HHS Public Access

Author manuscript

Psychiatry Res. Author manuscript; available in PMC 2021 November 01.

Published in final edited form as:

Psychiatry Res. 2020 November ; 293: 113468. doi:10.1016/j.psychres.2020.113468.

Impulsivity as a Multifactorial Construct and its Relationship to PTSD Severity and Threat Sensitivity

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Abstract

Changes to the DSM-5's conceptualization of posttraumatic stress disorder (PTSD) highlight the importance of impulsivity within the context of PTSD-related arousal dysregulation. While the relationship between PTSD and threat sensitivity is well defined, how they relate to impulsivity remains understudied. We examined the relationship between PTSD symptom severity, threat sensitivity, and impulsivity. 124 participants completed the PTSD Checklist (PCL-C) and the Barratt Impulsiveness Scale 11th ed (BIS-11). BIS-11 items were separated to define cognitive and behavioral impulsivity subdomains. A trauma-exposed subsample of 39 participants were also exposed to no, ambiguous, and high threat conditions in a threat-enhanced acoustic startle paradigm with psychophysiological response as the outcome variable. PTSD severity was significantly associated with greater overall impulsivity and behavioral impulsivity. Greater overall impulsivity and both cognitive and behavioral impulsivity subdomains were significantly associated with psychophysiological magnitudes across threat conditions in the traumatized subsample. Our results suggest PTSD severity may linked to behavioral impulsivity and both cognitive and behavioral impulsivity are associated with threat sensitivity and hyperarousal.

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Author's contributions

All authors have made substantial contributions to the research projects and/or the preparation of the manuscript. They have also reviewed and approved the final version of this manuscript. The following specific contributions have been made. *DAY*: conceptualization and design of the study; analysis and interpretation of the data; drafting the manuscript, manuscript revision, and final approval of the submitted version of the manuscript. *TCN*: conceptualization and design of the study; interpretation of the data; manuscript revision, and final approval of the submitted version of the manuscript. *SSI*: acquisition of the data; conceptualization and design of the study; interpretation of the data; manuscript revision; and final approval of the submitted version of the manuscript. *AO*: conceptualization and design of the study; interpretation of the data; manuscript revision; and final approval of the submitted version of the manuscript. *HZ*: interpretation of the data; manuscript revision; and final approval of the submitted version of the manuscript.

Declarations of Interest.

None.

Assessing impulsivity within the context of PTSD, particularly in terms of its cognitive and behavioral subdomains, may provide important, clinically relevant information.

Keywords

impulsivity; PTSD/Posttraumatic Stress Disorder; startle; trauma; biological markers; psychophysiological response

1. Introduction

Posttraumatic stress disorder (PTSD), which impacts a significant portion of the population, is a crippling mental health condition linked to adverse health and social outcomes that include poor physical health, unstable housing and homelessness, under- and unemployment, incarceration, increased odds of engaging in intimate partner violence, and suicide (Bartholdy et al., 2017; Elbogen et al., 2012; Gallaway et al., 2012; McClelland et al., 2016; Thomas et al., 2010). Marked alterations in arousal, a key feature of PTSD, includes previously classified symptoms of elevated threat sensitivity such as exaggerated startle, difficulty concentrating, and hypervigilance, but also symptoms associated with impulsivity such as irritability, aggression, and risky or self-destructive behavior. While the relationship between PTSD and threat sensitivity has been studied in detail (Shvil et al., 2013), research that has examined the PTSD-impulsivity relationship, particularly within the context of threat is sparse.

Theoretical models posit impulsivity as a major factor mediating PTSD-related adverse outcomes. However, divergent conceptualizations of impulsivity have complicated this body of research. Various theorists have suggested conceptual models of impulsivity that included anywhere from three to six subfactors (Stanford et al., 2009). More recent factor analyses based on neuroscience research have concluded that impulsivity can be accurately and concisely described by two factors: cognitive and behavioral impulsivity (Liu et al., 2017; Reise et al., 2013). Cognitive impulsivity (also called choice impulsivity) is associated with an oversensitivity to immediate gratification and impatience to receiving a reward along with a lack of planning and regard for future consequences when making choices (Hamilton et al., 2015b). Research suggests that sensation seeking, a tendency to seek excitement and a strong openness to trying new activities, which is a major factor associated with cognitive impulsivity is associated with both negative alterations in mood and alterations in arousal in individuals with PTSD (Roley et al., 2017). While adverse outcomes associated with cognitive impulsivity in individuals with PTSD symptoms include substance misuse, risky sexual behavior, and suicide (Hamilton et al., 2015b), the literature investigating cognitive impulsivity's relationship to PTSD is sparse (Amlung et al., 2019). Conversely, behavioral impulsivity (also called rapid-response impulsivity) is associated with difficulties in behavioral inhibition (i.e. preventing a behavior from starting or stopping a behavior once initiated; Hamilton et al., 2015a). PTSD is linked to various factors associated with behavioral disinhibition such as emotion dysregulation, aggression, and risky sexual behavior (Casada and Roache, 2005; Pawliczek et al., 2013; Sani et al., 2017). In particular recent findings suggest that negative urgency, or the tendency to act

rashly during episodes of intense negative affect and a core component of behavioral impulsivity is strongly associated with PTSD and its symptom sub-clusters (Roley et al., 2017). Research suggests that cognitive and behavioral impulsivity are associated with different neural networks and are at best loosely correlated with each other (Hamilton et al., 2015a; Liu et al., 2017). Furthermore, previous findings have implicated both cognitive and behavioral impulsivity in suicidal behavior across different psychiatric disorders (Corruble et al., 2003; Gvion and Apter, 2011; Malloy-Diniz et al., 2011), which stresses the clinical and epidemiologic importance of studying impulsivity, particularly as it relates to PTSD and trauma exposure. While this conceptualization of impulsivity shows promise in explicating the multidimensional nature of impulsivity, it has not been examined in PTSD and research that has focused on the PTSD – impulsivity relationship remains sparse.

A large body of literature has shown that individuals diagnosed with PTSD exhibit higher fear-potentiated physiological responses to sudden or threatening stimuli compared to those without a PTSD diagnosis, which suggests greater threat sensitivity (Grillon et al., 1998; Niles et al., 2018; Orr et al., 1995; Pole et al., 2003). However, it remains to be seen how PTSD – related threat sensitivity might be associated with impulsivity and its subdomains. Exploring how cognitive and behavioral impulsivity might be related PTSD severity and threat sensitivity in trauma-exposed indiviuals may elucidate how different facets of impulsivity are associated with other aspects of PTSD. Thus, to examine the relationship between PTSD, impulsivity, and threat in trauma-exposed individuals, we investigated whether impulsivity was associated with PTSD severity and psychophysiological reactivity to startling sounds across three different threat conditions in an acoustic startle paradigm. Threat conditions included no threat, ambiguous threat, and high threat. We hypothesized that: higher levels of self–reported impulsivity would be associated with 1) worse PTSD symptom severity, and 2) higher psychophysiological response magnitudes. We also used recommendations outlined by Reise et al. (2013) when using the Barratt Impulsivity Scale (11^{th} ed.) to specifically explore whether cognitive and behavioral impulsivity subscales were linked to PTSD severity as measured by PTSD Checklist scores (PCL-C) or threat sensitivity as measured by psychophysiological response magnitudes.

2. Methods

2.1. Participants

Participants aged between 18 and 65 were recruited from San Francisco Veterans Affairs Medical Center, non-VA outpatient and community clinics in the surrounding geographic area. Trauma-exposed individuals either met full or subthreshold criteria for the Diagnostic and Statistical Manual IV (DSM-IV TR) diagnosis of PTSD or were subthreshold for the diagnosis. Exclusion criteria included organic mental disorder, schizophrenia, bipolar disorder, current alcohol dependence, current drug abuse or dependence (within the prior 3 months), seizure disorders, neurological disorders, previous moderate or severe head injuries, current infectious illness, and systemic illness affecting CNS function. Exclusionary medications included alpha and beta-adrenergic agents, antipsychotics, benzodiazepines, mood stabilizers, anticonvulsants, antihypertensives, sympathomimetics, and steroids. The University of California, San Francisco and Veterans Administration Committees on Human

Research and the Department of Defense Human Subjects Research Review Board approved all research. This research was carried out in accordance with The Code of Ethics of the World Medical Association and all participants provided consent to be included in this study.

2.2. Demographic and Clinical Measures

Demographic variables including participants' age, sex, race (white versus minority), and education level were recorded for use in subsequent analyses based upon prior literature linking them to traumatic stress response (Engelhard et al., 2006; Neylan et al., 2005). 124 participants engaged in research activities related to an overall study that examined the impact of hydrocortisone on fear conditioning. Of those 124 participants, 39 participants who endorsed trauma exposure also provided startle data. Current PTSD symptoms (e.g. in the past month) were evaluated using the PTSD Checklist (PCL-C; Blanchard et al., 1996). Based upon our previous research (Young et al., 2019), early trauma (i.e. before 16 years old) was evaluated using the childhood neglect (item 8), childhood physical abuse, (item 18), childhood sexual abuse (item 20) items from the Life Stressor Checklist, Revised (LCS-R; (Wolfe et al., 1996)). The Barratt Impulsiveness Scale 11th ed. (BIS-11), a self-report impulsivity scale that has been used in many different research settings, was used to assess impulsivity (Cronbach's $\alpha = 0.85$; Patton et al., 1995). Based upon confirmatory factor analyses by Reise et al., (Reise et al., 2013), the items of the BIS-11 appear to be more accurately expressed between two cognitive and behavioral impulsivity subconstructs with separate parcels under these constructs. Parcels and items that are associated with cognitive impulsivity were the following: "not planful" (items 1 and 7); "lives in the moment" (items 13, 27, and 30); "no concentration/self – control" (items 8 and 9); "buying and spending sprees" (items 10, 22, and 25); "not a steady thinker" (items 12 and 20); and "likes complicated things" (items 15, 18, 23 and 29). Parcels and items that were associated with behavioral impulsivity were the following: "acts impulsively" (items 17 and 19); can't sit still (items 11 and 28); "changes, moves around" (items 16, 21, and 24); "extraneous/racing thoughts" (items 6 and 26); and "no cognitive mediation" (items 2, 5, and 14). Therefore, we generated separate cognitive (Cronbach's $\alpha = 0.81$) and behavioral (Cronbach's $\alpha = 0.86$) based upon these factor analyses (factor loadings for 6the current data are reported in this manuscript's Supplementary Materials).

2.3. Psychophysiological Response Procedure

Skin conductance response (SCR), and heart rate were collected by trained technicians blind to participants' clinical status from the 39 trauma-exposed participants. The participant's SCR and heart rate were assessed during a two-minute baseline. Participants were fitted with headphones and told that they would hear startling sounds. They were asked to focus their eyes on a monitor in front of them. A Coulbourn Instruments Lablinc V Modular System binaurally presented 106-dB(A), 40 millisecond white noise bursts with nominal 0-millisecond rise and fall times separated by inter-trial intervals of between 30 and 50 seconds in each threat condition. In the "no threat" condition, participants were instructed that they would not be shocked until later in the study. They were then exposed to ten startling sounds. Only their last five responses were included in analyses. In the "ambiguous threat" condition, participants were fitted with a Coulbourn Instruments Transcutaneous Aversive Finger Stimulator but were told that they would not be shocked. Five additional

startling sounds were presented. In the “high threat” condition, participants wore the finger stimulator and were told that shocks were imminent. Specific to the high threat condition, five additional startling sounds were presented followed by a 2.5 mA shock. Each condition lasted approximately 4 minutes and was separated by about 1 minute. The ambiguous and high threat conditions were counterbalanced to minimize carry-over effects between these conditions. All physiological signals were sampled at 2 Hz during the resting baseline for 5 minutes prior to stimulus presentation and increased to 1000 Hz 4 seconds prior to acoustic startle stimulus presentations. After testing, physiologic signals were digitized, and stored for off-line analysis. SCR was measured in microsiemens with InVivo Metrics Ag/AgCl electrodes placed on the hypothenar surface of the medial phalanges of the middle and index fingers of the non-dominant hand as described in (Young et al., 2019). Heart rate was measured in beats per minute and recorded via electrodes attached in a Type-I EKG configuration. Human Startle Software (Coulbourn Instruments, Allentown, PA) automatically calculated mean levels of SCR at baseline, during the one second prior to each stimulus onset and within 1 to 4 seconds for SCR. An accelerative heart rate response score was calculated for each trial by subtracting the mean heart rate level preceding tone onset, from the highest heart rate level measured within 1 – 4 seconds after the onset of the tone. No minimum response threshold was designated for any physiological measure. Each measurement of psychophysiological response was recorded prior to and following exposure to the startle stimulus on each of five trials under each threat condition. Participants needed at least four (of five) valid responses for all three psychophysiological measures within each threat condition to be included in the study. Responses were inspected for potential artifact and rejected accordingly (6 cases).

2.4. Data Analyses

Due to a non-normal distribution, the BIS-11 was log transformed and entered in as continuous variable in all models. Prior to linear models, a PCL-C/BIS-11 correlation matrix was generated, which included PCL-C scores and overall BIS-11 scores and cognitive and behavioral impulsivity subscales (as outlined by Reise et al., 2013). To assess the relationship between PTSD symptom severity and selfendorsed impulsivity, a two-step hierarchical multiple linear regression model was used with the initial model including age in years, education in years, and dichotomous indicators of race (White versus non-White), sex (female versus male), and early trauma history on log-transformed BIS-11 scores. PCL scores were introduced into the second step of the model. Cognitive and behavioral BIS subscales were also analyzed using the same two-step multiple linear regression analysis as described above. To adjust for multiple comparisons, a Bonferroni correction was used where $p \leq 0.05/2$; thus, the corrected alpha level for BIS subscales was $p \leq 0.025$. Psychophysiological response outcome was assessed by using within trial square root post- minus pre-SCR and heart rate responses. Repeated measures linear mixed models were used to assess the relationship between self-reported impulsivity and threat condition on startle reactivity (McCulloch and Neuhaus, 2001). This model included a log-adjusted BIS score x threat condition interaction term. Depending on significance of the initial model, cognitive and behavioral BIS subscales were also analyzed using two additional repeated measures linear mixed models with a Bonferroni correction of $p \leq 0.025$. Age, race (white vs. non-white), sex (female vs. male), education (in years), and child trauma

history (presence versus absence) were included as covariates in all repeated measures linear mixed models. Stata Statistical Software: Release 15.1 was used to conduct all statistical analyses (StataCorp LP, 2013 College Station, TX). Cohen's f^2 was used to assess proportion of model variance explained. f^2 was generated using user written code based on previously published methods described elsewhere (Selya et al., 2012). We calculated the derivative of psychophysiological response magnitude with respect to threat condition (i.e. the interaction between BIS score with respect to its between threat condition changes in slope of psychophysiological response magnitude) to examine within model slope change, where SCR or heart rate magnitude = m and threat condition = t; thus, in standard notation, $m'(t) \approx 1/h\,[m(t+h) - m(t)]$.

3. Results

3.1. Demographics and Measurement Correlations

Pairwise demographics are described in Table 1. Approximately 43% of participants were female and White (52.42%) was the most identified race in the sample. The mean PCL and BIS scores for the sample were 45.92 and 60.93, which were somewhat lower in comparison to a PTSD clinic population, and might be related to the stringent inclusion and exclusion criteria of the broader study that also included non-treatment seeking participants from the community (Blanchard et al., 1996; Patton et al., 1995). Regarding measurement correlations, the overall BIS score was significantly correlated with PCL scores ($r = 0.27$, $p = 0.002$). The behavioral impulsivity subscale was also significantly correlated with the PCL scores ($r = 0.48$, $p < 0.001$; see Table 2.). Cognitive and behavioral impulsivity subscales were inversely correlated with each other ($r = -0.34$. $p < 0.001$). The cognitive impulsivity subscale was not correlated with PCL scores.

3.2. Relationship between PTSD Severity and Impulsivity

As Table 3 indicates, model significance was not achieved in Step 1 and sex was significantly associated with BIS score with women endorsing less impulsivity compared to men ($b = -0.04$, $p = 0.038$). Results from Step 2 indicate model significance ($F_{6,117} = 3.46$; $R^2 = 0.150$; $p = 0.004$). In Step 2, greater PCL scores emerged as a significant predictor of BIS score ($b = 0.16$, $p = 0.004$). Post hoc analyses confirmed the significant relationship between greater PCL scores and greater BIS scores and that the PCL parameter was significantly additive to the model ($F_{1,117} = 8.30$; $p = 0.004$; change in $R^2 = 0.064$). Sex was not significantly associated with impulsivity in Step 2.

3.3. Subscales of Impulsivity as they Relate to PTSD Severity

As shown in Table 4, Step 1 on the behavioral impulsivity subscale model was significant ($F_{5,118} = 3.31$; $R^2 = 0.08$; $p = 0.008$). Women endorsed less behavioral impulsivity compared to men ($b = -0.04$, $p = 0.001$). Step 2 of the model was also significant ($F_{6,117} = 7.79$; $R^2 = 0.285$; $p < 0.001$) and elevated PCL scores were significantly associated with the endorsement of greater behavioral impulsivity ($b = 0.01$, $p < 0.001$). Post hoc analyses confirmed that the addition of PCL score parameter was significantly additive to the model ($F_{1,117} = 26.61$; $p < 0.001$; change in $R^2 = 0.163$). Sex continued to be significant predictive of behavioral impulsivity in Step 2. To assess this in greater detail, we briefly

ran post hoc analyses to examine whether behavioral impulsivity was associated with DSM – IV TR PTSD symptom clusters. We found that behavioral impulsivity was associated with all symptom clusters (reexperiencing: $b = 0.22$, $p = 0.001$; avoidance: $b = 0.12$, $p = 0.004$; hyperarousal: $b = 0.17$, $p < 0.001$). Models assessing cognitive impulsivity were not significant and no significant parameters emerged in either step of the analyses (see Table 4.).

3.4. Relationship between Impulsivity and Startle Reactivity in the Trauma-exposed Subsample

Model effects for heart rate were significant ($Wald\ \chi^2 = 51.83$; $p < 0.001$) but not for SCR. Post-hoc analyses revealed a significant BIS score x threat condition interaction where participants who endorsed greater levels of impulsivity exhibited greater mean heart rate magnitudes over the three threat conditions ($\chi^2 = 20.48$; $f^2 = 0.32$; $p < 0.001$; see Figure 2a.). Derivative analyses indicated that participants who endorsed high levels of impulsivity had significantly greater changes in mean heart rate slope over threat conditions ($m'(t) = 4.56$; $SE = 1.00$; $z = 4.37$; $p < 0.001$ in 'No Threat' compared to 'Ambiguous Threat' conditions and $m'(t) = 2.37$; $SE = 1.00$; $z = 2.37$; $p = 0.018$ in 'No Threat' compared to 'High Threat' conditions). BIS scores did not significantly interact with threat condition on SCR.

3.5. Relationship between Impulsivity Subscales and Startle Reactivity in Trauma-exposed Subsample

Model effects examining cognitive impulsivity on psychophysiological response were significant for SCR ($Wald\ \chi^2 = 20.12$; $p = 0.005$) but not for heart rate. Post-hoc analyses revealed a significant BIS cognitive impulsivity subscale x threat condition interaction where participants who endorsed greater levels of cognitive impulsivity exhibited greater mean SCR magnitudes over the three threat conditions ($\chi^2 = 12.89$; $f^2 = 0.28$; $p = 0.002$; see Figure 2b.). Derivative analyses indicated that participants who endorsed greater levels of cognitive impulsivity also had significantly greater changes in mean heart rate slope over threat conditions ($m'(t) = 0.88$; $SE = 0.27$; $z = 3.29$; $p = 0.001$ in 'No Threat' compared to 'Ambiguous Threat' conditions and $m'(t) = 0.70$; $SE = 0.25$; $z = 2.82$; $p = 0.005$ in 'No Threat' compared to 'High Threat' conditions). Cognitive impulsivity did not significantly interact with threat condition on heart rate. Model effects examining behavioral impulsivity were significant for heart rate ($Wald\ \chi^2 = 36.75$; $p < 0.001$) but not for SCR. Post-hoc analyses revealed a significant BIS behavioral impulsivity subscale x threat condition interaction where participants who endorsed greater levels of impulsivity exhibited greater mean heart rate magnitudes across the three threat conditions ($\chi^2 = 21.20$; $f^2 = 0.33$; $p < 0.001$; see Figure 2c.). Derivative analyses indicated that participants who endorsed greater levels of behavioral impulsivity also had significantly greater changes in mean heart rate slope over the three threat conditions ($m'(t) = 1.70$; $SE = 0.38$; $z = 4.51$; $p < 0.001$ in 'No Threat' compared to 'Ambiguous Threat' conditions and $m'(t) = 1.12$; $SE = 0.37$; $z = 3.03$; $p = 0.002$ in 'No Threat' compared to 'High Threat' conditions). BIS behavioral impulsivity subscales did not significantly interact with threat condition on SCR.

4. Discussion

Our primary finding is that PTSD severity was significantly associated with overall impulsivity. More specifically, PTSD severity appears to be strongly associated with elevated behavioral impulsivity but not cognitive impulsivity. Given that the effect size was quite large for the behavioral impulsivity model ($R^2 = 0.285$), these results align with recent findings suggesting that behavioral impulsivity may mediate the adverse outcomes associated with trauma exposure and PTSD (Armour et al., 2016; Roley et al., 2017). This finding appears to extend to men in particular as women endorsed lower levels of behavioral impulsivity, even after we controlled for PTSD symptoms. A previous metanalysis suggests that women generally endorse/exhibit lower levels of behavioral (but not cognitive) impulsivity (Cross et al., 2011). This may suggest that behavioral impulsivity drives PTSD-related difficulties in affect regulation in men, which manifests in negatively externalized, aggressive behavior during episodes of negative urgency (e.g. fighting, intimate partner violence; Roley et al., 2017). Recent findings that impulsivity mediates PTSD symptom severity and aggressive behavior prior to PTSD treatment in men substantiates this interpretation (Heinz et al., 2015). Of note, we also found that cognitive and behavioral impulsivity were inversely correlated with each other and this may have played a role in the lack of relationship between cognitive impulsivity and PTSD severity. While the generalizability of this finding remains to be seen and more research is needed to further clarify this observation, this may suggest that higher levels of either cognitive or behavioral impulsivity may be mutually exclusive of each other in this particular clinical sample.

We also found that while overall impulsivity as a general construct is associated with greater threat sensitivity in trauma-exposed individuals, which confirmed Hypothesis 2, *both* cognitive and behavioral impulsivity appear to be associated with threat sensitivity but in different ways based upon their respective psychophysiological magnitude slopes. Specifically, behavioral impulsivity was associated with greater psychophysiological reactivity to the startle probe in the no threat condition, which may suggest individuals with high levels of behavioral impulsivity may be particularly threat sensitive to novel stimuli even in the presence of safety cues, which could lead to adverse social outcomes. While there is limited research that has examined the relationship between behavioral impulsivity and threat sensitivity in individuals with PTSD, adverse outcomes linked to both PTSD and behavioral impulsivity (e.g. aggression, intimate partner violence, suicide) may be associated with elevated levels of psychophysiological reactivity (Derefinko et al., 2011; Heesink et al., 2017). One of the key features of PTSD is the inability to inhibit the fear response within the context of safety cues (Jovanovic et al., 2012). Further, our group and others have previously shown that individuals with PTSD who are more sensitive to threat also exhibit structural and functional abnormalities in specific prefrontal areas such as the ventromedial prefrontal cortex, the anterior cingulate cortex, and the insula (Etkin et al., 2011; Marusak et al., 2015; Young et al., 2018). Taken together, our findings suggest that the debilitating symptoms associated with PTSD along with behaviorally impulsive acts such as risk taking and aggression subsequent to trauma exposure may be rooted in a similar neurobiological etiology that involves threat system dysregulation.

Although we did not find any evidence that cognitive impulsivity was associated with PTSD severity, we did find a robust relationship between cognitive impulsivity and psychophysiological response where those who endorsed a greater amount of cognitive impulsivity had an elevated and flat response pattern across each of the three threat conditions. That cognitive impulsivity was not associated with PTSD symptom severity but was associated with greater psychophysiological response magnitude to startling stimuli is notable. While very few studies have examined these relationships, some recent findings have shown that sensation seeking is associated with PTSD arousal and negative alterations in mood sub-clusters (Contractor et al., 2016; Roley et al., 2017). On the other hand, only two studies that we are aware of have evaluated cognitive impulsivity within the context of PTSD and trauma exposure. One study found that depressed participants with a comorbid PTSD diagnosis made less cognitively impulsive choices compared to depressed participants without a comorbid PTSD diagnosis (Engelmann et al., 2013). On the other hand, a recent study found that trauma exposure was associated with cognitively impulsive choices in a low income, male, African American sample (van den Berk-Clark et al., 2018). Unfortunately, due to the dearth of research that has examined cognitive impulsivity within the context of PTSD (Amlung et al., 2019), it is unclear whether the lack of relationship between PTSD and cognitive impulsivity observed in the current study is generalizable or not and more work in this area will be needed to clarify these relationships.

The relationship between cognitive impulsivity and psychophysiological responses differs from the response pattern observed with general impulsivity and behavioral impulsivity in that the response pattern is elevated but flat. We have previously suggested that a high and flat psychophysiological response pattern might be indicative of more general elevated basal arousal level (Young et al., 2018). Further, a body of literature has connected cognitive impulsivity to maladaptive sensation-seeking behaviors. For example, a consistent finding in the cognitive impulsivity literature is that indices of cognitive impulsivity (e.g. delay discounting) are strongly associated with unhealthy substance abuse behavior where individuals who have higher levels of cognitive impulsivity abuse substances more often and more severely (Bickel and Yi, 2008; Petry, 2001). Moreover, a body of evidence exists that suggests higher levels of cognitive impulsivity are associated with risky sexual behavior where individuals who are higher in cognitive impulsivity would choose to engage in immediate unprotected sex rather than use a condom if there is a delay to obtaining the condom (Hahn et al., 2019; Johnson and Bruner, 2012). Thus, our findings may indicate that sensation seeking in individuals who have higher levels of cognitive impulsivity may be driven by higher levels of physiological arousal.

The results from the current study have a several important clinical implications. First, and foremost, our findings suggest that assessing impulsivity and its subconstructs is, at the very least, clinically judicious when treating patients with PTSD as the information obtained holds the potential to provide valuable clinical information, particularly in terms of treatment planning and harm reduction (Heinz et al., 2015). For example, in addition to the unhealthy sensation seeking behaviors such as risky sexual behaviors and substance misuse described above, higher levels of cognitive impulsivity and sensation seeking have also be linked to generalized aggressive acts (e.g. physical fights, severely injuring another person), which underscores the importance of cognitive impulsivity to public health (Derefinko et al.,

2011; Hamilton et al., 2015b). Therefore, implementing measures of cognitive impulsivity in clinical settings may provide a means to identify patients that are of high risk of engaging in future harmful acts. Contingency management has been suggested as a potentially beneficial treatment paradigm for mitigating harmful cognitive impulsive actions (Hamilton et al., 2015b). Similarly, cognitive training that focuses on strengthening prefrontal regions have also shown promise in reducing cognitive impulsivity (Bickel et al., 2011; Verbeken et al., 2013). Regarding the behavioral impulsivity-PTSD relationship, research suggests that the inability to inhibit behavior may be particularly acute during episodes of intense affect (Derefinko et al., 2011), which most likely is exacerbated in individuals with PTSD given their elevated threat sensitivity and tendency to have negative alterations in cognition and mood (American Psychiatric Association, 2013). Therefore, interventions that target the drive to engage in immediate action that also reduce the psychological tension associated with intense negative emotion states may prove to be particularly therapeutic for patients with PTSD–related behavioral impulsivity (Derefinko et al., 2011; Roley et al., 2017). These forms of therapy may be particularly relevant for male patients with a history of engaging in intimate partner violence.

As described above, both cognitive and behavioral impulsivity have been implicated in suicidality. However, differences suicide type (i.e. planned versus non-planned suicide) is important when considering the impulsivity-suicide relationship. Specifically, planned suicide, which by definition is preceded by far more preparation and forethought, is more so associated with completed suicide, and appears to have greater lethality due to its inherent *lack* of impulsivity (Gvion and Apter, 2011). On the other hand, findings have implicated impulsivity in its relationship to non-planned suicide, which involves marginal preparation, is more impulsive but also less lethal (Gvion and Apter, 2011). For example, an earlier study found that people who engaged in near lethal suicidal attempts that had less than five minutes of preparation tended to use less terminally lethal means, had higher odds of the attempt being thwarted through intervention, and had lower expectations of death (Simon et al., 2001). Thus, while planned suicide appears to be more complex and most likely driven by other factors outside of impulsivity, tools that assess overall impulsivity along with cognitive and behavioral impulsivity subdomains may hold significant clinical utility in reducing morbidity and mortality rates associated with non-planned suicide attempt.

This study has several limitations of note. First, despite the large effect sizes found in the results from the threat-enhanced acoustic startle paradigm, the interpretability and generalizability of our results is tempered by the small size of the trauma-exposed subsample. Similarly, important intergroup differences may have emerged if a control group was used. Therefore, future studies examining impulsivity within the context of PTSD and threat sensitivity that employ a both a larger sample size and non-trauma exposed control group would benefit from greater power and an increased interpretability due to group comparison. Secondly, this was a cross-sectional study and therefore we lack the ability to make causal inferences. Additionally, while the BIS – 11 is a well validated impulsivity measure, it is also a self-report instrument and by nature susceptible to response bias. There are several well–validated paradigms that have been shown to empirically capture cognitive (e.g. delay discounting) and behavioral (e.g. go/no-go, stop-signal) impulsivity (Hamilton et al., 2015a; Hamilton et al., 2015b). Future studies exploring the relationship

between threat reactivity and impulsivity in PTSD should aim to include these tasks in addition to self-report measures when examining impulsivity in PTSD samples. Another limitation in our study is that it was somewhat racially homogenous, which did not allow us for a broad between – group comparison of race as it relates to PTSD, impulsivity, and psychophysiological response. Additionally, participants with current and recent substance abuse/dependence were also excluded from this study. Given previous research has shown that PTSD and impulsivity are both associated with substance misuse (Weiss et al., 2012; Bickel el al., 2014), future studies should examine these relationships within the context of substance use comorbidity. Finally, while there is substantial evidence that psychophysiological reactivity appears to related to prefrontal morphometric and functional abnormalities (Admon et al., 2013; Etkin et al., 2011), the current study lacks imaging data and therefore cannot infer any conclusions regarding the neurobiological or neurofunctional origins of the observed threat sensitivity–impulsivity relationships.

In summary, we found that impulsivity was strongly correlated with both PTSD severity and threat sensitivity. Furthermore, while cognitive impulsivity was strongly associated with psychophysiological reactivity, behavioral impulsivity was associated with both PTSD severity and psychophysiological reactivity in men but not women. Based upon the interpretation of our findings, behavioral impulsivity may be a significant predictor of both PTSD severity and adverse social outcomes such as aggressive behavior along with verbal, and physical altercations due to the misinterpretation of novel but innocuous stimuli. Conversely, cognitive impulsivity, while not associated with PTSD in this study per se, may also be a clinically relevant feature of trauma exposure, particularly within the context of treatment planning given its previously shown relationship to unhealthy actions such as substance abuse and risky sexual behavior. The current study adds a novel contribution to the burgeoning PTSD–impulsivity literature and our results highlight the importance of considering maladaptive impulsivity and its subtypes within the context of clinical assessment and treatment planning for patients with PTSD. Specifically, targeting both cognitive and behavioral impulsivity may afford clinicians the ability to reduce sensation seeking and behavioral disinhibition related harm in PTSD patients and other traumatized individuals. In addition to replicating the current findings, examining these relationships both in an appropriately powered sample with PTSD and appropriate comparison groups would be the next logical step in terms elucidating the relationship between impulsivity, PTSD, threat sensitivity, and arousal. Finally, a body of research demonstrates the utility of neuroimaging for interrogating cortical and subcortical brain regions while participants engage in tasks designed to elicit cognitive (delay discounting) and behaviorally (go/no-go) impulsive responses (Peters and Büchel, 2011; Simmonds et al., 2008) but exceedingly few studies have examined these relationships within the context of PTSD. Employing imaging techniques to examine cognitive and behavioral impulsivity within the context of PTSD would be an important step toward understanding the neurobiological etiology behind impulsivity and PTSD. Such studies would also provide valuable information towards the development of novel, targeted, and patient – centered treatments for PTSD–related impulsivity.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

The authors wish to thank the Veteran and civilian study participants along with the members of the Stress and Health Research Program for all of their help in making this manuscript possible. All authors have approved the final version of this manuscript.

Role of Funding Source

This work was supported by Veterans Affairs Career Development Award CDA-2-037-07F, entitled “Effects of Hydrocortisone and D-Cycloserine on Fear Extinction in Veterans with PTSD,” awarded to Dr. Inslicht and a National Institutes of Mental Health Diversity Supplement linked to the Research Project Grant R01MH115020-01 entitled “Individual Variation in Effects of Traumatic Stress on Gray Matter Myelin” awarded to Drs. Young and Neylan. This material is the result of work supported with resources and the use of facilities at the San Francisco VA Medical Center. This work also supported by the VA Advanced Fellowship Program in Women's Health at the San Francisco Veterans Affairs Medical Center. The authors declare no potential conflicts of interest.

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Highlights

- PTSD severity was associated with overall impulsivity and behavioral impulsivity.
- PTSD severity was not associated with cognitive impulsivity.
- Cognitive and behavioral impulsivity were both associated with threat sensitivity.
- Clinical implications are discussed.

Figure 1.

Note: BIS-11 scores were log transformed

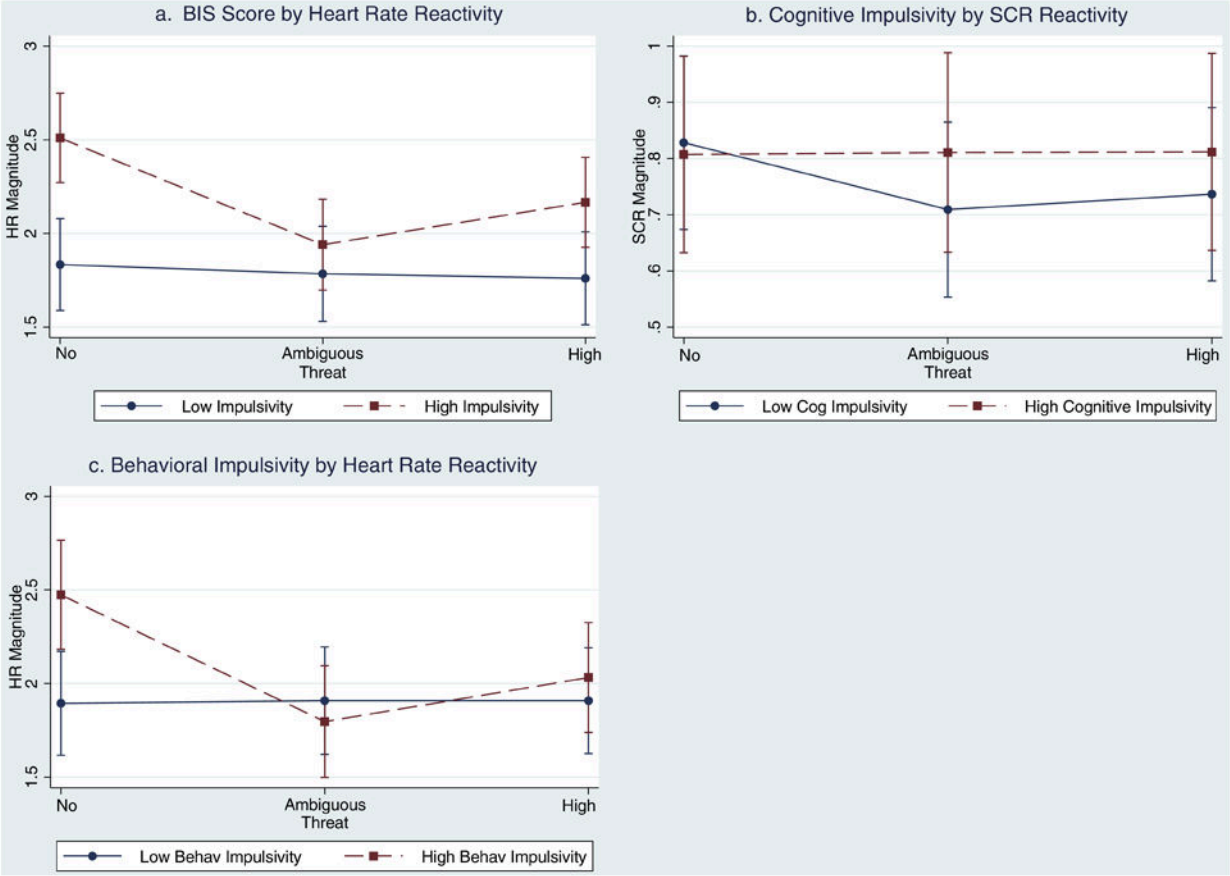

Figure 2.

Table 1.

Descriptive statistics N = 124

Characteristics			
N (%)			
Sex	Male	71 (57.26)	
	Female	53 (42.74)	
Race	Asian/PI	16 (12.90)	
	Black	19 (15.32)	
	Latino	9 (7.26)	
	White	65 (52.42)	
	Other	15 (12.10)	
Child trauma		14 (11.29)	
Mean (SD)			**Range***
Age		38.55 (12.45)	20 – 64
Education*		4.50 (1.65)	1 – 8
BIS score		60.93 (10.20)	49 – 76
PCL score		45.92 (17.78)	18 – 73

Note: SD = standard deviation; PI = Pacific Islander; +Education is given in years.

*Range was given by the 5th and 95th percentiles.

Table 2.

Correlations between PTSD and Impulsivity Variables

Measure	1.	2.	3.	4.
1. PCL – C Score	-			
2. BIS score	0.27**	-		
3. Cognitive Impulsivity Subscale	−0.18	0.51***	-	
4. Behavioral Impulsivity Subscale	0.48***	0.61***	−0.34***	-

Note. BIS scores were log-transformed

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$.

Psychiatry Res. Author manuscript; available in PMC 2021 November 01.

Table 3.

Hierarchical Regression Models Assessing the Relationship Between PTSD and Impulsivity (N = 124)

Variable	Step 1			Step 2		
	b	**95% CI**	***Omnibus test***	***b***	**95% CI**	***Omnibus test***
			$F_{5,118} = 2.28$; $R^2 = 0.03$			$F_{6,119} = 3.46^{**}$; $R^2 = 0.15$
Age*	−0.01	(−0.01 – −0.01)		−0.01	(−0.08 – −0.02)	
Female	−0.03*	(−0.01 – 0.02)		−0.03	(−0.06 – 0.01)	
Race	0.01	(−0.13 – 0.21)		−0.01	(−0.01 – 0.01)	
Years of Education*	0.04	(−0.07 – 0.06)		0.01	(−0.01 – 0.03)	
Child trauma	0.03	(−0.02 – 0.07)		0.02	(−0.02 – 0.07)	
PCL – C Score				0.16**	(0.14 – 0.18)	

Note. BIS scores were log-transformed; CI = confidence interval

* age and education were continuous variables within the model

adjusted R^2 is given for effect size

$p < 0.05$

** $p < 0.01$.

Table 4.

Hierarchical Regression Models Assessing the PTSD and Impulsivity Subscales (N = 124)

Cognitive Impulsivity						
	Step 1			**Step 2**		
Variable	***b***	**95% CI**	***Omnibus test***	***b***	**95% CI**	***Omnibus test***
			$F_{5,118} = 1.57$; $R^2 = 0.02$			$F_{6,119} = 1.70$; $R^2 = 0.03$
Age[1]	−0.01	(−0.01 – 0.02)		−0.01	(−0.05 – 0.02)	
Female	−0.04	(−0.01 – 0.09)		0.03	(−0.35 – 0.08)	
Race	−0.02	(−0.04 – 0.01)		−0.01	(−0.03 – 0.02)	
Years of education[1]	0.02	(−0.01 – 0.03)		0.02	(−0.01 – 0.03)	
Child trauma	−0.06	(−0.16 – 0.10)		−0.01	(−0.02 – 0.01)	
PCL Score				0.01	(−0.01 – 0.02)	
Behavioral Impulsivity						
			$F_{5,118} = 3.31^{**}$; $R^2 = 0.09$			$F_{6,119} = 7.76^{**}$; $R^2 = 0.29$
Age[1]	−0.01	(−0.01 – 0.02)		−0.01	(−0.08 – 0.02)	
Female	−0.09**	(−0.22 – 0.05)		−0.09*	(−0.17 – −0.01)	
Race	0.03	(−0.01 – 0.03)		0.01	(−0.02 – 0.04)	
Years of Education[1]	0.01	(−0.04 – 0.02)		0.01	(−0.02 – 0.02)	
Child trauma	0.04	(−0.05 – 0.12)		−0.01	(−0.07 – 0.10)	
PCL Score				0.01***	(0.01 – 0.02)	

Note. BIS scores were log-transformed; CI = confidence interval; * age and education were continuous variables within the model; adjusted R^2 it is given for effect size

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$.