ (**A**), or the fMRI sample, $t(64) = 0.663$, $p = 0.510$, $d = 0.163$, $BF_{10} = 0.304$ (**B**).

Figure 1. DASS-21 Anxiety Subscale Scores

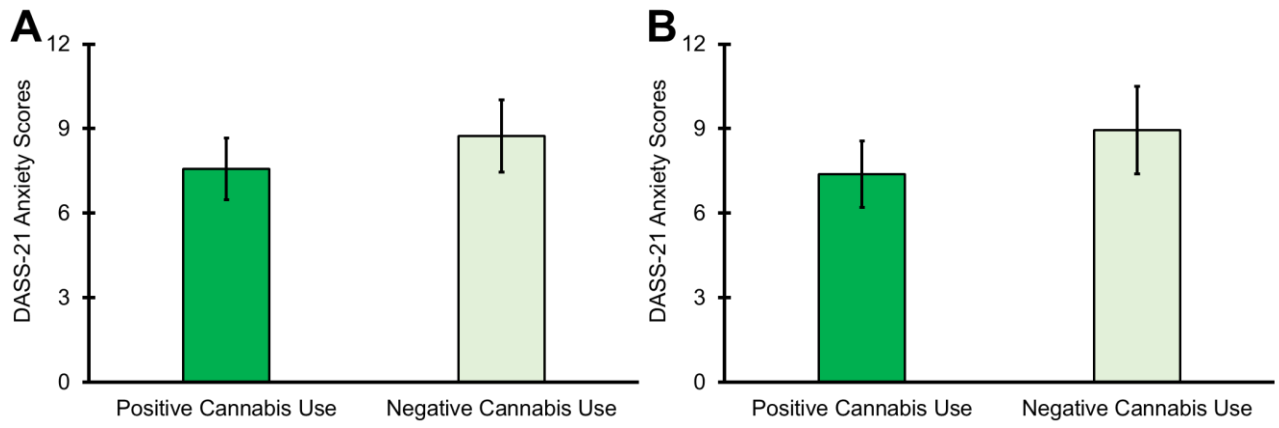


Figure 2. PCL-5 Total Scores

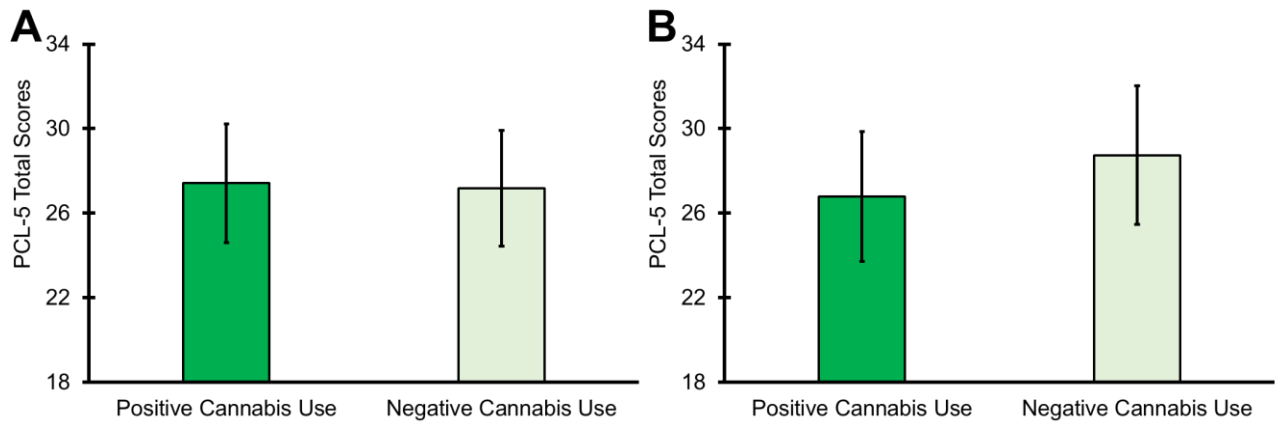


Figure 3. AUDIT Total Scores

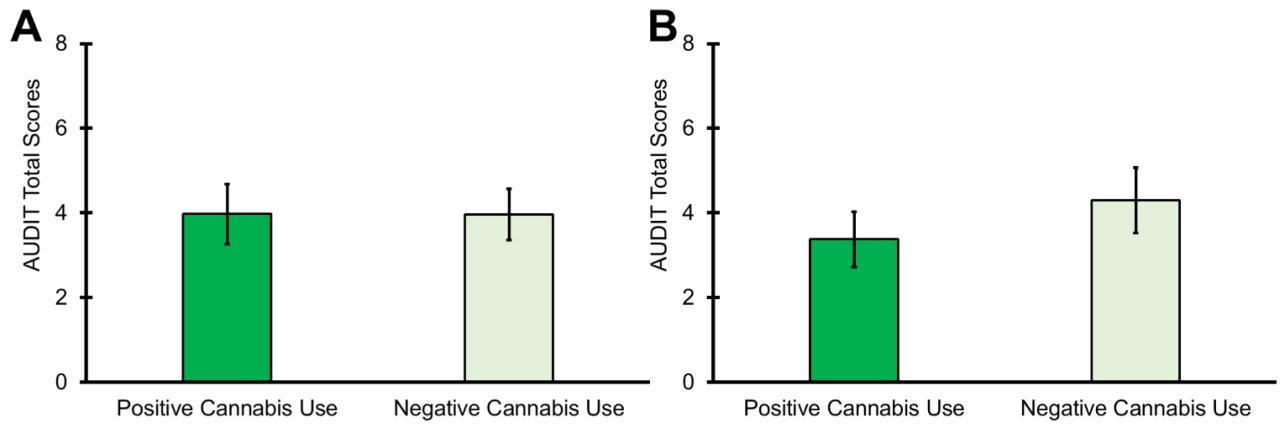
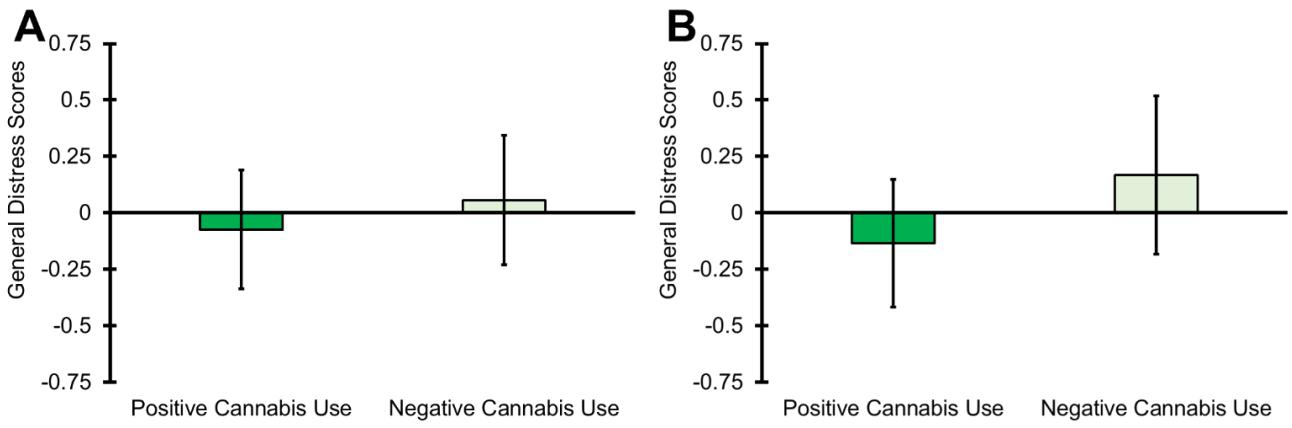


Figure 4. General Distress Scores



# Appendix B

## Table Captions

**Table 1.** Frequencies of self-reported cannabis use within the past month for the behavioral and fMRI samples.

Table 1. Frequencies of Self-Reported Cannabis Use Within the Past Month

*Frequencies of Self-Reported Cannabis Use Within the Past Month*

| Sample | Never | *1-3 Times* | *3-5 Times* | 5-10 Times | 10-25 Times | Every Day or Almost Everyday | More Than Once Per Day |
|---|---|---|---|---|---|---|---|
| Behavioral Analyses | 27 (19.4%) | 1 (0.7%) | 1 (0.7%) | 2 (1.4%) | 3 (2.2%) | 4 (2.9%) | 3 (2.2%) |
| fMRI Analyses | 21 (15.1%) | 2 (1.4%) | 0 (0%) | 1 (0.7%) | 3 (2.2%) | 4 (2.9%) | 2 (1.4%) |

## Figure Captions

**Figure 1.** Behavioral sample consistency of self-reported cannabis use within the past month with urine analyses. Participants' self-reported cannabis use in the behavioral sample were largely consistent with their urine analyses (**A**). Both the recent negative (**B**) and positive (**C**) cannabis use groups also showed high consistency between these measures of recent cannabis use.

**Figure 2.** fMRI sample consistency of self-reported cannabis use within the past month with urine analyses. Participants' self-reported cannabis use in the fMRI sample were largely consistent with their urine analyses (**A**). Both the recent negative (**B**) and positive (**C**) cannabis use groups also showed high consistency between these measures of recent cannabis use.

Figure 1. Behavioral Sample Consistency of Self-Reported Cannabis Use Within the Past Month with Urine Analyses

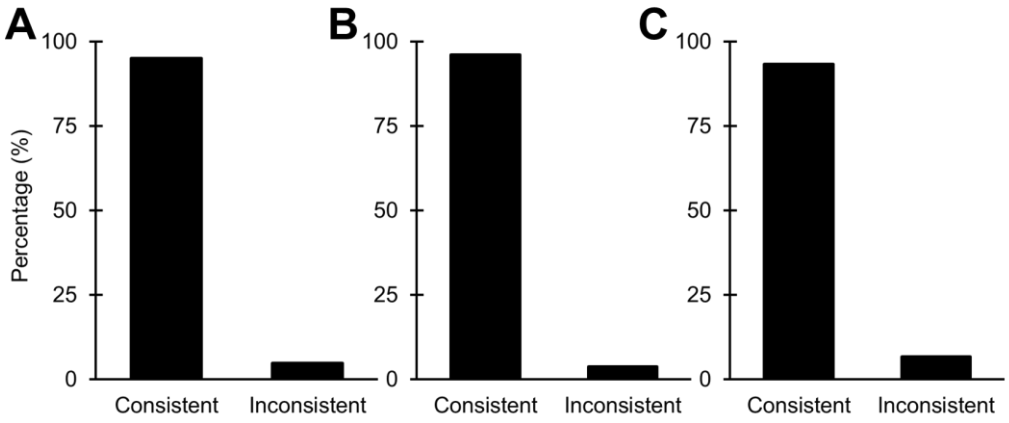
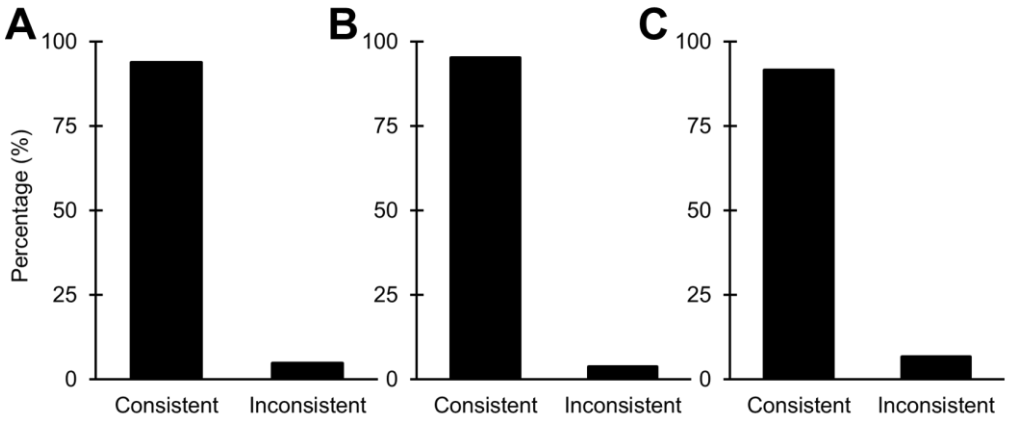


Figure 2. fMRI Sample Consistency of Self-Reported Cannabis Use Within the Past Month with Urine Analyses



## Appendix C

## Figure Captions

**Figure 1.** Total days cigarettes were smoked in the past month for behavioral (**A**) and fMRI (**B**) samples. Reported days cigarettes were smoked in the past month did not differ between cannabis use groups for the behavioral sample, $t(18.75) = -2.055$, $p = 0.054$, $d = 0.728$, $BF_{10} = 1.797$ (**A**), or the fMRI sample, $t(15.57) = -1.710$, $p = 0.107$, $d = 0.673$, $BF_{10} = 0.965$ (**B**).

**Figure 2.** Averaged cigarettes smoked per day in the past month for behavioral (**A**) and fMRI (**B**) samples. Average cigarettes smoked per day in the past month did not differ between cannabis use groups for the behavioral sample, $t(1.05) = 0.926$, $p = 0.518$, $d = 0.844$, $BF_{10} = 0.856$ (**A**), or the fMRI sample, $t(1.07) = 0.958$, $p = 0.505$, $d = 0.935$, $BF_{10} = 1.036$ (**B**).

Figure 1. Total Days Cigarettes were Smoked in the Past Month for Behavioral and fMRI Samples

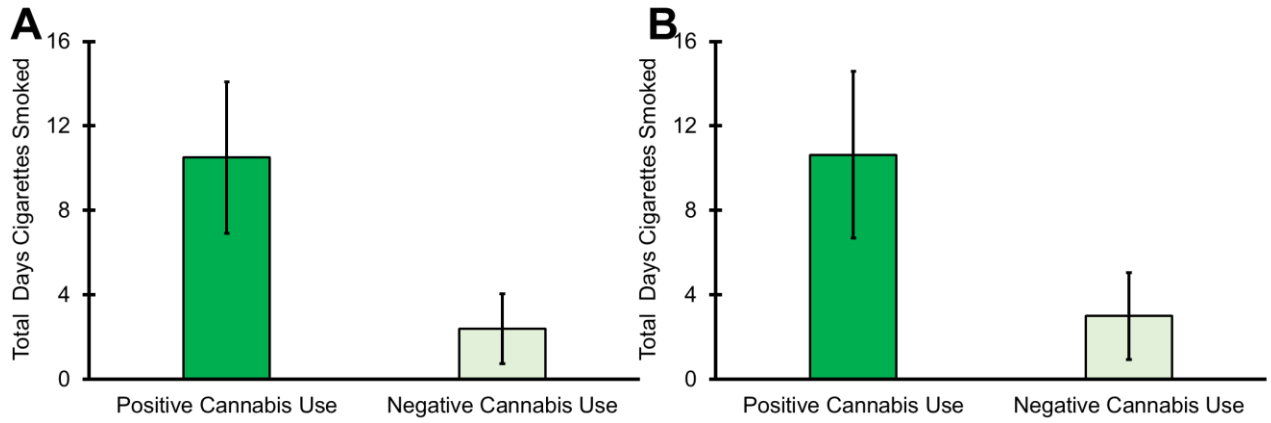
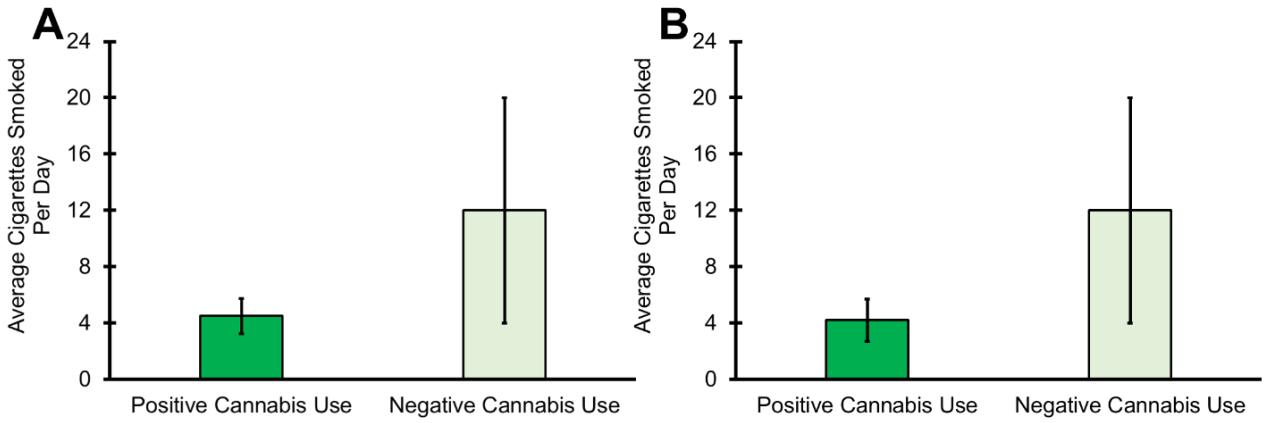


Figure 2. Average Cigarettes Smoked Per Day in the Past Month for Behavioral and fMRI Samples



## Appendix D

## Table Captions

**Table 1.** Proportion of variability from components extracted from the fMRI sample. Results revealed one retained component accounted for approximately 44.79% of variability.

Table 1. Proportion of Variability from Components Extracted from fMRI Sample

*Proportion of Variability from Components Extracted from Full Behavioral Sample*

| Components | Proportion of Variance | Cumulative Proportion | Standard Deviation |
|---|---|---|---|
| Principal Component 1***** | 0.448 | 0.448 | 3.478 |
| Principal Component 2 | 0.073 | 0.521 | 1.405 |
| Principal Component 3 | 0.062 | 0.583 | 1.299 |
| Principal Component 4 | 0.055 | 0.638 | 1.216 |
| Principal Component 5 | 0.046 | 0.684 | 1.116 |
| Principal Component 6 | 0.038 | 0.722 | 1.013 |
| Principal Component 7 | 0.035 | 0.757 | 0.970 |
| Principal Component 8 | 0.031 | 0.788 | 0.912 |
| Principal Component 9 | 0.026 | 0.814 | 0.839 |
| Principal Component 10 | 0.026 | 0.840 | 0.833 |
| Principal Component 11 | 0.021 | 0.861 | 0.759 |
| Principal Component 12 | 0.021 | 0.882 | 0.745 |
| Principal Component 13 | 0.017 | 0.899 | 0.687 |
| Principal Component 14 | 0.017 | 0.916 | 0.669 |
| Principal Component 15 | 0.014 | 0.930 | 0.622 |
| Principal Component 16 | 0.013 | 0.943 | 0.594 |
| Principal Component 17 | 0.011 | 0.954 | 0.537 |
| Principal Component 18 | 0.009 | 0.962 | 0.487 |
| Principal Component 19 | 0.008 | 0.971 | 0.473 |
| Principal Component 20 | 0.006 | 0.977 | 0.405 |
| Principal Component 21 | 0.005 | 0.982 | 0.372 |
| Principal Component 22 | 0.004 | 0.986 | 0.346 |
| Principal Component 23 | 0.004 | 0.990 | 0.314 |
| Principal Component 24 | 0.003 | 0.993 | 0.297 |
| Principal Component 25 | 0.003 | 0.996 | 0.283 |
| Principal Component 26 | 0.003 | 0.999 | 0.273 |
| Principal Component 27 | 0.001 | 1.000 | 0.165 |

*****Retained component

## Appendix E

## Figure Captions

**Figure 1.** General distress and BOLD activity change between Go and Stop trials. Greater general distress scores predicted greater BOLD activity (% change) between Go and Stop trials (i.e., greater activity during Go compared to Stop trials) in the right supplementary motor area.

**Figure 2.** AUDIT scores and BOLD activity change between Go and Stop trials. Greater AUDIT scores predicted greater BOLD activity (% change) between Go and Stop trials (i.e., greater activity during Go compared to Stop trials) in the right middle cingulate cortex (**A**), and the left hippocampus (**B**).

**Figure 3.** Interaction between general distress and recent cannabis use in predicting BOLD activity change between Go and Stop trials. The positive association between general distress scores and BOLD activity (% change) between Go and Stop trials (i.e., greater activity during Go compared to Stop trials) in the right middle occipital gyrus (**A**), and right postcentral gyrus (**B**) increased in individuals testing positive for recent cannabis use.

Figure 1. General Distress and BOLD Activity Change Between Go and Stop Trials

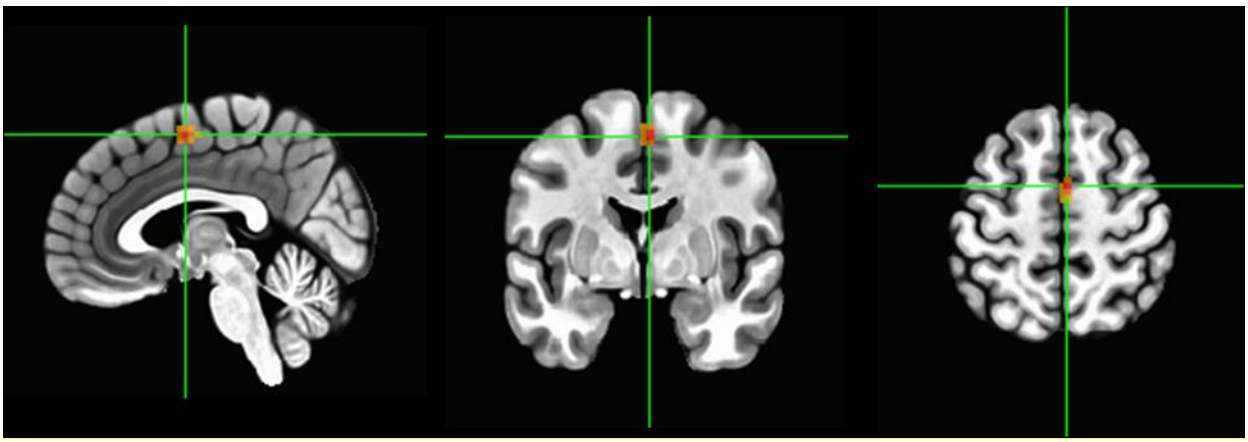


Figure 2. AUDIT Scores and BOLD Change Between Go and Stop Trials

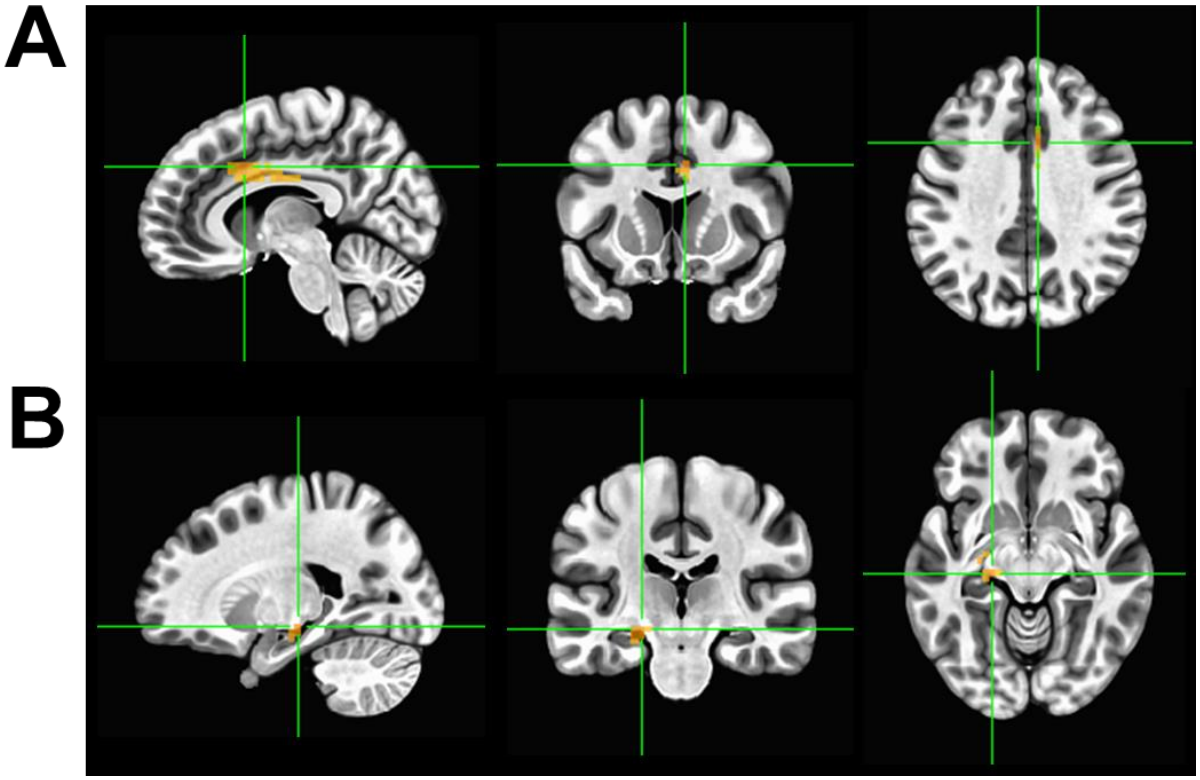
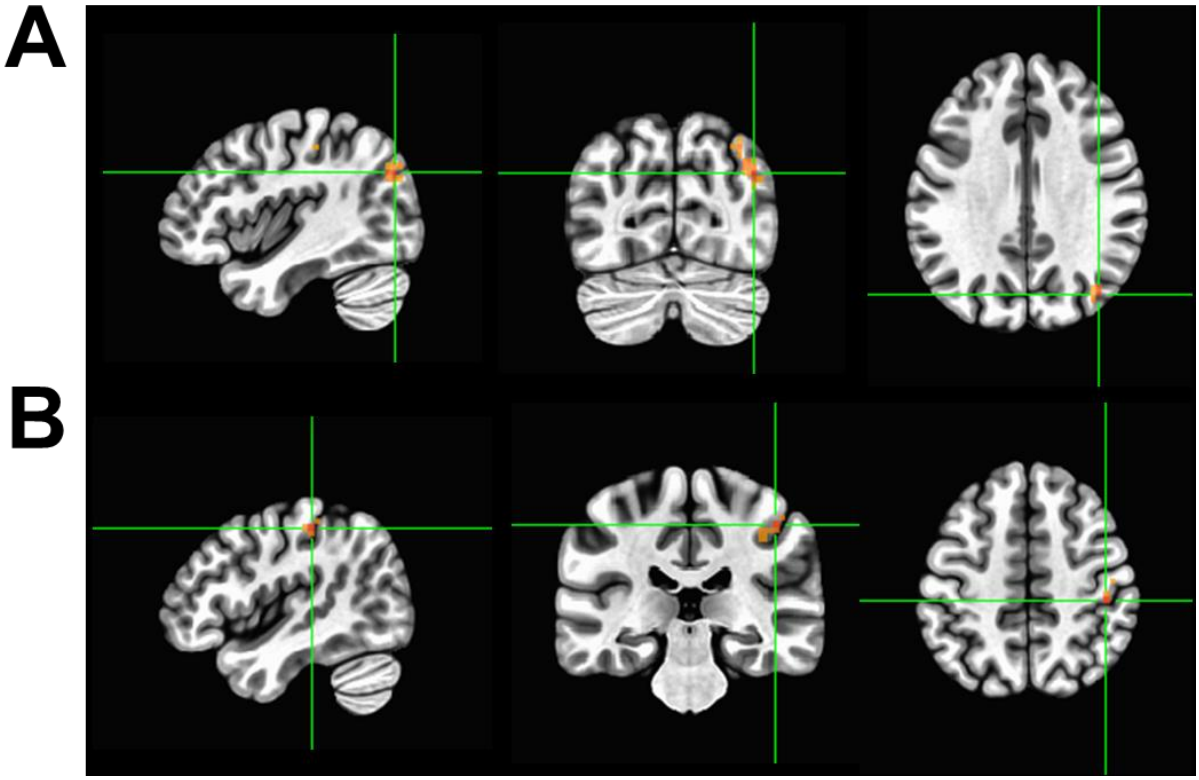


Figure 3. Interaction Between General Distress and Recent Cannabis Use in Predicting BOLD Activity Change Between Go and Stop Trials



## Appendix F

## Figure Captions

**Figure 1.** DASS-21 anxiety subscale scores and BOLD activity change between Go and Stop trials. Greater DASS-21 anxiety subscale scores predicted greater BOLD activity (% change) between Go and Stop trials (i.e., greater activity during Go compared to Stop trials) in the right supplementary motor area**.**

**Figure 2.** AUDIT scores and BOLD activity change between Go and Stop trials. Greater AUDIT scores predicted greater BOLD activity (% change) between Go and Stop trials (i.e., greater activity during Go compared to Stop trials) in the right middle cingulate cortex (**A**), and the left hippocampus (**B**).

**Figure 3.** Interaction between DASS-21 anxiety subscale scores and recent cannabis use in predicting BOLD activity change between Go and Stop Trials. The positive association between DASS-21 anxiety subscale scores and BOLD activity (% change) between Go and Stop trials (i.e., greater activity during Go compared to Stop trials) in the left angular gyrus (**A**), and right post central gyrus (**B**) increased in individuals testing positive for recent cannabis use.

Figure 1. DASS-21 Anxiety Subscale Scores and BOLD Activity Change Between Go and Stop Trials

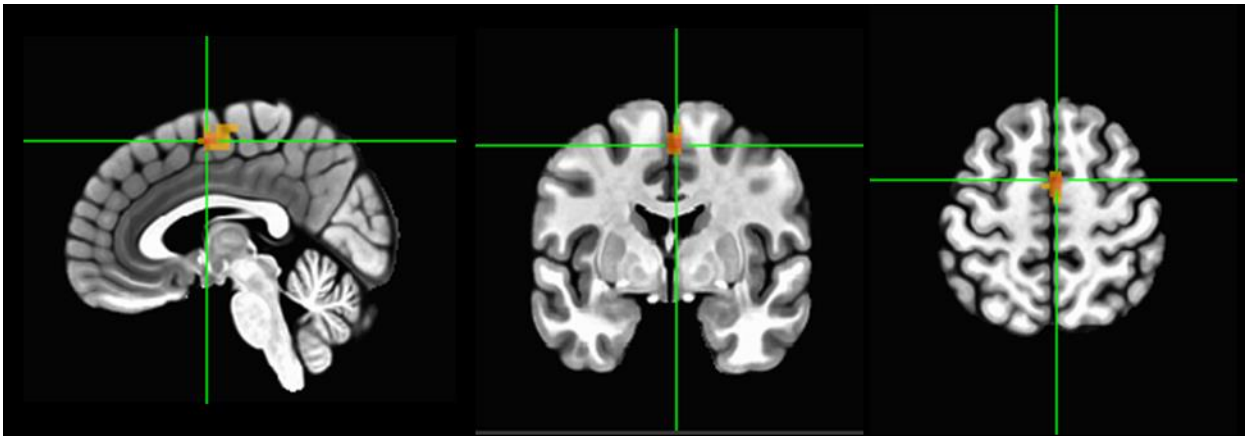


Figure 2. AUDIT Scores and BOLD Activity Change Between Go and Stop Trials

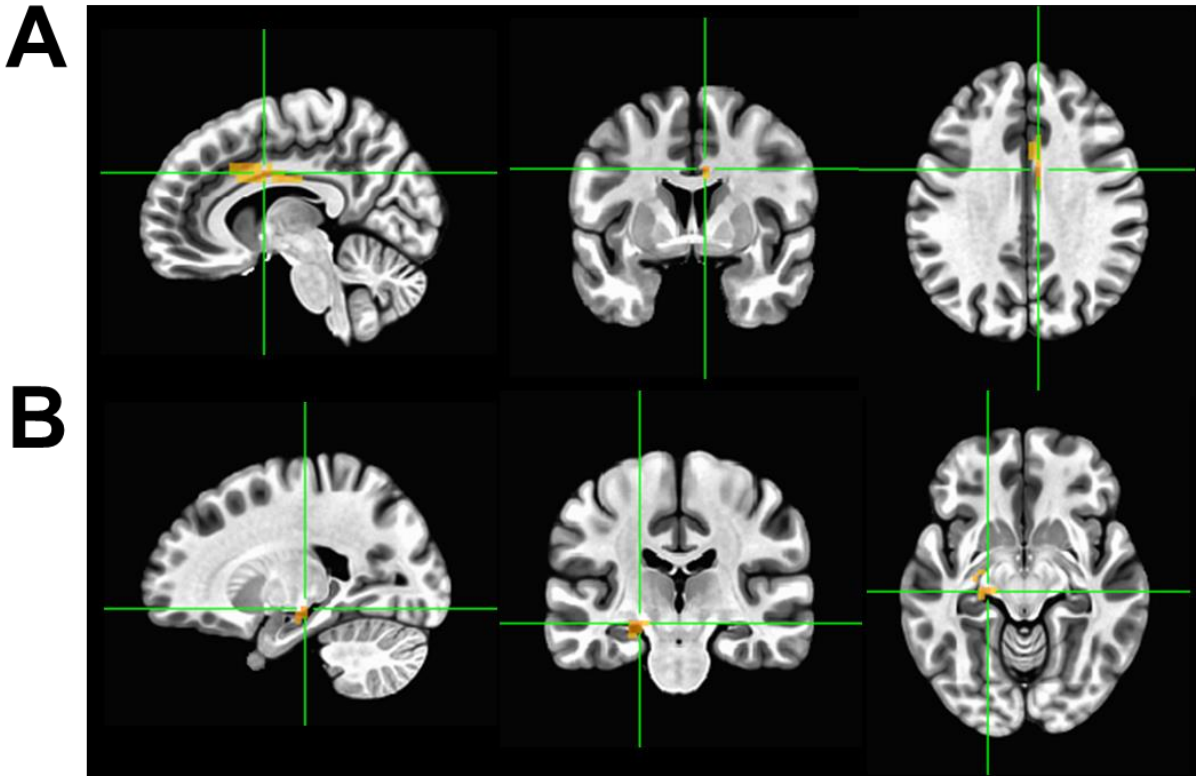
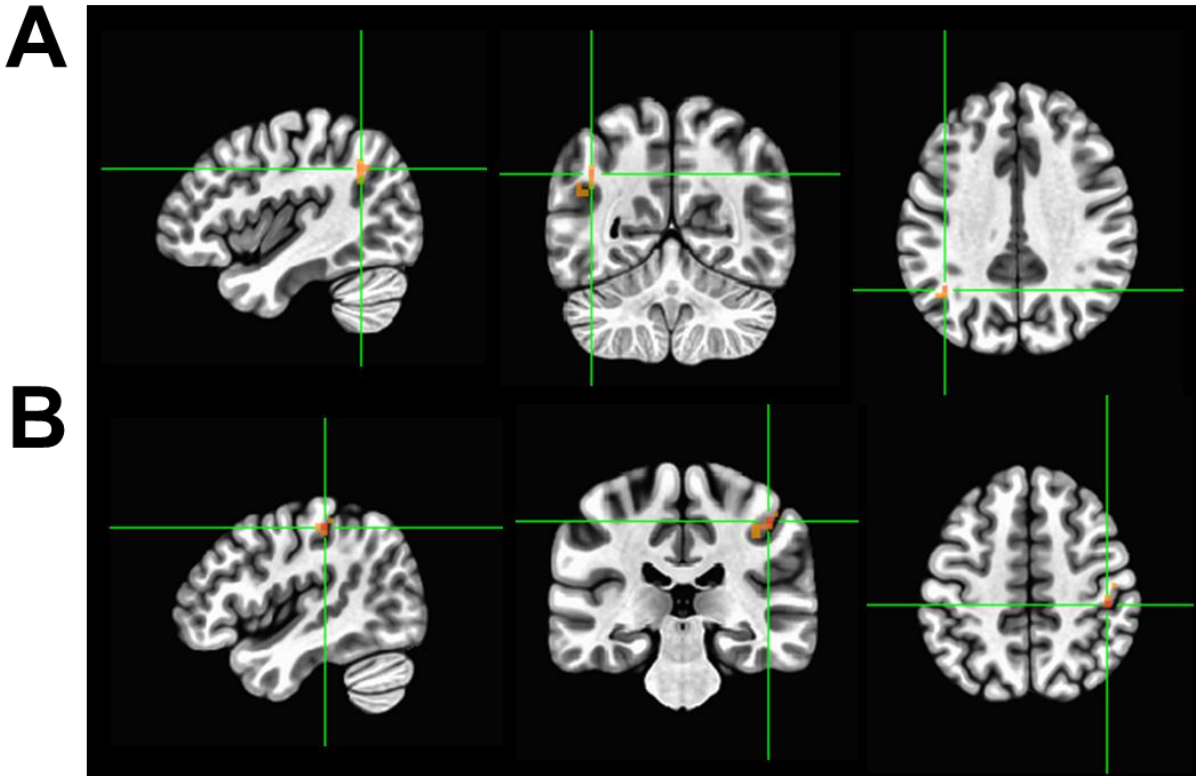


Figure 3. Interaction Between DASS-21 Anxiety Subscale Scores and Recent Cannabis Use in Predicting BOLD Activity Change Between Go and Stop Trials



## Appendix G

## Figure Captions

**Figure 1.** ADUIT scores and BOLD activity change between Go and Stop trials. Greater AUDIT scores predicted greater BOLD activity (% change) between Go and Stop trials (i.e., greater activity during Go compared to Stop trials) in the right middle cingulate cortex (**A**), and the left hippocampus (**B**).

**Figure 2.** Interaction between PCL-5 total scores and recent cannabis use in predicting signal BOLD activity change between Go and Stop trials. The positive association between PCL-5 total scores and BOLD activity (% change) between Go and Stop trials (i.e., greater activity during Go compared to Stop trials) in the right middle occipital gyrus (**A**), right lingual gyrus (**B**), left middle occipital gyrus (**C**), right middle frontal gyrus (**D**), left superior occipital gyrus (**E**), and the left precuneus (**F**) increased in individuals testing positive for recent cannabis use.

Figure 1. AUDIT Scores and BOLD Activity Change Between Go and Stop Trials

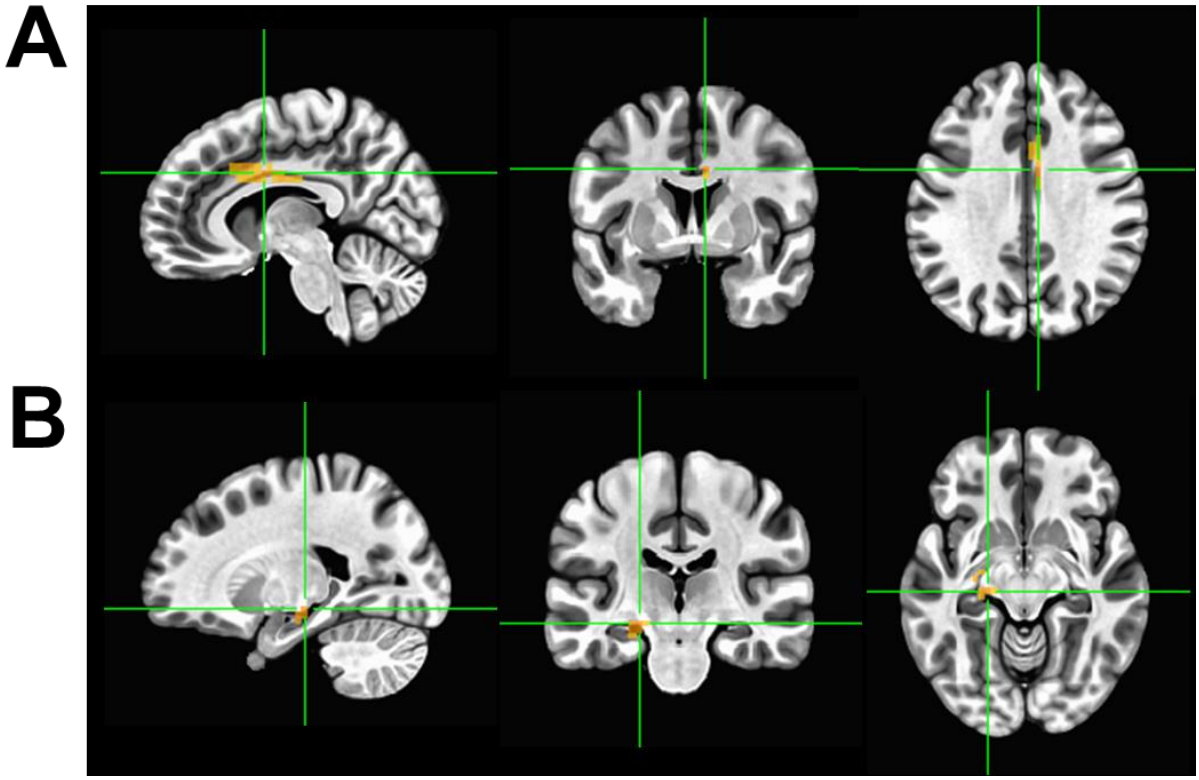
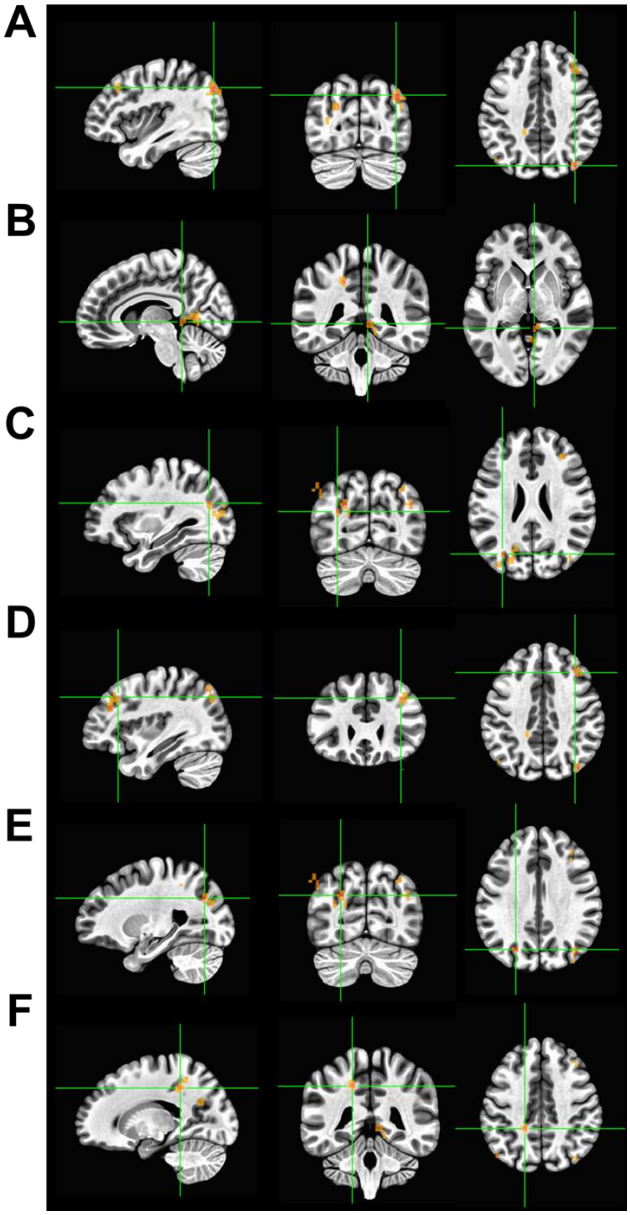


Figure 2. Interaction Between PCL-5 Total Scores and Recent Cannabis Use in Predicting BOLD Activity Change Between Go and Stop Trials



# Richard T. Ward

Department of Psychology | University of Wisconsin – Milwaukee

## EDUCATION

**Ph.D. Psychology** **2017 – 2021**
University of Wisconsin – Milwaukee
Program: Neuroscience
Minor: Quantitative Methods
Advisor: Christine L. Larson
Dissertation: *Neural correlates underlying the interactions between anxiety and cannabis use in predicting motor response inhibition*

**M.A. Clinical Psychology** **2015 –2017**
Ball State University
Advisor: Stephanie L. Simon-Dack
Thesis: *Neural correlates of attentional control theory in high trait anxious individuals*

**B.S. Psychology** **2011 –2015**
Morehead State University
Capstone: *Amphetamine and morphine may produce symptoms of acute withdrawal via a common dopamine-dependent pathway*

## PEER-REVIEWED PUBLICATIONS

Underlined Text = Student Mentee ***** = Shared First Authorship

8. **Ward, R.**, Lotfi, S., Stout, D., Mattson, S., Lee, H. J., & Larson, C. L. (2021). Neutral and threatening distracter word stimuli are unnecessarily stored in working memory but do not differ in their degree of working memory storage. *Biological Psychology*, 108091.

7. Poorhamze, M., Moravvej, F. G., Arabi, M., Shahriari, E., Mehrabi, S., **Ward, R.**, Ahadi, R., & Joghataei, M. T. (2021). The roles of serotonin in neuropsychiatric disorders. *Cellular and Molecular Neurobiology*, 1-22.

6. Lotfi, S., **Ward, R.**, Ayazi, M., Bennett, K., Larson, C. L., Lee, H. J. (2020). The effects of emotional working memory training on worry symptoms and error-related negativity of individuals with high trait anxiety: A randomized controlled study. *Cognitive Therapy and Research*, 1-17

5. Lotfi, S., Rostami, R., Shokoohi-Yekta, M., **Ward**, **R**., Motamed, N., Mathew, A., & Lee, H. J. (2020). Effects of computerized cognitive training for children with dyslexia: An ERP study. *Journal of Neurolinguistics, 55,* 100904.

4. **Ward, R.**, Lotfi, S., Sallmann, H., Lee, H. J., & Larson, C. L. (2020). State anxiety reduces working memory capacity but does not impact filtering cost of neutral distracters. *Psychophysiology, 57*, e13625.

3. **Ward, R.***, Miskovich, T.*, Stout, D., Bennett, K., Lotfi, S., & Larson, C. L. (2019). Reward-related distracters and working memory filtering. *Psychophysiology*, *56*, e13402

2. **Ward, R.**, & Butler, D. L. (2019). An investigation of metacognitive awareness and academic performance in college freshmen. *Education, 139,* 120-126.

1. **Ward, R.**, Smith, S., Kraus, B. T., Allen, A., Moses, M., & Simon-Dack, S. (2018). Alpha band frequency differences between low and high trait anxious individuals. *NeuroReport, 2*, 79-83.

## MANUSCRIPTS UNDER REVIEW & IN REVISION

3. Webb, E., Weis, C., **Ward, R.**, Huggins, A., Fitzgerald, J., Bennett, K., Parisi, E., Miskovich, T., Krukowski, J., deRoon-Cassini, T., Larson, Cl. L. (Under Review). Neural effect of neighborhood disadvantage on response inhibition in acutely traumatically-injured adults. *Cognitive Neuroscience*.

2. Smith, S.*, **Ward, R.***, Martin, H., Gagne, J., & Mills, C. (Under Review). The role of depressive symptomology in text comprehension. *Cognition & Emotion.*

1. Lotfi, S., **Ward, R.,** Mathew, A., Shokoohi-Yekta, M., Rostami, R., Motamed-Yeganeh, N., Larson, C. L., & Lee, H. J. (In Revision). Limited visual working memory capacity in children with dyslexia: An ERP study. *Scientific Studies of Reading*.

## MANUSCRIPTS IN PREPERATION

2. Lotfi, S., **Ward, R.**, Ayazi, M., Larson, C. L., & Lee, H.-J. (In Preparation). Temporal dynamics of stimulus presentation on flanker congruency proportion effect and cognitive control: Eye-gaze and ERP insights.

1. Lotfi, S., **Ward, R.**, Ayazi, M., Larson, C. L., & Lee, H.-J. (In Preparation). Effects of contextual manipulation paradigm on proactive versus reactive distraction filtering: Evidence from a combined EEG and eye-tracking study.

## CONFERENCE SYMPOSIA CHAIRED

1. **Ward, R.** & Larson, C. L. (2021). *Anxiety, working memory, and attentional control: The neural and biological mechanisms instantiating this complex interaction*. Symposium to be given at Society for Psychophysiological Research annual convention. Postponed to 2021 due to COVID-19.

## CONFERENCE TALKS

7. **Ward, R.**, Lotfi, S., Stout, D., Lee, H.J., & Larson, C. L. (2021). *The effects of anxiety on working memory and unnecessary storage of distracters*. Talk to be given at the Society for Psychophysiological Research annual convention. Postponed to 2021 due to COVID-19.

6. Mattson, S., Shaw, C., Montoto, K., Kornkven, J., Siegel, E., Santiago, J., **Ward, R.**, Larson, C. L. (2021). *Effects of differentially conditioned stimuli on working memory processes*. Virtual talk given at the the annual University of Wisconsin – Milwaukee Undergraduate Research Symposium. Milwaukee, WI. Virtual conference due to COVID-19.

5. Santiago, J., **Ward, R.**, & Larson, C. L. (2020). *How does attention and anxiety interact to predict threat word filtering*. Virtual talk given at the annual McNair Student Symposium at the University of Wisconsin – Milwaukee. Virtual conference due to COVID-19.

4. **Ward, R.**, Mattson, S., Kornkven, J., Stout, D., Lotfi, S., Lee, H. J., & Larson, C. L. (2020). *Individual differences in filtering threat-related distracter words from working memory*. Talk accepted at the annual Association for Graduate Students in Psychology symposium at the University of Wisconsin – Milwaukee. Milwaukee. WI. Cancelled due to COVID-19

3. Lagunez-Garcia, J., **Ward, R.**, & Larson, C. L. (2019). *The relationship between anxiety and implicit racial bias*. Talk given at the annual McNair Student Symposium at the University of Wisconsin – Milwaukee. Milwaukee, WI.

2. Allen, A., Smith, S., **Ward, R.**, & Simon-Dack, S. (2018). *Mild traumatic brain injury, working memory, and theta synchronization*. Talk given at the 2018 Student Symposium at Ball State University. Muncie, IN.

1. **Ward, R**., Ellis, A., Blackledge, J.T., Rollins, E., & Crager, K. (2013). *Initial validation of the fusion and inflexible responding scale*. Talk given at the Kentucky Academy of Science annual convention. Morehead, KY.

## CONFERENCE POSTER RESENTATIONS

39. Santiago, J., Mattson, S., Kornkven, J., Krause, A., Ramczyk, B., Lagunez-Garcia, J., Merrill, G., Shaw, C., Siegel, E., Athan, A., Schaefer, I., Thao, N., Steffes, M., **Ward, R.**, & Larson, C. L. (2021). *How do neutral and threatening distracter words influence attention and working memory?* Poster presented at the annual University of Wisconsin – Milwaukee Undergraduate Research Symposium. Milwaukee, WI. Virtual conference due to COVID-19.

38. Mattson, S., **Ward, R.**, Siegel, E., Kornkven, J., Montoto, K., & Larson, C. L. (2021). *Working memory performance for differentially-conditioned stimuli*. Poster presented at the Midwestern Psychological Association. Virtual Conference due to COVID-19.

37. **Ward, R.**, Lotfi, S., Stout, D., Mattson, S., Lee, H. J., Larson, C. L. (2020). *No differences in attentional bias and working memory filtering between threatening and neutral distracter words*. Poster presented at the Society for Psychophysiological Research annual convention. Virtual conference due to COVID-19.

36. Kornkven, K., Mattson, S., Krause, A., Ramczyk, B., Lagunez-Garcia, J., Merrill, G., Shaw, C., Siegel, E., Athan, A., Schaefer, I., Steffes, M., **Ward, R.**, & Larson, C. L. (2020). *Are threatening words inefficiently filtered from gaining access to working memory?* Poster presented at the Wisconsin Psychological Association annual convention. Virtual conference due to COVID-19.

35. Mattson, S., Kornkven, J., Krause, A., Lagunez-Garcia, J., Merrill, G., Shaw, C., Siegel, E., Athan, A., Schaefer, I., Steffes, M., Thao, N., Santiago, J., **Ward, R.**, & Larson, C. L. (2020). *Filtering efficiency of threatening words in working memory*. Poster presented at the annual University of Wisconsin – Milwaukee Undergraduate Research Symposium. Milwaukee, WI. Virtual conference due to COVID-19.

34. Lagunez-Garcia, J., **Ward, R.**, Pederson, W., & Larson, C. L. (2020). *Individual factors associated with anxiety-induced stereotype-specific deficits in cognitive control*. Poster presented at the annual University of Wisconsin – Milwaukee Undergraduate Research Symposium. Milwaukee, WI. Virtual conference due to COVID-19.

33. Mattson, S., Kornkven, J., Krause, A., Ramczyk, B., Lagunez-Garcia, J., Merrill, G., Shaw, C., Siegel, E., Athan, A., Schaefer, I., Steffes, M., **Ward, R.**, & Larson, C. L. (2020). *Working memory filtering efficiency of threatening words*. Poster presented at the Association of Psychological Science annual convention. Chicago, IL. Virtual conference due to COVID-19.

32. Lotfi, S., **Ward, R.**, Rech, M., Ayazi, M., Larson, C. L., & Lee, H. J. (2020). *Does threat of shock modulate reactive and proactive cognitive control?* Poster presented at the Cognitive Neuroscience Society annual convention. Virtual conference due to COVID-19.

31. **Ward, R.**, Mattson, S., Kornkven, J., Lotfi, S., Lee, H. J., & Larson, C. L. (2020). *Aversive distracter words and working memory filtering*. Poster presented at the Cognitive Neuroscience Society annual convention. Virtual conference due to COVID-19.

30. Lagunez-Garcia, J., **Ward, R.**, & Larson, C. L. (2019). *The relationship between anxiety and implicit racial bias*. Poster presented at Baylor University McNair Research Conference. Baylor, TX.

29. **Ward, R.**, Sallmann, H., Ginter, C., Lotfi, S., Lee, H. J., & Larson, C. L. (2019). *Threat of shock-induced anxiety reduces working memory capacity*. Poster presented at the Society for Psychophysiological Research annual convention. Washington, D.C..

28. Lotfi, S., Rech, M., **Ward, R.**, Ayazi, M., Larson, C. L. & Lee, H. J. (2019). *Proactive versus reactive distraction filtering under threatening conditions: Evidence from a combined EEG and eye-tracking study.* Poster presented at the Society for Psychophysiological Research annual convention. Washington, D.C..

27. Lotfi, S., Rech, M, **Ward, R.**, Larson, C. L. & Lee, H. J. (2019). *State anxiety and cognitive control: Evidence from a combined study of shock paradigm, eye-tracking, and EEG.* Poster presented at the 33rd annual meeting of the Society for Research in Psychopathology. Buffalo, NY.

26. Lotfi, S., **Ward, R.**, Larson, C. L., & Lee, H. J. (2019). *Proactive versus reactive distraction filtering under threat of shock: EEG and eye-tracking study.* Poster presented at the 25th Wisconsin Symposium on Emotion. Madison, WI.

25. Olsem, M., Vanderbilt, E., **Ward, R.**, Lotfi, S., & Larson, C. L. (2019). *The neuroscience of cognitive control in anxiety*. Poster presented at the annual UR@UWM Research Symposium. Milwaukee, WI.

24. Sallmann, H., Mattson, S., Ginter, C., Sendek, M., Krause, A., Kornkven, J., Merrill, G., Ramczyk, B., Grimm, S., **Ward, R.**, & Larson, C. L. (2019). *Threat of shock reduces working memory capacity: An ERP study*. Poster presented at the annual University of Wisconsin – Milwaukee Undergraduate Research Symposium. Milwaukee, WI.

23. Sallmann, H., Ginter, C., Sendek, M., Krause, A., Kornkven, J., Mattson, S., Merrill, G., Grimm, S., Ramczyk, B., **Ward, R.**, & Larson, C. L. (2019). *Working memory capacity is reduced under threatening conditions: An ERP study*. Poster presented at National Conferences on Undergraduate Research annual convention. Kennesaw, GA.

22. Smith, S., Wittman, S., Robinson, C., Johnson, J., Grzywana, J., Myers, A., Seaman, A., Taylor, A., Price, K., Tatum, A., **Ward, R.**, & Simon-Dack, S. (2019). *An ERP analysis comparing visual and verbal long-term memory mechanisms through access-based forgetting*. Poster presented at the University of New Hampshire Graduate Student Research Conference. Durham, NH.

21. **Ward, R.**, Sallmann, H., Ginter, C., Lotfi, S., Lee, H. J., & Larson C.L. (2019). *Reduced working memory capacity under threatening context*. Poster presented at the Cognitive Neuroscience Society annual convention. San Francisco, CA.

20. Lotfi, S., Burdis, C., Rech, M., Dommer, L., Michalki, C., Anhalt, E., **Ward, R.**, Larson, C. L., & Lee, H. J. (2019) *Proactive versus reactive distraction filtering: Evidence from a combined EEG and eye-tracking study*. Poster presented at the Cognitive Neuroscience Society annual convention. San Francisco, CA.

19. Smith, S., Wittman, S., Robinson, C., Johnson, J., Grzywana, J., Myers, A., Seaman, A., Taylor, A., Price, K., Tatum, A**., Ward, R.**, & Simon-Dack, S. (2019). *An ERP analysis comparing visual and verbal long-term memory mechanisms through access-based forgetting*. Poster presented at the Cognitive Neuroscience Society annual convention. San Francisco, CA.

18. **Ward, R.**, Sallmann, H., Ginter, C., Lotfi, S., Lee, H. J., & Larson, C. L. (2018). *Working memory capacity under threat of shock*. Poster presented at the Department of Psychology Pre-SfN Poster Session annual convention. Milwaukee, WI.

17. Allen, A., Smith, S., **Ward, R.**, & Simon-Dack, S. (2018). *The effects of mild traumatic brain injury on theta synchronization and working memory: A spectral analysis.* Poster presented at the Department of Psychological Science Poster Session annual convention. Muncie, IN.

16. **Ward, R.**, Mull, A., Benbow, A., & Diaz, A. (2016). *The role of temperament and metacognitive strategies in internalizing disorders*. Poster presented at the Occasional Temperament Conference annual convention. Seattle, WA.

15. Diaz, A., **Ward, R.**, & Benbow, A. (2016). *Anxiety and depression in first-semester freshmen: The role of temperament and social support*. Poster presented at the Occasional Temperament Conference annual convention. Seattle, WA.

14. Cole, Z., Butler, D., **Ward, R.**, Di Iorio, A., & Samaan, L. (2016). *Comparing recall for sentences formatted in a concept map, outline, or list*. Poster presented at the American Psychological Association annual convention. Denver, CO.

13. **Ward, R.**, Kraus, B. T., Cadle, C., Matsen, J., Kanazeh, T., Leichty-Wireman, Z., & Holtgraves, T. (2016). *Comprehending conversational scalar implicatures: An ERP study*. Poster presented at the Association for Psychological Science annual convention. Chicago, IL.

12. Kraus, B. T., Cadle, C., Roviso, N., **Ward, R.**, Walter, Z., Rohaly, T., Sheets, J., Price, D., & Simon-Dack, S. (2016). *EEG alpha band power and creative task ideation*. Poster presented at the Association for Psychological Science annual convention. Chicago, IL.

11. Butler, D., Cole, Z., **Ward, R.**, Di Iorio, A., & Samaan, L. (2016). *Recall as a function of study and test format: understanding the role of organization in memory*. Poster presented at the Association for Psychological Science annual convention. Chicago, IL.

10. Cole, Z., Butler, D., **Ward, R.**, Di Iorio, A., & Samaan, L. (2016). *The effect of organization on recall using concept map tests*. Poster presented at the Department of Psychological Science Poster Session annual convention. Muncie, IN

9. **Ward, R.**, Diaz, A., & Benbow, A. (2016). *Depression and anxiety in first semester freshmen*. Poster presented at the Midwest Graduate Research Symposium annual convention. Toledo, OH.

8. **Ward, R.**, Di Iorio, A., Leithner, S., Radabaugh, M., & Butler, D. (2016). *An investigation of metacognition, academic performance, and note-taking strategies*. Poster presented at the Student Symposium annual convention. Muncie, IN.

7. White, W., **Ward, R.**, & White, I. (2015). *Amphetamine and morphine may produce aspects of acute withdrawal by initially affecting a common pathway*. Poster Presented at the Society for Neuroscience annual convention. Chicago, IL.

6. **Ward, R.**, Abbott, Z., Morris, K., & White, W. (2015). *Amphetamine and morphine may produce symptoms of acute withdrawal via a common dopamine-dependent pathway*. Poster Presented at the Celebration of Student Scholarship annual convention. Morehead, KY.

5. Crisp, K., Stark, J., Huff, J., Fletcher, A., Caudill, S., Abbott, Z., Rice, A., Dennie, T., Baker, S., Singleton, J., **Ward, R.**, Hobert, C., & White, I. (2015). *2015 Brain awareness program: Brain drawing contest*. Poster presented at the Celebration of Student Scholarship annual convention. Morehead, KY.

4. **Ward, R.**, Blackledge, J.T., Alshafie, G., Crager, K., Ramos, W., Nichelson, K., & Rollins, E. (2014). *Targeting psychological distress with a brief defusion intervention*. Poster presented at the Kentucky Academy of Science annual convention. Lexington, KY.

3. Lewis, A., Mosley, A., **Ward, R.**, & White, W. (2014). *Dopamine d1 receptor involvement in the elicitation of symptoms of acute withdrawal by morphine in rats.* Poster presented at the Kentucky Academy of Science annual convention. Lexington, KY.

2. Ellis, A., Crager, K., Blackledge, J.T., & **Ward**, **R.** (2014). *Initial validation of the hexaflex process assessment scale.* Poster presented at the Kentucky Academy of Science annual convention. Lexington, KY.

1. Huff, J., Abbott, Z., Elmlinger, D., Banks, C., Baldwin, A., Edie, E., Fitzpatrick, C., Hall, C., Price, B., Rich, A., Sexton, A., Smith, M., Waddell, E., **Ward, R.**, Barber, J., & White, I. (2014). *2014 Brain drawing contest: Regional brain awareness program.* Poster presented at the Celebration of Student Scholarship annual convention. Morehead, KY.

## RESEARCH GRANTS

| | |
|---|---|
| Sigma Xi Grants in Aid of Research (**$910**) | **2020** |
| Society for Psychophysiological Research Training Grant **($1,800**) | **2018** |
| Ball State University Hollis Fund Grant (**$500**) | **2016** |

## TRAVEL GRANTS

| | |
|---|---|
| Association of Clinical and Cognitive Neuroscience Travel Grant (**$1,500**) | **2020** |
| Association of Clinical and Cognitive Neuroscience Travel Grant (**$1,342**) | **2019** |
| Association of Graduate Students in Psychology Travel Grant (**$3,081**) | **2019** |
| University of Wisconsin – Milwaukee Graduate Student Travel Award (**$180**) | **2019** |
| Association of Clinical and Cognitive Neuroscience Travel Grant (**$1,800**) | **2019** |
| Ball State University Student Government Association Leadership Fund (**$400**) | **2017** |

## FELLOWSHIPS & SCHOLARSHIPS

| | |
|---|---|
| R1 Distinguished Dissertator Fellowship (UWM, **$16,500**) - Declined | **2021 – 2022** |
| Cialdini Fellowship (UWM, **$20,000** – $5,000 per a year) | **2017 – 2021** |
| Distinguished Graduate Student Fellowship (UWM, **$15,000**) | **2019 – 2020** |
| Summer Graduate Research Fellowship (UWM, **$4,266** | **2018** |
| Graduate Merit Fellowship (BSU, **$3,000**) | **2015 – 2016** |
| Kentucky Education Excellence Scholarship (MSU, **$4,000**) | **2011 – 2015** |
| Eagle Excellence Award KEESx2 Scholarship (MSU, **$4,000**) | **2011 – 2015** |

## HONORS & AWARDS

| | |
|---|---|
| Neuromatch Academy Interactive Session (Virtual) - Declined | **2020** |
| 3-Minute Thesis Finalist (UWM) | **2020** |
| Graduate School Dean's Academic Excellence (BSU) | **2017** |
| Recognized Graduate Student Award (BSU) | **2016, 2017** |
| Magna Cum Laude (MSU) | **2015** |
| Outstanding Junior in Psychology Award (MSU) | **2014** |
| College of Science and Technology Dean's List (MSU) | **2013, 2014, 2015** |
| Morehead State University Dean's List (MSU) | **2013, 2014, 2015** |

## AD HOC JOURNAL REVIEWER

*Behavioural Brain Research*
*Biological Psychiatry* (under supervision)
*Cognitive, Affective, and Behavioral Neuroscience*
*Cognition and Emotion*
*Journal of Experimental Psychology: Learning, Memory, and Cognition* (under supervision)
*Neuropsychopharmacology* (under supervision)
*Neuropsychologia*
*Psychophysiology*
*Scientific Reports*

## PROFESSIONAL SERVICE

| | |
|---|---|
| Society for Psychophysiological Research Student Interests committee | **2020 – Present** |
| Cognitive Neuroscience Society Trainee Association committee | **2018 – Present** |
| Cognitive Neuroscience Society Professional Panel guest writer <**Post**> | **2021** |
| EEG and ERP Workshop Seminar Series (UWM) host | **2021** |
| Cognitive Neuroscience Society Professional Panel guest writer <**Post**> | **2020** |

## PROFESSIONAL ORGANIZATIONS

| | |
|---|---|
| Cognitive Neuroscience Society | **2018 – Present** |
| Society for Psychophysiological Research | **2018 – Present** |
| Association for Psychological Science | **2015 – 2018** |
| Kentucky Academy of Science | **2013 – 2015** |

## UNIVERSITY ORGANIZATIONS

| | |
|---|---|
| Data Science Club (UWM) | **2020 – 2021** |
| Association of Clinical and Cognitive Neuroscience (UWM) | **2018 – 2021** |
| Cognition, Learning, Attention & Memory Society (UWM) | **2018 – 2021** |
| Association of Graduate Students in Psychology (UWM) | **2017 – 2021** |
| Molecular Basis of Memory (UWM) | **2017 – 2020** |
| Psi Chi Psychology Honor Society (MSU) | **2014 – 2015** |
| Psi Lambda (MSU) | **2013 – 2015** |

## RESEARCH EXPERIENCE

**University of Wisconsin – Milwaukee, Department of Psychology**

| ***Lab*** | ***Role*** | ***PI*** | ***Duration*** |
|---|---|---|---|
| Affective Neuroscience Laboratory | Graduate Student | Christine L. Larson | 2018 – 2021 |
| Molecular & Behavioral Neuroscience Laboratory | Graduate Student | Fred Helmstetter | 2017 – 2018 |

**Ball State University, Department of Psychological Science**

| ***Lab*** | ***Role*** | ***PI*** | ***Duration*** |
|---|---|---|---|
| Cognition Laboratory | Graduate Student | Darrell Butler | 2015 – 2017 |
| Psychophysiological Attention, Cognition, & Emotion Laboratory | Graduate Student | Stephanie Simon-Dack | 2015 – 2017 |
| Language Processing Laboratory | Graduate Student | Thomas Holtgraves | 2015 – 2016 |

**Indiana University – Purdue University Indianapolis, Department of Psychology**

| ***Lab*** | ***Role*** | ***PI*** | ***Duration*** |
|---|---|---|---|
| Behavioral Neuroscience of Addiction Laboratory | Research Intern | Cristine Czachowski | 2016 |

**Morehead State University, Department of Psychology**

| ***Lab*** | ***Role*** | ***PI*** | ***Duration*** |
|---|---|---|---|
| Behavioral Neuroscience Laboratory | Undergraduate Student | Wesley White | 2014 – 2015 |
| Acceptance and Commitment Therapy Behavior Laboratory | Undergraduate Student | John T. Blackledge | 2013 – 2015 |

## TEACHING EXPERIENCE

**University of Wisconsin – Milwaukee, Department of Psychology**

| *Course* | *Instructor* | *Year* |
|---|---|---|
| Research Methods in Psychology (PSYCH 325) | Peter Lenz | 2019, 2020, 2021 |
| Psychological Statistics (PSYCH 210) | Jennifer Kunz | 2018 |
| Physiological Psychology (PSYCH 254) | Ira Driscoll | 2018 |
| Physiological Psychology (PSYCH 254) | Maeng-Sik Shin | 2017 |

**Ball State University, Department of Psychological Science**

| *Course* | *Instructor* | *Year* |
|---|---|---|
| Introduction to Psychology (PSYS 101) | Darrell Butler | 2015, 2016 |
| Learning and Memory (PSYS 321) | Anjolii Diaz | 2015, 2016 |

## MENTORING EXPERIENCE

**University of Wisconsin – Milwaukee, Department of Psychology**

| *Student* | *Awards* | *Duration* |
|---|---|---|
| Jonathan Santiago | McNair Scholar, SURF Award | 2020 – 2021 |
| Karina Montoto | McNair Scholar, SURF Award | 2020 – 2021 |
| Sofia Mattson | Senior Excellence in Research Award, SURF Award | 2018 – 2021 |
| Janet Lagunez-Garcia | McNair Scholar, SURF Award | 2018 – 2020 |
| Joseph Kornkven | | 2018 – 2020 |
| Matthew Olsem | UR@UWM | 2019 |
| Emma Vanderbilt | UR@UWM | 2019 |
| Hannah Sallmann | SURF Award | 2018 – 2019 |

**Ball State University, Department of Psychological Science**

| *Student* | *Duration* |
|---|---|
| Anna Allen | 2016 – 2017 |
| Alex Mull | 2015 – 2016 |

## LABORATORY SKILLS

**General Research**

| | |
|---|---|
| Assessment inventory formation and analyses | Media lab software coding |
| E-Prime 2.0 & 3.0 task design and administration | SONA administration |
| Easymap software administration | Qualtrics software administration |

**Coding Languages**

| | |
|---|---|
| Basic MATLAB coding experience | Basic Python coding experience |
| Basic R coding experience | Basic SAS coding experience |

| Statistical Analytics | |
|---|---|
| Within and between *t*-tests | Basic correlational analyses |
| Within, between, and mixed ANOVAs | Simple and multiple linear regression analyses |
| Hierarchal linear modeling | Mediation and Moderation linear regression analyses |
| **Physiological Measures** | |
| Actigraphy acquisition and analyses | |
| **Behavioral Neuroscience** | |
| Open field paradigm procedures | Associative conditioning paradigm procedures |
| Instrumental conditioning paradigm procedures | Subcutaneous & intraperitoneal pharmacology injections |
| Micro infusions | Optogentics procedures |
| Stereotaxic surgery | Transcardial perfusion |
| **Cognitive Neuroscience** | |
| EEG (64-channel & 32-channel) procedures | EEG data acquisition (BioSemi & ANT systems) |
| EEG LAB & ERP LAB processing | Independent component analysis for EEG data processing |
| Quantitative EEG spectral extraction | ERP extraction and analyses |
| Time frequency analyses with Morlet wavelets | fNIRS data acquisition (NIRx systems) |
| AFNI fMRI data processing and analyses | |

**TEACHING SKILLS**

| Lecture | |
|---|---|
| In-person course lectures | Created slides and material content for lectures |
| **Course Design** | |
| Syllabi creation | Quiz & exam creation |
| **Study Assistance** | |
| Discussion board Q&A sessions | Independent course study sessions outside of office hours |
| **Grading** | |
| Multiple-choice and open response format quizzes, exams, and homework | Research paper submissions and edits based on APA |