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Alexa Kondas April 7, 2025

White Matter Correlates of Dissociation in a Diverse Sample of Trauma-Exposed Women

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Dissociation is a common trauma response to trauma linked to functional brain disruptions in brain networks subserving emotion regulation and multisensory integration; however, structural neural correlates of dissociation are less known, particularly abnormalities in stress-sensitive white matter (WM) tracts. The present study examined associations between dissociation and WM microstructure, assessed via fractional anisotropy (FA), in a large, diverse sample of women recruited as part of a long-standing trauma study, the Grady Trauma Project (GTP). As part of GTP, 135 trauma-exposed women (18-62 years old, M = 34.25, SD = 12.96, 84% self-identifying as Black) were recruited, received diffusion-weighted imaging, and completed the Multiscale Dissociation Inventory (MDI); FA values were extracted from ten major WM tracts of interest. Partial correlations were conducted to examine associations between dissociation facets (MDI total and subscales) and FA while covarying age and temporal signal-to-noise ratio; false discovery rate corrected $p < .05$ indicated statistical significance. FA in seven tracts showed significant negative associations with overall dissociation (MDI total score; rs < -.19, $p_{FDR} < .05$); the corona radiata, corpus callosum, superior longitudinal fasciculus, thalamic radiation, anterior cingulum, fornix, and uncinate fasciculus. Among facets of dissociation, FA was most consistently associated with dissociative memory disturbance, showing a significant and negative association with all but one of tracts of interest, (rs < -.23, $p_{FDR} < .05$). Our findings indicated that dissociation severity was linked to proportionally lesser WM microstructural integrity in tracts involved with sensory integration, emotion regulation, memory, and self-referential processing. Disruptions in these pathways may underlie dissociative phenomena, representing important psychotherapeutic and neuromodulatory targets.

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1. Disclosure

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2. Abstract

Dissociation is a common trauma response to trauma linked to functional brain disruptions in brain networks subserving emotion regulation and multisensory integration; however, structural neural correlates of dissociation are less known, particularly abnormalities in stress-sensitive white matter (WM) tracts. The present study examined associations between dissociation and WM microstructure, assessed via fractional anisotropy (FA), in a large, diverse sample of women recruited as part of a long-standing trauma study, the Grady Trauma Project (GTP). As part of GTP, 135 trauma-exposed women (18-62 years old, M = 34.25, SD = 12.96, 84% self-identifying as Black) were recruited, received diffusion-weighted imaging, and completed the Multiscale Dissociation Inventory (MDI); FA values were extracted from ten major WM tracts of interest. Partial correlations were conducted to examine associations between dissociation facets (MDI total and subscales) and FA while covarying age and temporal signal-to-noise ratio; false discovery rate corrected $p < .05$ indicated statistical significance. FA in seven tracts showed significant negative associations with overall dissociation (MDI total score; rs < -.19, $p_{FDR} < .05$); the corona radiata, corpus callosum, superior longitudinal fasciculus, thalamic radiation, anterior cingulum, fornix, and uncinate fasciculus. Among facets of dissociation, FA was most consistently associated with dissociative memory disturbance, showing a significant and negative association with all but one of tracts of interest, (rs < -.23, $p_{FDR} < .05$). Our findings indicated that dissociation severity was linked to proportionally lesser WM microstructural integrity in tracts involved with sensory integration, emotion regulation, memory, and self-referential processing. Disruptions in these pathways may underlie dissociative phenomena, representing important psychotherapeutic and neuromodulatory targets.

3. Introduction

Despite years of discussion on dissociation, it still remains one of the most controversial and least understood aspects of mental health. The Diagnostic and Statistical Manual of Mental Disorders (DSM-5) defines dissociation as "a disruption of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behavior" (APA, 2013). Dissociation can take on many forms of mental dysfunction, such as dissociative amnesia, depersonalization (detachment from the body), derealization (detachment from one's surroundings), and identity confusion (Sar, 2014). Dissociation symptoms are often categorized into two different types: nonpathological and pathological dissociation. Nonpathological dissociation consists of everyday dissociative experiences that are present in the absence of psychopathology (Ellickson-Larew et al., 2020; Sar, 2022), with transient dissociation being relatively common (Hunter et al., 2004). On the other hand, pathological dissociation encompasses dissociative symptoms that occur frequently with negative impacts, comprising about 3.3% of the general population (Waller & Ross, 1997). Risk factors for pathological dissociation, or a dissociative disorder, include trauma or prolonged exposure to highly stressful events, drug use, sleep deprivation, harm avoidance, and immature defense mechanisms (APA, 2013; Selvi et al., 2015).

Because dissociation is normally accompanied by exposure to traumatic or stressful events, it has been primarily linked to posttraumatic stress disorder (PTSD), which is significant distress and impairment following a traumatic event, including avoidance of associated stimuli and negative changes in cognition, mood, and reactivity; these symptoms can sometimes include dissociative symptoms, warranting the dissociative subtype diagnosis that about 12-30% of individuals with PTSD meet the criteria for (APA, 2013; Lanius et al., 2012). Additionally,

dissociation that occurs during trauma (peritraumatic dissociation) significantly increases the risk of developing severe/chronic PTSD (Atchley & Bedford, 2021; Lebois, Harnett, et al., 2022).

Other negative effects of dissociation include decreased mental and physical health as well as an increased likelihood of psychiatric hospitalization and suicide attempts (Simeon & Putnam, 2022). Persistent, clinically-significant dissociation has major public health consequences, reducing quality of life and leading to significant disability in many individuals (Brand & Lanius, 2014; Leonard et al., 2005; Polizzi et al., 2022; Webermann et al., 2016). People with marginalized identities, including minoritized racial/ethnic groups, tend to report higher rates of dissociation (Douglas, 2009). Racial discrimination, a chronic stressor for racially/ethnically marginalized populations, has been repeatedly linked to dissociation (Fani & Khalsa, 2023; Harb et al., 2023), and clinically-significant dissociation is also more frequently reported in women (Atilan Fedai & Asoglu, 2022).

3.1. Models of Dissociation

To better understand dissociation, two different models have been proposed. The first, and perhaps most widely supported, theory is the Trauma Model (Bremner, 2010; van Ijzendoorn & Schuengel, 1996), which holds that dissociation may be a useful adaptation in the face of intense trauma. Particularly with chronic traumatic stressors, such as recurring childhood sexual maltreatment and racial discrimination (Amore & Serafini, 2020; Bolduc et al., 2019; Spiegel et al., 2011) dissociation can provide a psychological escape when physical escape is either highly risky or impossible (Boyer et al., 2022; Sel, 1997). This has been proposed as an inverted U-shaped defense mechanism "cascade" response to traumatic threats: "freeze, flight, fight, fright, flag, faint." The first and last stages of this cascade dip below baseline arousal to dissociative states, while the other four represent fear responses controlled by heightened arousal. The

upwards half of the cascade contains the typical “flight-or-fight” responses of hyperarousal and hypervigilance, but the initial “freeze” response is dissociative in nature. On the other hand, the downward portion contains the dissociative symptoms or a “shut-down state” that may occur when sympathetic arousal fails or is more dangerous (Lanius et al., 2018; Schauer & Elbert, 2010). While dissociation may be adaptive at the time, when it becomes entrenched, it can disrupt attentional control, sensory awareness, and memory encoding/retrieval (O. Özdemir et al., 2015), resulting in persistent dissociation and associated psychopathology.

Support for the Trauma Model dates back as early as 1889, in which Pierre Janet said that dissociation was an important defense mechanism used to keep overwhelming traumatic experiences from conscious memory (van der Kolk, 1989). However, some individuals have disagreed with this model, resulting in the creation of another theory. This alternative model, the sociocognitive or fantasy model, suggests that dissociation may be the result of social expectations, heightened suggestibility, fantasy proneness, and cognitive distortions (Giesbrecht et al., 2008). This model widens the scope of dissociation to include individuals who have not experienced trauma but may experience dissociation in response to everyday stressors. Together, these models contribute to a multifactorial understanding of dissociation, in which trauma plays a significant role but also interacts with cognitive deficits and social influences to facilitate pathological dissociation (Lynn et al., 2014).

3.2. Neurobiology of Dissociation

Despite the extensive impact of dissociative phenomena, particularly in marginalized populations of women, there has been proportionally less empirical attention to the neurobiological features of dissociation; consequently, there is a lack of mechanistically-informed treatments for these problems (Sar, 2014). Despite the lack of research, extant

neurobiological models of dissociation in the context of trauma implicate abnormal functional connectivity within pathways involved with emotion regulation, sensation, and social cognition (Menon, 2021; Sierra & Berrios, 1998).

3.2.1. Neurocircuitry of Fear

During exposure to threat, sensory information travels to the "fear matrix" through relay stations such as the thalamus. This matrix includes regulatory structures such as the prefrontal cortex and hippocampus, as well as integrative response structures like the amygdala. This circuit is highly adaptive and biologically conserved, while also flexible. This can be seen in typical "fight-or-flight" responses, where excitatory neurotransmitters are released with the defense response. However, when this stimulation of glutamate and dopamine is excessive, endocannabinoids are released to dampen the fear response, seen as the dissociative "freeze" (Riebe et al., 2012). This is supported by the fact that individuals with less released endocannabinoids exhibit more hyperarousal symptoms and increased attention to threat (Pietrzak et al., 2014).

During chronic and prolonged exposure to threat, active defense mechanisms controlled by the sympathetic nervous system become unsustainable. This is when tonic immobility or the "shut-down state" takes over, resulting in dissociative inhibition of motor responses and reduced awareness of external stimuli. This response is controlled by the parasympathetic nervous system and opioid-mediated analgesia, where the kappa-opioid system shuts down the body so that it can take time to heal without experiencing pain, both physical and emotional. This is thought to be related to the inhibition of the thalamus so that sensory signals do not reach the cortex (Lanius et al., 2018).

3.2.2. Triple Network Model and Relations to PTSD and Dissociation

Psychopathology often involves dysfunction of cognitive and emotion regulation processes reliant on several functional networks within the brain. One of the major theories of connected networks within psychopathology is the triple network model, which implicates the central executive network (CEN) or frontoparietal network (FPN), the default mode network (DMN), and the salience network (SN). The CEN/FPN is involved in working memory, problem-solving, and decision-making, the DMN in memory, emotion regulation, and self-referential mental processes, and the SN in integrating interoceptive, autonomic, and emotional information. The SN acts as an interface between the CEN/FPN and DMN to balance internal mental processes with external cognitive and affective processes (Menon, 2011).

This model has been used to understand the neurobiology of many psychiatric and neurological disorders. Dissociation within the context of PTSD has been associated with hyperconnectivity between the three networks of the triple network model (Lebois, Kumar, et al., 2022) as well as with hyperconnectivity between the amygdala and prefrontal regions including the DMN (Nicholson et al., 2015). More severe trauma-related dissociation symptoms have also been associated with greater connectivity between the DMN and the CEN/FPN, which may suggest maladaptive integration of internal mental processes such as regulating goal-directed behaviors and autobiographical planning (Lebois et al., 2021).

3.2.3. Other Neurobiological Models of PTSD and Dissociation

One integrative model for dissociative symptoms comes from the emotion modulation theory of PTSD (Lanius et al., 2010). According to this model, hyperarousal and re-experiencing symptoms of PTSD are associated with emotional undermodulation, which consists of low activity of the medial prefrontal cortex and failure to inhibit the corticolimbic regions. On the

other hand, dissociative symptoms can be seen as the opposite, in which emotional overmodulation results in excessive inhibition of corticolimbic regions. Research has shown that individuals with the dissociative subtype of PTSD exhibit increased recruitment of neural regions involved in executive control, particularly heightened activation of the dorsal anterior cingulate cortex and medial prefrontal cortex, both involved in modulating arousal and emotion, alongside relatively attenuated activation in limbic regions such as the amygdala (Lanius et al., 2010). These findings suggest that trauma-exposed individuals with more dissociative symptoms rely more heavily on prefrontal mechanisms to regulate arousal, potentially as a compensatory response to extreme and chronic threat.

Dissociative disturbances following trauma have also been linked to abnormal amygdala and prefrontal functional connectivity with the insula (Fani et al., 2018; Harricharan et al., 2020; Rabellino, Densmore, Harricharan, et al., 2018), thalamus (Krystal et al., 1995; Yoshii, 2021), cerebellum (Rabellino, Densmore, Theberge, et al., 2018), and brainstem nuclei (Harricharan et al., 2017). This may lead to disruptions in interoceptive awareness—the conscious detection of afferent visceral signals—which, in turn, can disrupt emotion regulation. The temporoparietal junction has additionally been proposed as a key structure in dissociation due to its role in bodily awareness (Orru et al., 2021), and altered functional connectivity in temporoparietal pathways connecting to this structure has been shown in individuals with elevated dissociation (Harricharan et al., 2016; Olive et al., 2018; Rabellino, Densmore, Theberge, et al., 2018).

3.2.4. White Matter and Dissociation

Although functional neuroimaging studies have provided insights into alterations in brain activation and/or functional connectivity patterns that characterize dissociation, strikingly few studies have examined structural neural features of dissociation, particularly white matter (WM),

which has a protracted period of development and is sensitive to the effects of stress/trauma (Chen et al., 2013; Honey et al., 2010; Reber et al., 2021). Diffusion tensor imaging (DTI) is often used to examine WM microstructure; fractional anisotropy (FA) is a commonly used metric derived from DTI that reflects the strength of directionality of water diffusion (anisotropy) in myelinated WM tracts, with higher values representing greater WM microstructural integrity (Fox et al., 2012; Le Bihan et al., 2001; Mori & Zhang, 2006).

The three extant studies of WM in dissociation had small sample sizes (*N*s~30-52) and comprised all European participants (race was not reported). One study revealed significantly lower FA in the right anterior corona radiata in individuals with dissociative disorders (Kandemir et al., 2016). Another study found that individuals with depersonalization/derealization disorder showed altered microstructural integrity in temporoparietal and frontolimbic pathways involved in multimodal integration and emotion regulation (Sierk et al., 2018). A third study compared individuals with the dissociative subtype of PTSD with a non-dissociative PTSD group and found lower FA in connections between the amygdala, hippocampus, and thalamus, but higher FA in connections between the putamen, pallidum, and ventral diencephalon, as well as connections between the left thalamus, hippocampus and brainstem (Sierk et al., 2021).

As such, functional and structural neuroimaging studies of dissociation reveal alterations within pathways involved in multisensory integration, autonomic processes, emotion regulation, and memory (Lotfinia et al., 2020); this includes WM disruptions, particularly in the anterior corona radiata (Kandemir et al., 2016) and pathways connecting the amygdala, hippocampus, thalamus, basal ganglia, and brainstem (Sierk et al., 2021). The limited WM studies had small samples, focused on WM tracts within a limited scope (e.g., single hemisphere, less than four tracts), and included predominantly European populations. Given that dissociation is a common

and disabling phenomenon that affects many individuals, particularly people who have experienced multiple types of adversity/marginalization (Douglas, 2009), it is imperative to include racially and ethnically marginalized populations in dissociation neuroimaging research. Further, previous studies largely focused on dissociative disorders rather than transdiagnostic dissociation and its facets, which has relevance to a broad range of psychiatric disorders.

2.3. Current Study

Thus, in this study, we examined associations between dissociative symptoms and WM microstructural integrity, assessed via FA, in a large sample (N = 135) of trauma-exposed, majority Black, women who were recruited as part of a long-standing study of trauma, the Grady Trauma Project (GTP) (Gillespie et al., 2009; Schwartz et al., 2005). While not fully generalizable to the general public, GTP's population includes individuals at high risk for dissociation and of a demographic typically excluded from neuroimaging research, making it an ideal sample for expanding dissociation WM research. WM tracts of interest were selected *a priori* based on their relevance to dissociation, trauma, and PTSD in prior studies (Kandemir et al., 2016; Lotfinia et al., 2020; Sierk et al., 2018; Sierk et al., 2021). This includes tracts involved in sensorimotor integration (corona radiata, corpus callosum, internal and external capsule, thalamic radiation, and superior longitudinal fasciculus), emotion regulation (uncinate fasciculus and anterior and posterior cingulum), and memory (fornix).

We examined associations between overall dissociative symptom severity and FA of these pathways; we also conducted secondary analyses to examine potential associations of FA with facets of dissociation, including Disengagement, Depersonalization, Derealization, Emotional Constriction, Memory Disturbance, and Identity Dissociation. Given inconsistent findings in previous literature, we refrained from making directional hypotheses.

We also conducted additional sensitivity and exploratory analyses to better understand our results and the relationship between WM microstructural integrity, dissociation, and PTSD symptoms. Given the strong linear relationship between PTSD and dissociation symptoms (r = .70, p < .001) and the fact that dissociation accompanies PTSD in many individuals (Nijenhuis & van der Hart, 2011; Zucker et al., 2006), collinearity may have influenced the results, which could increase the risk of type II error, so PTSD was not included in the initial analyses. However, for completeness, a sensitivity analysis was conducted that included PTSD symptoms as a covariate. Additionally, given that a large percentage of our participants (84%) identified as Black, a sensitivity analysis identical to the primary and secondary analyses was conducted, except only including Black participants.

To further explore the relationship between dissociation and PTSD symptoms in the context of WM microstructure, sensitivity mediation analyses were conducted: with dissociation and PTSD symptoms both serving as mediators. Finally, exploratory analyses were conducted to better understand the memory facet of dissociation. Given the many different types of memory (Camina & Guell, 2017), we examined the relationship between participants' Memory Disturbance subscale scores, their performance on a neuropsychological test of working memory, and self-reported difficulties remembering aspects of their trauma; this analysis aimed to better identify the specific type of memory assessed by the subscale.

4. Methods

4.1. Participants and Procedure

Clinical and neuroimaging data from 139 adult women (assigned female at birth) were collected as part of GTP (study dates ranging from 2012-2023); however, four individuals were excluded from analyses due to data quality issues resulting in a final sample of 135 women aged

18 to 62 (*M* = 34.25, *SD* = 12.96). Participants were recruited from medical clinics at a publicly funded hospital in Atlanta, Georgia (i.e., Grady Hospital), as well as community advertisements. The sample included 87 participants from prior GTP studies that primarily recruited women (MH101380, MH071537, MH094757) and 48 participants from an ongoing GTP clinical trial for dissociation (Mechanistic Interventions and Neuroscience of Dissociation, MIND; NCT04670640). DTI data from 80 of these participants (59% of total sample) has been published previously in association with PTSD symptoms and racial discrimination (Fani et al., 2022; Fani et al., 2012; Fani et al., 2014; Fani et al., 2016; Haller et al., 2023).

Exclusion criteria for GTP studies were as follows: 1) younger than 18 years of age or older than 65 years of age; 2) currently at risk for suicide, experiencing active psychosis, or cognitively compromised (e.g., intoxication, severe intellectual disability). Inclusion criteria for these studies were as follows: trauma exposure, as defined by the *DSM-IV* (APA, 1994)/*DSM-5* (APA, 2013) Criterion A stressors; age >= 18 years but <= 65 years; the ability to understand English (assessed by a study researcher); willingness to provide informed consent. All participants were screened prior to consent into their respective studies to assess eligibility for clinical trials. Since participants were largely recruited from the GTP and extant projects were recruiting Black women only (e.g., MH101380), a majority of the sample was Black women.

All participants in the current study completed a magnetic resonance imaging (MRI) scan; MRI exclusion criteria were as follows: 1) non-removable ferrous material, 2) prolonged loss of consciousness, and 3) a history of neurological conditions or injuries including strokes, seizures, traumatic brain injury, or history of any disorder with central nervous system involvement (e.g., HIV). Exclusion criteria for the intervention studies were the following: 1) a history of bipolar disorder, schizophrenia, or primary psychotic disorder; 2) current severe

alcohol or substance use disorder, as determined by clinician during diagnostic assessment. Four eligible individuals with clinical and neuroimaging data were excluded from the sample due to poor scan quality based on visual inspection (i.e., shimming artifacts, head motion; $n = 3$) or extreme FA values (mean FA averaged across the ten hypothesized tracts was three standard deviations below the mean; $n = 1$).

After informed consent was obtained, participants completed a battery of self-report demographic questionnaires (age, race/ethnicity), educational attainment, household monthly income, employment status, psychotropic medication usage) and clinical assessments (trauma exposure, PTSD, dissociation; detailed below) with the assistance of a trained researcher, followed by a magnetic resonance imaging (MRI) scan on the same day ($n = 87$) or on a separate visit that occurred within one month of the initial visit ($n = 48$).

4.2. Clinical Assessments

The Multiscale Dissociation Inventory (MDI) (Briere, 2002), a validated and normed 30-item self-report measure, was administered to assess dissociative symptomatology. It yields a total dissociation score (possible score range = 30-150) as well as six subscales: Disengagement, Depersonalization, Derealization, Emotional Constriction/Numbing, Memory Disturbance, and Identity Dissociation (possible score range = 5-25), with higher scores indicating greater dissociative symptomatology. Each item in the MDI is rated according to its frequency of occurrence over the prior month, using a scale ranging from 1-5 with the corresponding rating scale descriptors of “never,” “once or twice,” “sometimes,” “often,” and “very often.” The MDI and its subscales show good internal consistency in the general population: Disengagement (α = .83), Depersonalization (α = .90), Derealization (α = .91), Emotional Constriction/Numbing (α =

.94), Memory Disturbance (α = .74), Identity Dissociation (α = .75), and Total Dissociation Score (α = .96) (Briere et al., 2005).

Trauma exposure was assessed with either of two measures: the Life Events Checklist for *DSM-5* (LEC-5; *n* = 48) (Weathers, Blake, et al., 2013) or the Traumatic Events Inventory (TEI; *n* = 87) (Gillespie et al., 2009). The LEC-5 is a self-report measure used to screen for exposure to 16 potential traumatic events; for each event, participants could state that the event happened to them, that they witnessed, that they learned about it, that it was part of their job, or that it does not apply (Weathers, Blake, et al., 2013). The LEC-5 is a minimal update from the LEC for DSM-IV, and while psychometrics is not available for the LEC-5, there are few expected differences. The test-retest reliability of the LEC is relatively stable overtime, with kappas ranging from .37-.84 with the mean being .61, and the retest correlation being $r = .82$, $p < .001$ (Gray et al., 2004). The TEI is also used to assess trauma exposure and was developed in the context of the GTP, assessing 13 different traumas, as detailed previously (Gillespie et al., 2009). For each event, participants could state that they experienced or witnessed the event, as well as elaborate on the number of times and the ages at which the events occurred. For both trauma exposure measures, total trauma exposure was calculated by summing together the number of experienced Criterion A events. Further details on the types of traumatic events experienced are listed in **Table 1**.

Given that data collection spanned both the *DSM-IV* and *DSM-5*, PTSD symptomatology was assessed with either of two measures: the PTSD Checklist for *DSM-5* (PCL-5; *n* = 48) (Weathers, Litz, et al., 2013) or the modified PTSD Symptom Scale (mPSS; *n* = 87) (Falsetti et al., 1993). For both PTSD measures, higher scores indicated greater PTSD symptom severity. To harmonize the PTSD symptom scales for use in analyses, a conversion equation created using the

combined association test with generalized additive models (ComBat-GAM) algorithm was used to convert mPSS scores to PCL-5, generating a harmonized PTSD symptom score for all participants. The equation used to convert mPSS scores to PCL-5 scores was $S_{PCL\text{-}5} = 3.081 + 1.216(S_{mPSS}) + 0.0077(S_{mPSS}^2)$. This equation yielded a root mean squared error of 1.54 and an R^2 of .986 (Kennedy et al., 2023).

The PCL-5 is a 20-item self-report measure used to assess *DSM-5* symptoms of PTSD (possible score range = 0-80); a shortened version of the PCL-5 without criterion A assessment was used since the LEC-5 was used to assess trauma exposure. Each item is rated according to how much the individual was bothered by it in the past month, using a 5-point Likert scale (0-4) with the corresponding rating scale descriptors of "not at all," "a little bit," "moderately," "quite a bit," and "extremely." PCL-5 total scores have shown strong internal consistency (α = .94) and test-retest reliability (r = .82) (Blevins et al., 2015). The scale also has *DSM-5* symptom cluster severity scores that are obtained by summing the scores from the items for that cluster: cluster B (items 1 - 5), cluster C (items 6 - 7), cluster D (items 8 - 14), and cluster E (items 15 - 20). The cut-off score for probable PTSD is between 31 and 33 as determined by the measure developers (Weathers, Litz, et al., 2013).

The mPSS is a 17-item self-report measure used to assess *DSM-IV* (APA, 1994) symptoms of PTSD (possible score range = 0-51). Each item on the mPSS is rated according to how upsetting the symptom was in the past two weeks on a 4-point Likert scale consisting of 0 ("not at all"), 1 ("once per week or less/a little bit/once in a while"), 2 ("two to four times per week/somewhat/half the time"), and 3 ("five or more times per week/very much/almost all the time"). mPSS total scores have shown good internal consistency (α = .94-.97) and concurrent validity with the Clinician-Administered PTSD Scale (CAPS; r = .57-.87) (Ruglass et al., 2014).

4.2.1. Exploratory Clinical Assessments

Of the 135 participants of the primary and secondary analyses, 116 of them had completed the Letter N-Back (LNB) subtest of the Penn Web-Based Computerized Neurocognitive Battery (WebCNP) (Gur et al., 2010), used to measure attention and working memory. In this task, participants viewed a sequence of flashing letters on a computer screen and responded by pressing the spacebar according to three different conditions: 0-back, 1-back, and 2-back. In the 0-back condition, participants pressed the spacebar whenever the letter "X" appeared. In the 1-back condition, they responded when the current letter matched the one immediately before it (e.g., pressing for the second “R” in the sequence “T, R, R”). In the 2-back condition, they pressed the spacebar when the current letter matched the one presented two positions earlier (e.g., pressing for “T” in the sequence “T, G, T”). Participants had 2.5 seconds to respond in each trial. Before starting the main task, they completed a practice phase for all three conditions. Once they successfully completed the practice, they proceeded to the test phase, which consisted of three blocks of 0-back, 1-back, and 2-back conditions presented in a predetermined order. For each condition, the number of true positives, false positives (spacebar press not following the ruling principle), and median response time (in milliseconds) for correct trials was recorded. On average, the task takes 11.9 minutes to administer. Cronbach’s α for accuracy and speed are 0.77 and 0.82, respectively (Cohen et al., 1999; Gur et al., 2010; Ragland et al., 2002).

Of the 135 participants of the primary and secondary analyses, 105 of them had completed the Clinician-Administered PTSD Scale for *DSM-5* (CAPS-5), a widely used and validated structured diagnostic interview for PTSD (Weathers et al., 2018). The item of interest within the CAPS-5 for assessing disturbances to episodic memory was D1: “Inability to

remember an important aspect of the traumatic event(s) (typically due to dissociative amnesia and not to other factors such as head injury, alcohol, or drugs).” This item contains several follow up questions, and ultimately has a severity rating on a 5-point Likert scale consisting of 0 (“absent”), 1 (“mild/ subthreshold”), 2 (“moderate/threshold”), 3 (“severe/markedly elevated”), and 4 (“extreme/ incapacitating”). While this is only one measure on the scale, the CAPS-5 has good convergent validity with the previous iteration, CAPS-IV (r = .83), and the PTSD Checklist for *DSM-5* (r = .66), as well as good discriminant validity with measures of anxiety, depression, somatization, functional impairment, psychopathy, and alcohol abuse (rs = .02 to .54) (Weathers et al., 2018).

4.3. MRI Acquisition and Image Processing

MRI scans were acquired on four different research-dedicated Siemens 3-Tesla MRI systems (three Tim Trio scanners and one Prisma^{fit} scanner that underwent a software update mid-study). Scanner details and acquisition parameters for each of the five scanners used are listed in **Table 2**. Sequence parameters were altered slightly to accommodate different scanner models but were largely the same except for slight differences in the T1-weighted modality and number of axial slices. Diffusion weighting was isotropically distributed along 60 directions using a b-value of 1,000 s/mm^2. Four normalization images, with no diffusion encoding (b=0), were acquired and averaged for each direction using linear rigid-body registration (FLIRT) (Jenkinson et al., 2002; Jenkinson & Smith, 2001). All diffusion-weighted image processing and analysis were conducted using FMRIB Software Library (FSL version 4.1; www.fmrib.ax.ac.uk/fsl) (Woolrich et al., 2009).

Correction for head motion and eddy current distortion was performed for data from each individual participant using an automated affine registration algorithm. Normalization images

were skull-stripped using the FSL brain extraction tool (Smith, 2002). All data were visually inspected for artifacts (i.e., shimming artifacts, head motion), resulting in an exclusion of three participants from the initial 139. Formal quality checks were also performed on the data by calculating the temporal signal-to-noise ratio (tSNR) across each diffusion volume (Roalf et al., 2016). tSNR was calculated for each scan using the python nibabel package v.5.1 and tSNR maps were created for each participant by dividing the mean signal of the diffusion weighted data by the standard deviation of the signal, and then extracting the mean tSNR value from the map. Maps were created only using the b-value shell of 1,000 as that was the volume for diffusion weighted imaging.

FA maps were generated using the DTIfit in the FMRIB Diffusion Toolbox, and all maps were aligned for data extraction using tract based spatial statistics (TBSS; version 1.2), an approach that increases the sensitivity and interpretability of the results because it uses non-linear registration (Smith et al., 2006). All participants' FA maps were co-registered using the non-linear registration to the most 'typical' participant's FA [as determined by FMRIB's nonlinear image registration tool (FNIRT) (Andersson et al., 2007)], then affine transformed into 1×1×1 mm MNI space. All transformed FA images were averaged to create a mean FA image, then thresholded by FA > 0.2 to create a WM skeleton to ensure that gray matter regions would be excluded from data extractions.

Binarized masks for the ten bilateral tracts of interest (**Figure 1**) were created and used to extract the mean FA value for each tract from each participant. The atlases used were the probabilistic Johns Hopkins University White Matter Atlas (Mori et al., 2005), the ICBM-DTI-81 white-matter labels atlas (Burgel et al., 2006), and the Juelich Histological Atlas (Burgel et al., 2006). Multiple atlases were used since all tracts of interest were not provided in a single

atlas and bilateral masks were created by merging the lateralized tracts in FSLmaths. Because of differences in scanner software and acquisition parameters, there were significant mean differences (*p*s<.05) between the extracted FA values for each of the five scanner conditions (**Table 3**). Differences in FA and tSNR values across each scanner were harmonized using ComBat-GAM, now referred to as neuroHarmonize (Pomponio et al., 2020). Participants with extreme mean FA values (average FA for all ten tracts three standard deviations below the sample mean; *n*=1) were excluded from analyses.

4.4. Statistical Analyses

Using the IBM Statistical Package for Social Sciences (SPSS) v.29, we first examined associations between participant demographics and overall dissociation severity (MDI total score), as well as harmonized FA data of each of the ten tracts of interest. We then examined partial correlations between harmonized FA data and MDI total and subscale scores while covarying age and harmonized tSNR. Age was included as a covariate given its potential relationship with WM microstructure (Gunning-Dixon et al., 2009; Lebel et al., 2019), and tSNR was included as a covariate to ensure associations were not driven by individual differences in data quality. To correct for multiple comparisons across all partial correlations (MDI total and subscale scores), we used false discovery rate (FDR) correction (Benjamini & Hochberg, 1995), with FDR-corrected $p < .05$ indicating statistical significance.

PTSD symptoms were not covaried in primary analyses due to multicollinearity concerns; however, for completeness, a sensitivity analysis was conducted that included harmonized PCL-5 score, age, and tSNR as covariates (FDR-corrected $p < .05$ indicated statistical significance). A further sensitivity analysis was conducted with only participants who identified as Black, with analyses identical to the primary and secondary analyses, except for the

reduced sample. (FDR-corrected $p < .05$ indicated statistical significance). Sensitivity mediation analyses were also conducted to test the relationship between the FA values of the tracts of interest, harmonized PCL-5 scores, and MDI total scores: first with MDI as the mediator then with PCL-5 as the mediator. Ten thousand bootstrapped samples were used to generate a 95% confidence interval for the indirect effects, which were considered statistically significant if the confidence interval did not include zero. These mediation models were conducted using the PROCESS macro in SPSS using model 4 (Hayes, 2022). Uncorrected $p < .05$ was used to determine statistical significance for paths a, b, c, and c' in each mediation model.

Significant findings were noted if they fell below an α level of .005 to indicate the findings that might survive a more stringent correction (though no formal correction method was applied). Results were interpreted for each set of mediation models, whether MDI or PCL-5 was the mediator, to understand which measure better explained their overlapping relationship with FA in each of the ten tracts of interest. Note that path a for each set of models will be identical across each of the ten tracts because this is the relationship between the self-report measures and that path b from one set of models is identical to path c' from the other set of models. The primary effects of interest in these complimentary mediation models are the indirect effects, which were standardized to ensure confidence intervals had enough range to detect whether zero was included (unstandardized confidence in PROCESS were most frequently output as .0000 in these models due to FA values being quite small). The other effects of interest were path b and path c' (direct effects).

4.4.1. Exploratory Statistical Analyses

To assess the relationship between working memory and dissociation, MDI total score and all subscales were correlated with the number of true positives, number of false positives,

and reaction time for each condition of the LNB. To assess the relationship between trauma-specific episodic memory and dissociation, MDI total score and all subscales were correlated with the item D1 CAPS-5 severity rating. Given the established trend of age-related decline in working (Kirova et al., 2015) and episodic memory (Kinugawa et al., 2013), these relationships were assessed using partial correlations while covarying age. For these exploratory analyses, $p < .05$ indicated statistical significance.

5. Results

Demographic and clinical characteristics for the sample are provided in **Table 4**. Dissociation severity (MDI total score) showed a significant association with age [$r(133) = -.41$, $p < .001$] and harmonized PTSD scores [$r(133) = .70$, $p < .001$], but not with education [Spearman's $\rho(133) = .11$, $p = .20$] or income [Spearman's $\rho(130) = .10$, $p = .24$]. All MDI subscale scores also showed a positive association with harmonized PTSD scores (rs > .32, ps < .001), as well as with one another (rs > .28, ps <= .001), with the strongest correlation between Depersonalization and Derealization, and the weakest between Disengagement and Identity Dissociation. Age was significantly and negatively correlated with FA for six tracts (rs < -.20, ps < .02) except the corpus callosum, posterior cingulum, fornix, and internal capsule (rs > -.16, ps < .06). All correlations between harmonized PTSD scores and MDI subscales are in **Table 5** and all correlations between harmonized FA and demographic characteristics are in **Table 6**.

5.1. Primary Analysis

Fractional anisotropy of seven of the ten hypothesized tracts showed a significant negative association with overall dissociation severity (MDI total score), as shown in **Figures 2** and **3**, including the corona radiata [$r(131) = -.39$, $p_{FDR} < .001$], corpus callosum [$r(131) = -.35$, $p_{FDR} < .001$], SLF [$r(131) = -.31$, $p_{FDR} < .001$], thalamic radiation [$r(131) = -.31$, $p_{FDR} =$

.002], anterior cingulum [$r(131) = -.30$, $p_{FDR} = .002$], fornix [$r(131) = -.23$, $p_{FDR} = .02$], and uncinate fasciculus [$r(131) = -.19$, $p_{FDR} = .05$]. The other tracts showed no significant association with MDI total score ($rs > -.19$, $ps_{FDR} > .05$).

5.2. Secondary Analyses

MDI subscale analyses showed that FA was most consistently associated with the MDI Memory Disturbance subscale, with a significant and negative correlation with nine of the ten tracts ($rs < -.23$, $p_{FDR} < .02$). The MDI Disengagement subscale was associated with FA in seven tracts ($rs < -.20$, $p_{FDR} < .04$) followed by FA of six tracts being associated with the MDI Depersonalization and Derealization subscales ($rs < -.20$, $p_{FDR} < .04$). The MDI Emotional Constriction subscale was associated with FA in two tracts ($rs < -.21$, $p_{FDR} < .03$) and the MDI Identity Dissociation subscale was not associated with FA in any of the ten tracts ($rs > -.14$, $p_{FDR} > .17$). Full results are shown in **Figure 2** and **Table 7**.

5.3. Sensitivity Analyses

After covarying age, tSNR, and harmonized PCL-5 scores, harmonized tract FA was only associated with the MDI Disengagement and Memory Disturbance subscales. MDI Disengagement was significantly and negatively correlated with FA of two tracts: the corona radiata [$r(130) = -.29$, $p_{FDR} = .03$] and the corpus callosum [$r(130) = -.29$, $p_{FDR} = .03$]. MDI Memory Disturbance was significantly and negatively correlated with FA of one tract: the corona radiata [$r(130) = -.28$, $p_{FDR} = .03$]. Full results are listed in **Table 8**.

When analyses were restricted to only participants who identified as Black, results were largely the same as the primary analyses. FA of the internal capsule significantly associated with MDI Total score [$r(130) = -.21$, $p_{FDR} < .05$] while FA of the uncinate fasciculus was no longer significantly associated with MDI Total Score [$r(130) = -.21$, $p_{FDR} = .05$]. FA of the internal

capsule was no longer significantly associated with the MDI Disengagement subscale and FA of the external capsule was no longer significantly associated with the MDI Memory Disturbance subscale. FA of the corpus callosum and thalamic radiation showed significant negative relationships with MDI Emotional Constriction subscale. Full results are listed in **Table 9**.

Results from the mediation models testing MDI score as a mediator of the relationship between PCL-5 score and tract FA for each of the ten tracts showed that MDI had a significant indirect effect on FA of three tracts (corpus callosum, corona radiata, thalamic radiation). The effect of MDI on FA (path b) was significant for two tracts (corpus callosum, corona radiata). The direct effect of PCL-5 (path c') was not significant for FA of any of the ten tracts. Results from the mediation models testing PCL-5 score as a mediator of the relationship between MDI score and tract FA for each of the ten tracts showed no significant indirect effects. The effect of PCL-5 on FA (path b) was not significant for any of the ten tracts. The direct effect of MDI (path c') was significant for two tracts (corpus callosum, corona radiata). Based on these results, there is evidence that MDI score mediates the relationship between PCL-5 and FA for multiple tracts but not vice versa. Full results are listed in **Table 10** (MDI as the mediator) and **Table 11** (PCL-5 as the mediator).

5.4. Exploratory Analyses

Performance on the LNB task was not significantly associated with the MDI Memory Disturbance subscale. Only the Disengagement subscale was significantly associated with LNB task performance, showing a negative correlation with total LNB false positives [$r(113) = -.25$, $p = .01$] and LNB 0-back false positives [$r(113) = -.24$, $p = .01$]. Trouble remembering aspects of the traumatic event(s), assessed using the CAPS-5 item D1, was not significantly associated with the MDI Memory Disturbance subscale [$r(102) = .10$, $p = .30$]. CAPS-5 item D1 was positively

and significantly associated with MDI total score [$r(102) = .25$, $p = .01$] as well as all other subscale scores (rs > .24, ps < .02), except for Emotion Constriction [$r(102) = .13$, $p = .21$]. Full results are listed in **Table 12.**

6. Discussion

6.1. Review of Study

The present study is the first, to our knowledge, to investigate associations between transdiagnostic dissociation severity and WM microstructure in a large, predominantly Black, trauma-exposed sample of women. Results showed that greater overall dissociation severity was associated with proportionally lower FA among six of the ten hypothesized WM tracts: the corona radiata, corpus callosum, SLF, thalamic radiation, anterior cingulum, fornix, and uncinate fasciculus. Among the different facets of dissociation, memory disturbance was most consistently correlated with FA, revealing a negative association within nine of the tracts. These findings reveal links between dissociative phenomena and disruptions in pathways involved with attention, memory, sensorimotor integration, and emotion regulation; disruptions in these tracts represent salient neuropathophysiological features of dissociation, and likewise, targets for psychotherapeutic and neuromodulatory interventions.

6.2. Previous Literature

Although previous literature on WM correlates of dissociation is limited, our findings were generally consistent with prior studies, showing that dissociation was linked to lesser structural integrity within temporoparietal, frontolimbic, and subcortical striatal–brainstem pathways (Lotfinia et al., 2020; Sierk et al., 2018). The direction of our findings contrast with some findings from two prior studies, which showed that dissociation was linked to higher FA in connections between basal ganglia nuclei, thalamus, and hippocampus in individuals with the

dissociative subtype of PTSD (Sierk et al., 2021) or between the left superior frontal gyrus, right medial frontal cortex, and the right amygdala (Sierk et al., 2018); however, these results were restricted to subcortical or cross hemisphere connections rather than full frontolimbic circuits, and differences in FA estimation methods, limited sample size, and population differences (European sample) may have influenced these contrasting results.

6.3. Primary and Secondary Analyses

We observed some of the strongest associations between dissociation severity and FA of tracts involved in sensorimotor integration: the corona radiata, corpus callosum, thalamic radiation, and SLF. The corona radiata represents a large, fan-shaped network of WM fibers radiating out from the internal capsule, extending out to the cortex and descending to subcortical brainstem and thalamic nuclei (Yakar et al., 2018); as such, it supports a large volume of neuronal activity subserving different processes ranging from executive functioning (e.g., attentional control) (Niogi et al., 2010; Stave et al., 2017) to perception, sensory integration, and motor control (Chang et al., 2016; Curcic-Blake et al., 2015; Jiang et al., 2019; Owen et al., 2013; Yin et al., 2018). Alterations in corona radiata microstructure have been observed in transdiagnostic meta- and mega-analytic studies of psychiatric disorders associated with dissociation, particularly schizophrenia (Renard et al., 2017), a disorder that is characterized by sensory/perceptual dysfunction and cognitive impairments (Burke et al., 2023; Kelly et al., 2018; Koshiyama et al., 2020; Zhao et al., 2022); these findings align with results from a prior study of WM in dissociative disorders (Kandemir et al., 2016). The SLF, a major temporoparietal pathway connecting the parietal lobe to secondary motor areas is involved in attention, memory, emotion processing, and sensorimotor integration (Kamali et al., 2014; Vergani et al., 2021). Decrements in the SLF have been shown in populations who have endured racial discrimination

(Fani et al., 2022) and in adults with PTSD (O'Doherty et al., 2018; Siehl et al., 2018) and dissociative disorders (Sierk et al., 2018).

The corpus callosum, the major commissural pathway connecting the left and right hemispheres of the brain (Goldstein et al., 2024) is crucial for processing and integrating sensory, motor, and cognitive signals (Tzourio-Mazoyer, 2016) and has been implicated in schizophrenia as well as memory, mood, and intellectual impairments (Ghavipisheh et al., 2023; Piras et al., 2021), including PTSD (Graziano, Bruce, & Paul, 2021). Abnormal information relay across the brain's two hemispheres through the corpus callosum has also been implicated in trauma-related disorders, as seen by reduced integrity to the structure (Siehl et al., 2018; Villarreal et al., 2004). The thalamic radiation connects the thalamus to the cerebral cortex and is involved in relaying sensory and motor information (Jones, 2002). Cortico-thalamic circuits within this tract have been observed to mediate shifts in attentional states, ranging from high arousal alertness to low arousal and sleep states (Zikopoulos & Barbas, 2007), which is likely the tract involved arousal shifts for dissociative responses. Alterations to this pathway have been associated with cognitive impairments in schizophrenia (Mamah et al., 2010) and have been found in survivors of childhood maltreatment (Lim et al., 2020).

As previously mentioned, peritraumatic dissociation in response to traumatic events is associated with the release of anesthetic neurochemicals that disrupts communication throughout the brain, ultimately impairing normal conscious experience (Smith, 2015). While transient experiences of dissociation may occur via sudden inhibition of fear responses, such as through increased regulatory prefrontal cortex function to suppress amygdala activity, endocannabinoids and opioids may contribute to persistent dissociation. Given that cannabis use has been associated with reduced FA in the SLF and corpus callosum (Robinson et al., 2023), and that

opioid-dependent patients tend to have lower FA in many tracts involved in motor function such as the corona radiata, corpus callosum, and thalamic radiation (Upadhyay et al., 2010), there is evidence to suggest that prolonged release of these chemicals in response to stress may damage WM. Additionally, decrements to these information relay WM tracts may contribute to the continuing of dissociative responses even after the initial traumatic experience.

6.3.1. Sensitivity Analyses

Our mediation analyses revealed that dissociation played a more prominent role than PTSD in decrements observed for FA in the CC and corona radiata, shown by the significant direct and indirect effects of MDI on FA of those tracts. While some of the decrements to other significant WM tracts, such as the SLF, may be similarly impacted by both dissociation and PTSD, those involved in sensorimotor integration appear to be particularly influenced by dissociation. Notably, PTSD symptoms did not mediate the relationship between dissociation and FA in any tracts, suggesting that changes to WM microstructural integrity in PTSD may be contingent on the presence of dissociation. This mediation pathway is supported by the fact that dissociation serves as a risk factor for developing PTSD (Atchley & Bedford, 2021),

Further examination of this relationship for the dissociation subscales showed that disengagement and memory disturbance remained significant with FDR correction, even when covarying for PTSD, suggesting these facets capture distinct dissociative features that are not solely attributable to PTSD symptomatology. This distinction is especially relevant given that depersonalization and derealization are often considered core dissociative symptoms within the PTSD dissociative subtype (Association, 2013) and tend to be captured within measures of PTSD symptoms. On the other hand, facets of dissociation looking at cognitive disruptions such as attentional and memory deficits are often seen as opposite to hyperarousal and reexperiencing.

Moreover, WM changes in the brain do not appear to be markedly different when looking at only Black individuals in our sample. While numbers of non-Black individuals were too small to make meaningful analyses as to how race may impact FA microstructure, our sensitivity analysis suggests that race-related factors did not largely influence the relationship between dissociation and FA. Racial discrimination may be one of the most prominent chronic stressors associated with dissociation, but other stressors, like childhood sexual maltreatment, which are not specific to any racial group, can have similar long-term symptoms (Amore & Serafini, 2020; Bolduc et al., 2019; Fani & Khalsa, 2023; Harb et al., 2023; Spiegel et al., 2011) and WM differences.

6.3.2. Exploratory Analyses

Exploratory analyses revealed that the MDI Memory Disturbance subscale was not associated with measures of working or episodic memory. Both measures had a reduced sample, which may have influenced power, as well as episodic memory being assessed using only a single question from the CAPS-5. However, MDI disengagement was associated with better performance on the LNB, specifically 0-back trials. This finding aligns with prior research suggesting that highly dissociative individuals may struggle with attentional control in emotionally evocative tasks such as visual memory, but demonstrate comparable—or even superior—executive functioning in neutral conditions (Fani et al., 2019). Additionally, the LNB is not the most reliable assessment of working memory and may be getting at other higher order cognitive functions such as fluid intelligence (Jaeggi et al., 2010).

Additionally, trouble remembering aspects of the traumatic event was associated with increased dissociative symptomatology in all facets but memory disturbance and emotion constriction. Memory fragmentation is often associated with peritraumatic dissociation (Bedard-

Gilligan & Zoellner, 2012), but we only have measures of current dissociation and are uncertain as to whether the dissociation is persistent or if they experienced peritraumatic dissociation. Despite this, fragmentation was associated with other facets of dissociation, indicating that processes such as disengagement and depersonalization/derealization may have interfered with memory encoding of the traumatic event, suggesting the presence of peritraumatic dissociation.

Chronic stress can cause an immune response that inhibits the hippocampus, which has been hypothesized to account for declarative memory deficits following traumatic events (Brandes et al., 2002; McEwen, 2000). Notably, endocannabinoids, responsible for fear suppression and dissociation, are highly concentrated in the hippocampus, suggesting a mechanism of improper memory encoding that is associated with peritraumatic dissociation (Scarante et al., 2017). However, our results were less focused on WM tracts connecting to the hippocampus, such as the fornix, but rather tracts involved in sensorimotor integration. This suggests that the inhibition of the hippocampus may be localized and not affect surrounding WM, and also suggests that memory disruptions, as measured in the MDI, may be less due to failure to encode memories but rather failure to accurately process the events. Psychometrics of the MDI align with this hypothesis as the Memory Disturbance subscale also loaded onto the Disengagement subscale and may represent other sources of memory disruption such as attentional control deficits or neurodegenerative diseases (Briere et al., 2005; Jeffirs et al., 2023).

Overall, exploratory results indicate that memory disturbance may reflect a broader disruption in cognitive processing instead of deficits specific to working or episodic memory. Rather than impairing memory retrieval for specific periods, such as during a measured task or a traumatic event, these disruptions may reflect general lapses in attention and moment-to-moment memory failures. This aligns with the idea that dissociative symptoms involve a disconnect from

present experiences, which may interfere with encoding and recall but in a way that is distinct from traditional memory impairments. Given that the inhibitory thalamic reticular nucleus serves as an “emotional gatekeeper” that can inhibit thalamo-cortical signaling to divert attention during highly emotional events, such as trauma (John et al., 2016), it is likely that sensory input may never reach the cortex for further processing. As a result, if the event is not fully consciously experienced, it may fail to be properly encoded, even without hippocampal dysfunction. This inhibition of the thalamus is not even reliant on high stress neurochemical release as many prefrontal projections have GABAergic synapses onto the thalamus (Zikopoulos & Barbas, 2006), which may explain sensory disconnect and diverted attention during transient and seemingly unwarranted dissociative experiences.

Additionally, while different facets of dissociation such as disengagement, depersonalization, and memory disturbance are often related, they do not necessarily co-occur or influence cognition in the same way. This suggests that dissociation may manifest in distinct cognitive profiles, with some individuals experiencing attentional control deficits, while others exhibit more severe disruptions in memory or sensory integration. This highlights the importance of viewing dissociation through a multifaceted lens, as each domain of dissociation may have unique neurobiology with different cognitive processes involved.

6.4. Implications

Overall, these findings build upon prior studies of WM in dissociation and demonstrate that dissociation severity was linked to decrements in microstructural integrity of tracts involved with sensorimotor integration, memory, and emotion regulation in trauma-exposed women. As persistent dissociation has been associated with negative mental health outcomes (Atchley & Bedford, 2021; Lebois, Harnett, et al., 2022), there is a clear need for robust biomarkers of

dissociation, which may serve as clinical targets. Further, studies show that many people with elevated dissociative symptoms experience significant treatment barriers, including a lack of trained treatment providers and stigma-related barriers (Nester et al., 2022); this problem is exacerbated in marginalized populations, including populations of women and Black individuals (Ballone & Richards, 2023; Snowden, 2001). As such, the implicated tracts of this study represent attractive candidate biomarkers for dissociation and may be useful targets for treatment with respect to psychotherapy and neuromodulation.

We observed particularly consistent and strong associations between the Memory Disturbance and Disengagement subscales and FA in various white matter pathways. While memory disturbance was not linked to trauma-related memory fragmentation, dissociation—particularly disengagement—was associated with difficulty recalling aspects of trauma, suggesting a role in memory fragmentation in dissociation. Trauma-focused psychotherapy, (e.g., prolonged exposure therapy), can be used to address these problems, as it involves processing of trauma memories (Foa et al., 2005), and has been shown to decrease dissociative symptoms (Hagenaars et al., 2010). Changes in frontolimbic, thalamic, and commissural WM tracts have been observed in individuals who completed Trauma-Focused Cognitive Behavioral Therapy (TF-CBT) (Korgaonkar et al., 2021), and a case study using an adapted form of TF-CBT (i.e., adjunctive dissociation psychoeducation and emotion regulation) demonstrated benefit for an adult patient with dissociative PTSD (Vancappel et al., 2022). Although the effects of trauma-focused therapy on WM integrity in those with elevated dissociation have not been examined, guided imagery psychotherapy has been shown to increase FA of the left superior frontal cortex in adults with major depressive disorder (Wang et al., 2013), which could support the assertion

that trauma therapy treatment success in individuals with dissociation is related to improvements to WM integrity of frontolimbic tracts.

Given the connection between memory disturbance and disengagement, prevention of future memory deficits may also be achieved by helping dissociative individuals increase their awareness of the present moment. Self-regulation of attention and acceptance of internal experiences through mindfulness-based interventions have been shown to be effective in reducing dissociation (Vancappel et al., 2021). Additionally, mindfulness has been shown to help diminish age-related degeneration of limbic WM tracts (Laneri et al., 2016), and as PTSD has been hypothesized to accelerate aging (Wang et al., 2022), mindfulness may help diminish WM decrements observed in dissociation following trauma exposure. Degradation in these WM tracts may also correspond to dysfunction in sensorimotor processing and elevated disconnection between the mind and body, which is common in trauma-related dissociation (Kearney & Lanius, 2022). Thus, mindfulness training, such as mindful awareness in body-oriented therapy (MABT), may also help to strengthen these relevant pathways to improve interoceptive awareness and mind-body connection (Price & Hooven, 2018) to reduce dissociative symptoms. Finally, physical activity has been shown to increase the integrity of various frontal lobe WM tracts (Bashir et al., 2021), which may be a more accessible treatment option.

Our findings reveal salient targets for neuromodulation as well. Deep brain stimulation (DBS) of WM in the subgenual cingulate has provided antidepressant effects for patients with treatment-resistant depression (Mayberg et al., 2005), and DBS of the subgenual cingulum and uncinate fasciculus has produced reductions in PTSD symptoms (Hamani et al., 2022). Non-invasive neuromodulation techniques, including repetitive transcranial magnetic stimulation of anterior cingulate and prefrontal WM, have also shown positive effects on FA in these regions

and provided therapeutic effects in patients with depression (Ning et al., 2022). Given the links observed between FA in the anterior cingulum and the uncinate fasciculus and dissociative memory disturbances, these tracts may be excellent targets for neuromodulation. Additionally, the therapeutic effects of stimulation of frontal lobe tracts in patients with PTSD and depression indicate that neuromodulation, whether via DBS or non-invasive stimulation methods, of our other significant tracts has the potential to provide relief for dissociation, and merit attention in future research.

6.5. Limitations and Future Directions

We acknowledge several important limitations in this study. First, the correlational nature of our study precludes any causal inferences (whether dissociation influences the development of WM disruptions, or vice versa). The high correlation between the MDI and PCL-5 should be also considered when interpreting our results, but sensitivity mediation analyses indicated a unique role of dissociation. Additionally, our study sample comprised of mostly Black individuals who identified as women. Although this could potentially have implications for generalizability to other trauma-exposed populations, this may also be considered a strength, as Black women are disproportionately exposed to multiple types of adversity and show disproportionately high rates of PTSD and dissociation (Douglas, 2009; Gran-Ruaz et al., 2022; Meshberg-Cohen et al., 2016); they are also an underrepresented group in psychiatric neuroimaging research. Additionally, dissociation may be a response to racial adversities (Fani & Khalsa, 2023; Harb et al., 2023), so examining biomarkers of dissociation in this racially marginalized population has clinical salience. Regardless, conducting analyses with a more equal portion of races could be useful for generalizability, as well as analyzing race-related factors. Beyond race, our sample also did not include any individuals with military trauma. While community trauma is important

to consider in trauma studies, military populations are the hallmark of trauma studies, and tend to report higher rates of dissociation than those without combat experience (B. Özdemir et al., 2015), making them an important population to include in future dissociation and WM research.

There are also different kinds of additional information that we did not have access to that would be beneficial for further understanding our results. Firstly, since we had to harmonize PTSD scales to get a single measure of symptomatology, we were unable to analyze subscale scores to see how they may interact with PTSD. We ran into the same issue with trauma exposure, and having more detail specific to time since last trauma as well as non-Criterion A stressors, such as racial discrimination, would be helpful. While we excluded individuals with severe alcohol or substance use disorder and the MDI questions specified that the experiences are not due to drug or alcohol use, we did have additional information on substance use to include as a covariate in analyses. This would be an important variable to consider given that alcohol consumption is associated with reduced FA (Daviet et al., 2022). However, addiction is common among those with childhood trauma and dissociation (Craparo et al., 2014). Ecological validity is an important concept here; while variables such psychotropic medication usage and substance use may impact WM, individuals at higher risk for dissociation are more likely to use medications and substances. Not excluding for these variables helps increase sample size and make it more generalizable to dissociative populations, and not covarying these variables helps keep the meaningful variation in dissociative symptoms.

Finally, to better understand the cognitive mechanisms underlying dissociative memory disturbance, additional neuropsychological tests of attention, working memory, and declarative memory would be imperative. A more comprehensive understanding of executive functioning could be obtained using The Montreal Cognitive Assessment (Nasreddine et al., 2005), and

attentional control during emotionally evocative tasks could be measured using tests such as Trier Social Stress Test for emotional memory (Freund et al., 2023) or visual memory tasks using images from the International Affective Picture System (Taylor et al., 1998). Given the importance of sensorimotor integration to dissociation, sensory memory could be an interesting domain to examine, using tests such as Sperling's Sensory Memory Experiments (Sperling, 1960) or tactile cognitive tests used to assess Alzheimer's patients (Arambula et al., 2021). Finally, to better assess episodic and declarative memory, particularly measures of everyday events, there are many possible tasks, such as the Rivermead Behavioral Memory Test (Kurtz, 2011), the Rey Auditory Verbal Learning Test (Vakil & Blachstein, 1993), or the SEMantic and EPisodic Memory Test (Vallet et al., 2017).

Beyond expanding upon the current study, future directions should analyze the specific neurobiology of WM decrements in dissociative populations. FA is a relatively non-specific metric of WM microstructure; other methods such as Neurite Orientation Dispersion and Density Imaging and diffusion kurtosis imaging may provide other useful metrics of WM morphological alterations (Kamiya et al., 2020) to better understand how and why these structural differences occur. Additionally, animal models are an important method to use to gain a better understanding of brain changes that occur amid dissociation. While animals cannot say whether they feel disconnected from their body, the biologically conserved stress-induced analgesia, fear extinction, learned helplessness, and tonic immobility are useful processes to study.

6.6. Conclusion

In conclusion, we observed that dissociation was characterized by disruptions in WM microstructure in this large sample of trauma-exposed women, particularly among tracts that subserve information relay and processing, sensory integration, and memory: the corona radiata,

corpus callosum, SLF, thalamic radiation, anterior cingulum, fornix, and uncinate fasciculus. These stress-sensitive pathways represent promising targets for future psychotherapeutic and neuromodulatory interventions for a broad range of dissociative trauma-exposed populations, particularly those who have experienced multiple types of adversity and marginalization.

7. References

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Table 1. Types of traumas experienced by percentage and frequency

1A. Trauma types assessed with the Life Events Checklist (n = 48)

Trauma Type	**% (*n*)**
Natural Disaster	27.1 (13)
Fire or Explosion	8.3 (4)
Transportation Accident	45.8 (22)
Serious Accident	18.8 (9)
Toxic Substance Exposure	4.2 (2)
Physical Assault	41.7 (20)
Assault with a Weapon	14.6 (7)
Sexual Assault	83.3 (40)
Combat or War	0.0 (0)
Captivity	2.1 (1)

1B. Trauma types assessed with the Traumatic Events Inventory (n = 87)

Trauma Type	**% (*n*)**
Natural Disaster	29.9 (26)
Serious Accident or Injury	57.5 (50)
Sudden Life-Threatening Illness	20.7 (18)
Violence without a Weapon	79.3 (69)
Violence with a Weapon	43.7 (38)
Childhood Physical Abuse	50.6 (44)
Childhood Sexual Abuse	43.7 (38)
Sexual Assault	26.4 (23)

1C. Trauma types endorsed among all participants (n = 135)

Trauma Type	**% (*n*)**
Natural Disaster	29.1 (39)
Serious Accident or Injury	53.7 (72)
Violence without a Weapon	55.6 (75)
Violence with a Weapon	32.8 (45)
Sexual Assault	47.0 (63)

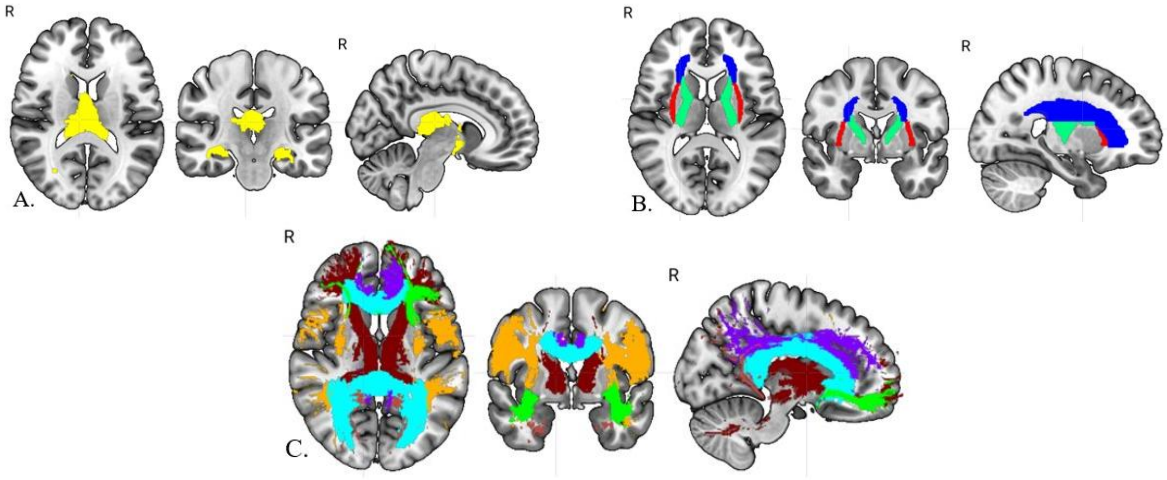
Table 2. Scanner details and acquisition parameters

	Scanner 1	Scanner 2	Scanner 3	Scanner 4	Scanner 5
Scanner Type	TIM-Trio	TIM-Trio	Prisma^{fit}	Prisma^{fit}	TIM-Trio
Software	MR_VB17A	syngo MR B17	syngo_MR_E 11	syngo MR XA30	MR_VB17A
Head Coil	HeadMatrix_ 12 Coil	HeadMatrix_ 12 Coil	32-Channel	32-Channel	HeadMatrix_ 12 Coil
Number of Subjects	31	7	32	16	49
T1-Weighted Modality	**TR** = 2600ms, **TE** = 3.02ms, **TI** = 900ms, **Flip angle** = 8 deg, **Slices** = 176, **FOV** = 256mm, **Voxel size** = 1.0mm x 1.0mm x 1.0mm	**TR** = 2600ms, **TE** = 3.02ms, **TI** = 900ms, **Flip angle** = 8 deg, **Slices** = 176, **FOV** = 256mm, **Voxel size** = 1.0mm x 1.0mm x 1.0mm	**TR** = 2500ms, **TE** = 2.22ms, **TI** = 1000ms, **Flip angle** = 8 deg, **Slices** = 208, **FOV** = 256mm, **Voxel size** = 0.8mm x 0.8mm x 0.8mm	**TR** = 2500ms, **TE** = 2.22ms, **TI** = 1000ms, **Flip angle** = 8 deg, **Slices** = 208, **FOV** = 256mm, **Voxel size** = 0.8mm x 0.8mm x 0.8mm	**TR** = 2600ms, **TE** = 3.02ms, **TI** = 900ms, **Flip angle** = 8 deg, **Slices** = 176, **FOV** = 256mm, **Voxel size** = 1.0mm x 1.0mm x 1.0mm
Diffusion-Weighted Modality	**Maximum gradient strength** = 40mTm^{-1}, **thick axial slices** = 39 x 2.5 mm, **matrix** = 128 x 128, **FOV** = 220 x 220 mm, **Voxel size** = 1.72 x 1.72 x 2.5 mm	**Maximum gradient strength** = 40mTm^{-1}, **thick axial slices** = 40 x 2.5 mm, **matrix** = 128 x 128, **FOV** = 220 x 220 mm, **Voxel size** = 1.72 x 1.72 x 2.5 mm	**Maximum gradient strength** = 40mTm^{-1}, **thick axial slices** = 40 x 2.5 mm, **matrix** = 128 x 128, **FOV** = 220 x 220 mm, **Voxel size** = 1.72 x 1.72 x 2.5 mm	**Maximum gradient strength** = 40mTm^{-1}, **thick axial slices** = 39 x 2.5 mm, **matrix** = 128 x 128, **FOV** = 220 x 220 mm, **Voxel size** = 1.72 x 1.72 x 2.5 mm	**Maximum gradient strength** = 40mTm^{-1}, **thick axial slices** = 39 x 2.5 mm, **matrix** = 128 x 128, **FOV** = 220 x 220 mm, **Voxel size** = 1.72 x 1.72 x 2.5 mm
Location	Biomedical Imaging Technology	Center for Advanced Brain Imaging	Center for Advanced Brain	Center for Advanced Brain	Center for Systems Imaging at

	Center at Emory University	at Georgia Institute of Technology	Imaging at Georgia Institute of Technology	Imaging at Georgia Institute of Technology	Emory University
Dates of Use	May 2012 - May 2014	November 2015 - October 2017	October 2021 - August 2022	November 2022 - May 2023	April 2014 - February 2017
Study	GTP [a]	MIND Pilot [b]	MIND [c]	MIND [c]	GTP [a]

GTP=Grady Trauma Project
MIND=Mechanistic Interventions and Neuroscience of Dissociation
[a] 80 participants were recruited from GTP studies, including K23MH101380, R01MH071537, and R01MH094757
[b] 7 participants were recruited from a clinical trial for PTSD (baseline scan) at Emory University (Fani et al., 2023)
[c] 48 participants were recruited from a clinical trial for dissociation (baseline scan) at Emory University (MIND; NCT04670640, recruitment dates between 09/01/2020 and 11/01/2023; study end date is 08/31/2025). The MIND study is an ongoing multi-site trial, and the 48 participants included were those from the Atlanta/GTP site who completed pre-intervention MRI scans before 11/01/2023, which is when statistical analyses began

Figure 1. Masks of white matter pathways of interest



A. Tracts from the Juelich Histological Atlas (Burgel et al., 2006): fornix (yellow)
B. Tracts from the ICBM-DTI-81 white-matter labels atlas (Mori et al., 2005): external capsule (red), internal capsule (green blue), and corona radiata (blue)
C. Tracts from the probabilistic Johns Hopkins University White Matter Atlas (Hua et al., 2008): corpus callosum (light blue), thalamic radiation (maroon), uncinate fasciculus (green), superior longitudinal fasciculus (orange), anterior cingulum (purple), and posterior cingulum (pink)

Table 3. Mean and standard deviation of tSNR values and fractional anisotropy for all tracts, all scanners, before harmonization

	Scanner 1 (*n*=31)		Scanner 2 (*n*=7)		Scanner 3 (*n*=32)		Scanner 4 (*n*=16)		Scanner 5 (*n*=49)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Corpus Callosum	.55	.025	.60	.026	.63	.017	.64	.015	.56	.027
Anterior Cingulum	.48	.024	.53	.012	.55	.018	.55	.015	.49	.026
Posterior Cingulum	.44	.026	.52	.017	.49	.022	.48	.019	.42	.022
Fornix	.44	.024	.50	.017	.49	.021	.49	.018	.44	.026
Corona Radiata	.47	.028	.51	.028	.54	.020	.55	.016	.48	.026
Internal Capsule	.62	.025	.65	.019	.69	.017	.70	.013	.63	.025
External Capsule	.40	.022	.42	.018	.50	.020	.50	.016	.41	.024
Thalamic Radiation	.47	.020	.52	.016	.53	.016	.54	.013	.48	.022
Superior Longitudinal Fasciculus	.44	.019	.48	.015	.51	.017	.51	.015	.45	.023
Uncinate Fasciculus	.41	.021	.47	.019	.49	.018	.49	.016	.43	.022
tSNR	7.52	.45	7.45	.54	9.96	.47	9.43	.38	7.79	.69

tSNR = temporal signal-to-noise ratio

Table 4. Clinical and Demographic Characteristics (N=135)

		% (*n*)
Race		
	Asian	0.7 (1)
	Black or African American	83.7 (113)
	Hispanic	2.2 (3)
	White	13.3 (18)
Ethnicity		
	Hispanic	5.9 (8)
	Not Hispanic	94.1 (127)
Education		
	< 12th grade	8.9 (12)
	High school graduate/GED	25.9 (35)
	Some college or technical school	37.0 (50)
	Technical school graduate/associate degree	6.7 (9)
	College graduate	12.6 (17)
	Graduate or professional degree	8.9 (12)
Monthly Income		
	$0 - 249	11.9 (16)
	$250 - 499	8.1 (11)
	$500 - 999	16.3 (22)
	$1,000 - 1,999	28.1 (38)
	$2,000+	33.3 (45)
	Prefer not to say	2.2 (3)
Employment Status		
	Employed	43.7 (59)
	Disability Recipient	8.9 (12)
Psychotropic Medication Usage		15.6 (21)
	Antidepressant	12.6 (17)
	Anticonvulsant	4.4 (6)
	Stimulant	1.5 (2)
	Antihistamine	3.0 (4)
	Anxiolytic	0.7 (1)
	Benzodiazepine	1.5 (2)
	Opioid	0.7 (1)
	Antipsychotic	0.7 (1)
		Mean (SD)
MDI Total		59.99 (23.72)
	Disengagement	13.41 (5.66)
	Depersonalization	9.27 (4.93)
	Derealization	10.19 (4.97)

	Emotional Constriction/Numbing	11.05 (5.71)
	Memory Disturbance	9.74 (4.52)
	Identity Dissociation	6.33 (3.38)
mPSS[a] Total		15.18 (13.49)
	Reexperiencing	4.07 (4.12)
	Avoidance and Numbing	5.69 (5.80)
	Hyperarousal	5.42 (4.52)
PCL-5[b] Total		39.85 (13.50)
	Cluster B	8.75 (4.06)
	Cluster C	5.06 (2.18)
	Cluster D	14.67 (6.12)
	Cluster E	11.38 (4.42)
Harmonized PTSD Symptoms		30.16 (19.71)
TEI Experienced Total[a]		3.74 (2.47)
LEC-5 Experienced Total[b]		3.25 (1.79)

MDI = Multiscale Dissociation Inventory

mPSS = modified PTSD Symptom Scale

PCL-5 = PTSD Checklist for *DSM-5*

PTSD = posttraumatic stress disorder

TEI = Traumatic Events Inventory

LEC-5 = Life Events Checklist for *DSM-5*

[a] $n = 87$

[b] $n = 48$

Table 5. Correlations between harmonized PTSD symptom scores and Multiscale Dissociation Inventory Subscale Scores

		PCL-5	Disengagement	Depersonalization	Derealization	Emotion Constriction	Memory Disturbance
Disengagement	*r*	**.61 ****					
	p	**< .001**					
Depersonalization	*r*	**.60 ****	**.66 ****				
	p	**<.001**	**< .001**				
Derealization	*r*	**.64 ****	**.66 ****	**.81 ****			
	p	**< .001**	**< .001**	**< .001**			
Emotion Constriction	*r*	**.64 ****	**.60 ****	**.71 ****	**.68 ****		
	p	**< .001**	**< .001**	**< .001**	**< .001**		
Memory Disturbance	*r*	**.52 ****	**.75 ****	**.61 ****	**.66 ****	**.53 ****	
	p	**< .001**	**< .001**	**< .001**	**< .001**	**< .001**	
Identity Dissociation	*r*	**.32 ****	**.28 ***	**.45 ****	**.39 ****	**.40 ****	**.37 ****
	p	**< .001**	**.001**	**< .001**	**< .001**	**< .001**	**< .001**

* Uncorrected $p < .05$, ** Uncorrected $p < .001$

PSTD = posttraumatic stress disorder

PCL-5 = PTSD Checklist for *DSM-5*

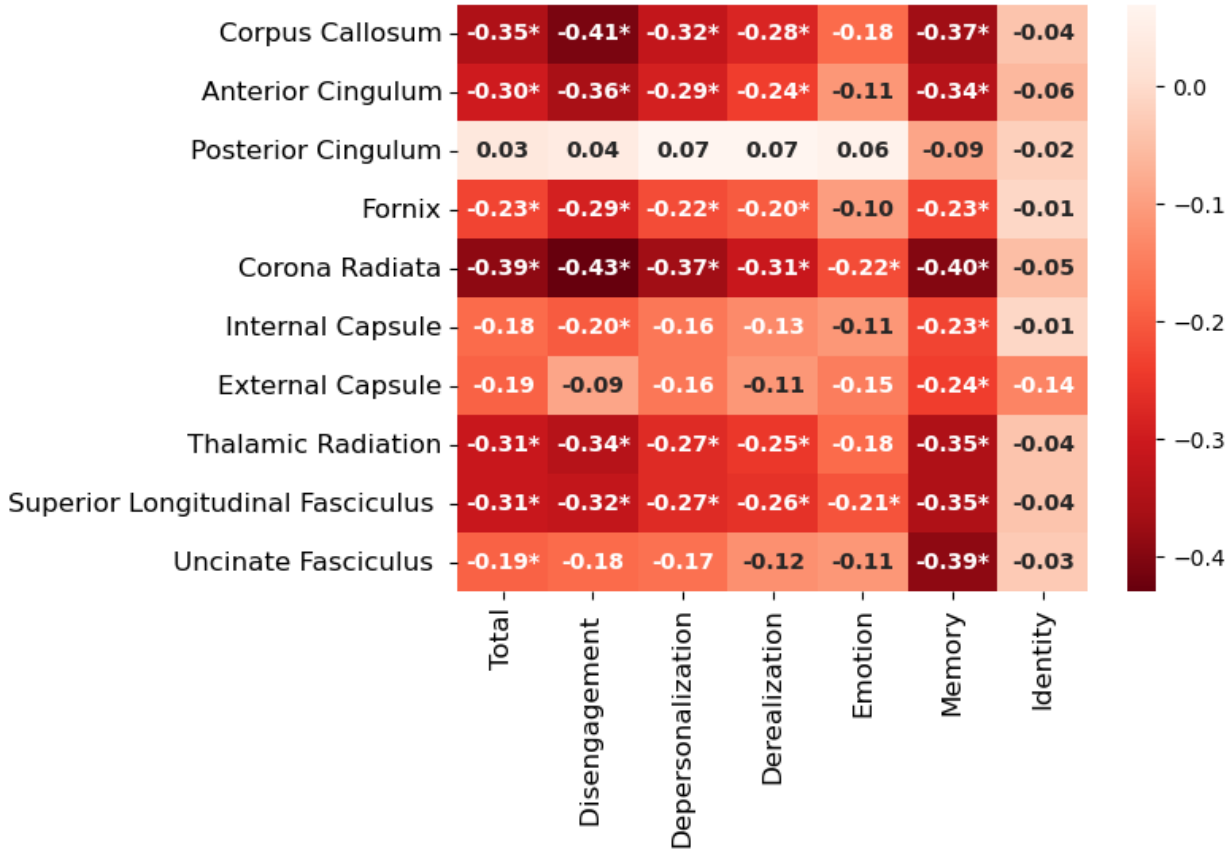
Table 6. Correlations between harmonized tract fractional anisotropy and clinical and demographic characteristics

			Traumas Experienced				
		Age	PTSD[a]	TEI Total[b]	LEC Total[c]	Education[d]	Income[d,e]
Corpus Callosum	*r*	-.16	**-.29 ****	-.19	-.04	**-.17 ***	.00
	p	.06	**<.001**	.08	.80	**.05**	.99
Anterior Cingulum	*r*	**-.20 ***	**-.25 ****	-.17	-.02	-.14	.03
	p	**.02**	**.004**	.13	.91	.11	.73
Posterior Cingulum	*r*	-.15	.03	-.05	-.03	.10	.17
	p	.08	.74	.66	.84	.23	.05
Fornix	*r*	-.10	**-.20 ***	-.13	-.05	.04	-.03
	p	.24	**.02**	.24	.76	.66	.78
Corona Radiata	*r*	**-.21 ***	**-.29 ****	-.18	-.08	**-.20 ***	.00
	p	**.02**	**<.001**	.09	.61	**.02**	.99
Internal Capsule	*r*	-.13	-.11	-.01	-.22	-.03	.10
	p	.12	.22	.92	.14	.75	.28
External Capsule	*r*	**-.42 ****	-.02	-.04	-.03	.04	.16
	p	**<.001**	.79	.72	.86	.66	.08
Thalamic Radiation	*r*	**-.29 ****	**-.21 ***	-.12	-.08	-.04	.09
	p	**<.001**	**.02**	.27	.60	.64	.34
Superior Longitudinal Fasciculus	*r*	**-.29 ****	**-.23 ***	-.10	-.06	-.06	.08
	p	**<.001**	**.01**	.34	.71	.53	.39
Uncinate Fasciculus	*r*	**-.44 ****	-.04	-.14	.06	-.02	.12
	p	**<.001**	.62	.21	.68	.83	.17

PSTD=posttraumatic stress disorder
TEI=Traumatic Events Inventory
LEC=Life Events Checklist for *DSM-5*
* uncorrected $p < .05$
** uncorrected $p < .001$

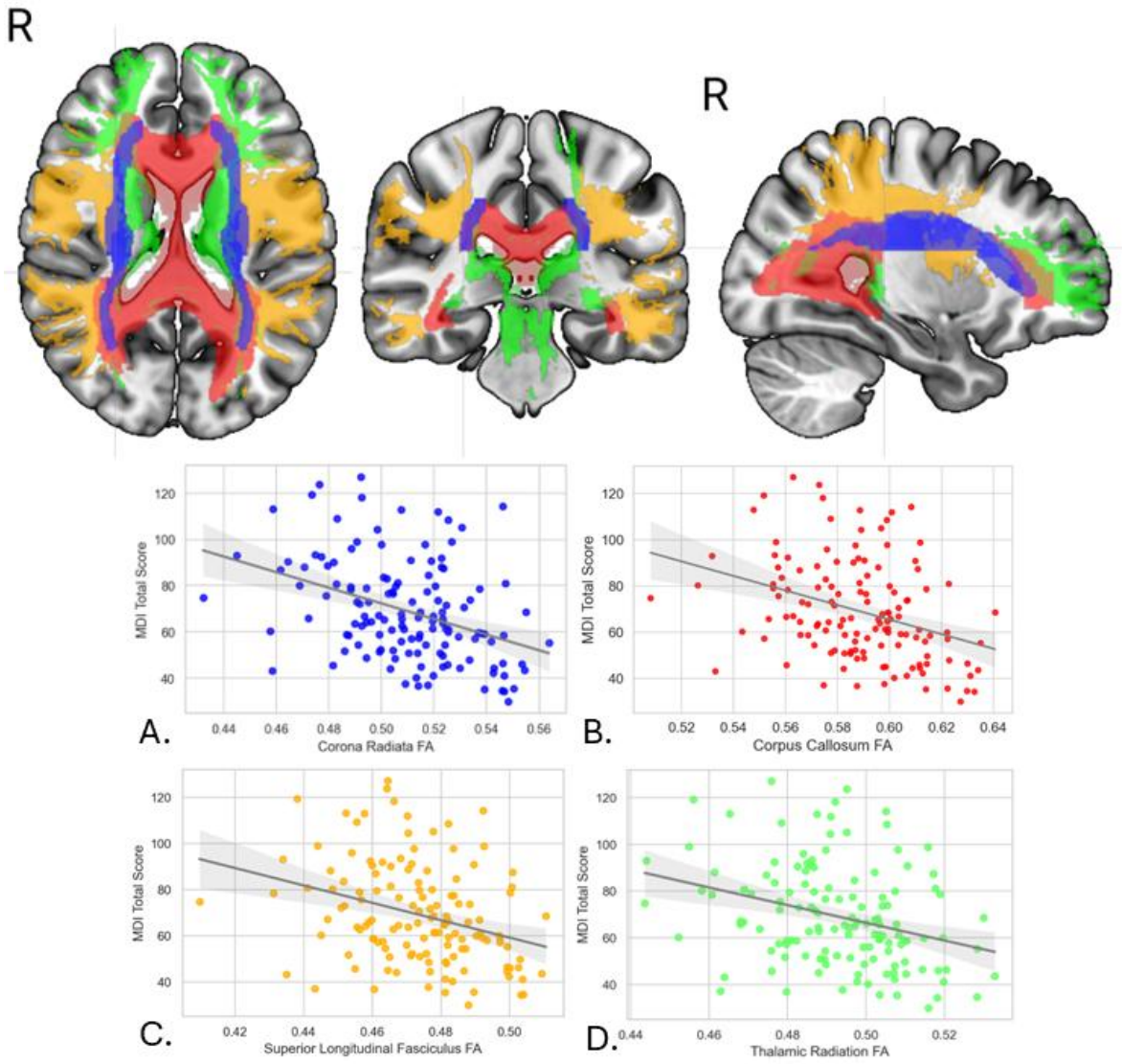
[a] harmonized PCL-5 total score
[b] $n = 87$
[c] $n = 47$
[d] Spearman's rho
[e] data missing for three participants

Figure 2. Associations among dissociation and white matter microstructure (N=135).



Partial correlations between MDI total and subscale scores and fractional anisotropy, age and temporal signal-to-noise ratio covaried. * $p_{FDR} < .05$

Figure 3. Significant associations among dissociation symptoms and tract FA.



Scatterplots include residuals of fractional anisotropy values and Multiscale Dissociation Inventory (MDI) total scores after covarying age and temporal signal-to-noise ratio. Following residualizing, these distributions were shifted back to their original scales to match the minimum value for the original distribution. Masks of the tracts are highlighted in color: corona radiata in blue, corpus callosum in red, superior longitudinal fasciculus (SLF) in orange, and thalamic radiation in green. MDI Total Score partial correlation scatter plots with fitted regression lines

are as follows: [A] corona radiata FA (r = -.39, p_{FDR} < .001), [B] corpus callosum FA (r = -.35, p_{FDR} < .001), [C] SLF FA (r = -.31, p_{FDR} = .001), [D] thalamic radiation FA (r = -.31, p_{FDR} = .001).

Table 7. Partial correlations between harmonized tract fractional anisotropy and Multiscale Dissociation Inventory scores with age and temporal signal-to-noise ratio as covariates

		Multiscale Dissociation Inventory						
		Total	Disengagement	Depersonalization	Derealization	Emotion	Memory	Identity
Corpus Callosum	*r*	**-.35 ***	**-.41 ***	**-.32 ***	**-.28 ***	-.18	**-.37 ***	-.04
	p	**<.001**	**<.001**	**<.001**	**<.001**	.04	**<.001**	.64
	p_{FDR}	**<.001**	**<.001**	**.001**	**.003**	.06	**<.001**	.71
Anterior Cingulum	*r*	**-.30 ***	**-.36 ***	**-.29 ***	**-.24 ***	-.11	**-.34 ***	-.06
	p	**<.001**	**<.001**	**<.001**	**.006**	.20	**<.001**	.48
	p_{FDR}	**.002**	**<.001**	**.003**	**.02**	.28	**<.001**	.57
Posterior Cingulum	*r*	.03	.04	.07	.07	.06	-.09	-.02
	p	.72	.67	.44	.46	.51	.31	.87
	p_{FDR}	.75	.72	.54	.55	.60	.38	.89
Fornix	*r*	**-.23 ***	**-.29 ***	**-.22 ***	**-.20 ***	-.10	**-.23 ***	-.01
	p	**.008**	**<.001**	**.01**	**.02**	.25	**.008**	.90
	p_{FDR}	**.02**	**.003**	**.03**	**.04**	.33	**.02**	.90
Corona Radiata	*r*	**-.39 ***	**-.43 ***	**-.37 ***	**-.31 ***	**-.22 ***	**-.40 ***	-.05
	p	**<.001**	**<.001**	**<.001**	**<.001**	**.01**	**<.001**	.56
	p_{FDR}	**<.001**	**<.001**	**<.001**	**.001**	**.02**	**<.001**	.64

Internal Capsule	*r*	-.18	**-.20 ***	-.16	-.13	-.11	**-.23 ***	-.01
	p	.04	**.02**	.07	.13	.23	**.01**	.90
	p_{FDR}	.06	**.04**	.10	.18	.31	**.02**	.90
External Capsule	*r*	-.19	-.09	-.16	-.11	-.15	**-.24 ***	-.14
	p	.03	.29	.06	.23	.08	**.006**	.11
	p_{FDR}	.06	.37	.09	.31	.12	**.02**	.17
Thalamic Radiation	*r*	**-.31 ***	**-.34 ***	**-.27 ***	**-.25 ***	-.18	**-.35 ***	-.04
	p	**<.001**	**<.001**	**.001**	**.004**	.04	**<.001**	.65
	p_{FDR}	**.001**	**<.001**	**.004**	**.01**	.06	**<.001**	.71
Superior Longitudinal Fasciculus	*r*	**-.31 ***	**-.32 ***	**-.27 ***	**-.26 ***	**-.21 ***	**-.35 ***	-.04
	p	**<.001**	**<.001**	**.002**	**.004**	**.02**	**<.001**	.61
	p_{FDR}	**.001**	**<.001**	**.005**	**.01**	**.03**	**<.001**	.69
Uncinate Fasciculus	*r*	**-.19 ***	-.18	-.17	-.12	-.11	**-.39 ***	-.03
	p	**.03**	.04	.05	.16	.23	**<.001**	.70
	p_{FDR}	**.05**	.06	.06	.23	.31	**.002**	.74

* FDR-corrected $p < .05$

Table 8. Partial correlations between harmonized tract fractional anisotropy and Multiscale Dissociation Inventory scores with age, temporal signal-to-noise ratio, and harmonized PCL-5 scores as covariates

		Multiscale Dissociation Inventory						
		Total	Disengagement	Depersonalization	Derealization	Emotion	Memory	Identity
Corpus Callosum	*r*	-.17	**-.29 ***	-.17	-.10	.04	-.24	.07
	p	.05	**< .001**	.06	.25	.65	.005	.45
	p_{FDR}	.20	**.03**	.25	.44	.78	.05	.64
Anterior Cingulum	*r*	-.14	-.24	-.14	-.07	.10	-.23	.03
	p	.12	.006	.10	.43	.27	.01	.88
	p_{FDR}	.33	.05	.30	.63	.46	.06	.96
Posterior Cingulum	*r*	.03	.04	.08	.08	.07	-.11	-.02
	p	.70	.66	.38	.39	.46	.21	.83
	p_{FDR}	.80	.78	.60	.59	.64	.43	.88
Fornix	*r*	-.09	-.19	-.10	-.07	.06	-.13	.06
	p	.29	.03	.25	.43	.49	.13	.47
	p_{FDR}	.48	.13	.44	.62	.66	.35	.64
Corona Radiata	*r*	-.22	**-.29 ***	-.22	-.13	-.01	**-.28 ***	.06
	p	.01	**<.001**	.01	.15	.88	**.001**	.51
	p_{FDR}	.08	**.03**	.08	.38	.91	**.03**	.69

Internal Capsule	*r*	-.14	-.16	-.11	-.08	-.04	-.20	.03
	p	.11	.07	.20	.39	.65	.02	.77
	p_{FDR}	.31	.23	.42	.60	.78	.10	.83
External Capsule	*r*	-.14	-.03	-.12	-.04	-.10	-.21	-.11
	p	.10	.76	.18	.65	.25	.02	.21
	p_{FDR}	.31	.83	.41	.78	.44	.10	.43
Thalamic Radiation	*r*	-.17	-.23	-.14	-.09	-.01	-.24	.05
	p	.06	.01	.11	.29	.95	.005	.58
	p_{FDR}	.22	.06	.31	.48	.95	.05	.74
Superior Longitudinal Fasciculus	*r*	-.16	-.19	-.12	-.10	-.03	-.24	.05
	p	.07	.03	.17	.25	.70	.006	.58
	p_{FDR}	.23	.13	.39	.44	.80	.05	.74
Uncinate Fasciculus	*r*	-.12	-.12	-.10	-.03	-.01	-.25	.01
	p	.16	.19	.25	.71	.90	.004	.88
	p_{FDR}	.39	.41	.44	.80	.91	.05	.91

* FDR-corrected $p < .05$

PCL-5 = PTSD Checklist for *DSM-5*

Table 9. Partial correlations between harmonized tract fractional anisotropy and Multiscale Dissociation Inventory scores with age and temporal signal-to-noise ratio as covariates including only participants who identified as Black

		Multiscale Dissociation Inventory						
		Total	Disengagement	Depersonalization	Derealization	Emotion	Memory	Identity
Corpus Callosum	*r*	**-.38 ***	**-.41 ***	**-.37 ***	**-.33 ***	**-.24 ***	**-.37 ***	-.13
	p	**<.001**	**<.001**	**<.001**	**<.001**	**.01**	**<.001**	.16
	p_{FDR}	**<.001**	**<.001**	**<.001**	**.002**	**.03**	**<.001**	.20
Anterior Cingulum	*r*	**-.33 ***	**-.34 ***	**-.35 ***	**-.28 ***	-.20	**-.32 ***	-.15
	p	**<.001**	**<.001**	**<.001**	**.003**	.04	**<.001**	.12
	p_{FDR}	**.001**	**.001**	**.001**	**.01**	.06	**.003**	.16
Posterior Cingulum	*r*	.02	.01	.06	.08	.04	-.04	-.06
	p	.83	.94	.55	.42	.72	.65	.55
	p_{FDR}	.84	.94	.60	.47	.74	.68	.60
Fornix	*r*	**-.26 ***	**-.30 ***	**-.27 ***	**-.22 ***	-.16	**-.24 ***	-.06
	p	**.006**	**.002**	**.004**	**.02**	.09	**.01**	.56
	p_{FDR}	**.01**	**.005**	**.01**	**.04**	.13	**.03**	.60
Corona Radiata	*r*	**-.42 ***	**-.43 ***	**-.40 ***	**-.36 ***	**-.29 ***	**-.39 ***	-.17
	p	**<.001**	**<.001**	**<.001**	**<.001**	**.002**	**<.001**	.08
	p_{FDR}	**<.001**	**<.001**	**<.001**	**<.001**	**.01**	**<.001**	.11

Internal Capsule	*r*	**-.21 ***	-.20	-.21	-.16	-.15	**-.23 ***	-.08
	p	**.03**	.04	.03	.09	.11	**.02**	.38
	p_{FDR}	**.05**	.06	.05	.13	.15	**.03**	.43
External Capsule	*r*	-.18	-.09	-.17	-.10	-.17	-.20	-.18
	p	.06	.35	.07	.28	.07	.04	.06
	p_{FDR}	.09	.40	.13	.33	.11	.06	.09
Thalamic Radiation	*r*	**-.34 ***	**-.34 ***	**-.31 ***	**-.28 ***	**-.22 ***	**-.34 ***	-.14
	p	**<.001**	**<.001**	**<.001**	**.003**	**.02**	**<.001**	.13
	p_{FDR}	**.001**	**.001**	**.003**	**.01**	**.05**	**.001**	.16
Superior Longitudinal Fasciculus	*r*	**-.34 ***	**-.35 ***	**-.29 ***	**-.29 ***	**-.24 ***	**-.33 ***	-.13
	p	**<.001**	**<.001**	**.002**	**.002**	**.01**	**<.001**	.18
	p_{FDR}	**.001**	**.001**	**.005**	**.01**	**.03**	**.002**	.21
Uncinate Fasciculus	*r*	-.21	-.16	-.20	-.15	-.13	**-.26 ***	-.15
	p	.03	.11	.03	.12	.17	**.01**	.12
	p_{FDR}	.05	.14	.06	.16	.20	**.01**	.16

* FDR-corrected $p < .05$

Table 10. Mediation analyses examining MDI score as a mediator of the relationship between PCL-5 score and harmonized tract FA

	PCL-5 on MDI Effect		MDI on FA Effect		Total Effect		Direct Effect		Standardized Indirect Effect		
	a	SE	b	SE	c	SE	c'	SE	a x b	CI lower	CI upper
Corpus Callosum	**.7778****	**.0703**	**-.0003***	**.0001**	**-.0004****	**.0001**	-.0002	.0001	**-.1596**†	**-.3028**	**-.0202**
Anterior Cingulum	**.7778****	**.0703**	-.0002	.0001	**-.0003****	**.0001**	-.0002	.0001	-.1224	-.2844	.0296
Posterior Cingulum	**.7778****	**.0703**	.0001	.0001	.0000	.0001	-.0001	.0001	.0459	-.1097	.2010
Fornix	**.7778****	**.0703**	-.0001	.0001	**-.0003***	**.0001**	-.0002	.0001	-.0925	-.2518	.0589
Corona Radiata	**.7778****	**.0703**	**-.0003***	**.0001**	**-.0004****	**.0001**	-.0002	.0001	**-.1907**†	**-.3493**	**-.0369**
Internal Capsule	**.7778****	**.0703**	-.0002	.0001	-.0001	.0001	.0000	.0001	-.1113	-.2911	.0611
External Capsule	**.7778****	**.0703**	-.0002	.0001	-.0001	.0001	.0000	.0001	-.1214	-.2866	.0319
Thalamic Radiation	**.7778****	**.0703**	-.0002	.0001	**-.0003****	**.0001**	-.0001	.0001	**-.1453**†	**-.3023**	**-.0023**
Superior Longitudinal Fasciculus	**.7778****	**.0703**	-.0002	.0001	**-.0003****	**.0001**	-.0002	.0001	-.1318	-.2772	.0065
Uncinate Fasciculus	**.7778****	**.0703**	-.0001	.0001	-.0001	.0001	.0000	.0001	-.1098	-.2561	.0186

* Uncorrected $p < .05$, ** Uncorrected $p < .005$, † Significant Indirect Effect (bootstrapped confidence interval did not contain zero)

Table 11. Mediation analyses examining PCL-5 score as a mediator of the relationship between MDI score and harmonized tract FA

	MDI on PCL-5 Effect		PCL-5 on FA Effect		Total Effect		Direct Effect		Standardized Indirect Effect		
	a	SE	b	SE	c	SE	c'	SE	a x b	CI lower	CI upper
Corpus Callosum	**.6184****	**.0559**	-.0002	.0001	**-.0004****	**.0001**	**-.0003***	**.0001**	-.1307	-.2721	.0045
Anterior Cingulum	**.6184****	**.0559**	-.0002	.0001	**-.0003****	**.0001**	-.0002	.0001	-.1306	-.2838	.0120
Posterior Cingulum	**.6184****	**.0559**	-.0001	.0001	.0000	.0001	.0001	.0001	-.0358	-.2042	.1221
Fornix	**.6184****	**.0559**	-.0002	.0001	**-.0003***	**.0001**	-.0001	.0001	-.1050	-.2597	.0522
Corona Radiata	**.6184****	**.0559**	-.0002	.0001	**-.0004****	**.0001**	**-.0003***	**.0001**	-.1159	-.2702	.0289
Internal Capsule	**.6184****	**.0559**	.0000	.0001	**-.0002***	**.0001**	-.0002	.0001	.0207	-.2138	.1555
External Capsule	**.6184****	**.0559**	.0000	.0001	**-.0002***	**.0001**	-.0002	.0001	.0050	-.1545	.1704
Thalamic Radiation	**.6184****	**.0559**	-.0001	.0001	**-.0003****	**.0001**	-.0002	.0001	-.0997	-.2491	.0429
Superior Longitudinal Fasciculus	**.6184****	**.0559**	-.0002	.0001	**-.0003****	**.0001**	-.0002	.0001	-.1234	-.2743	.0140
Uncinate Fasciculus	**.6184****	**.0559**	.0000	.0001	**-.0002***	**.0001**	-.0001	.0001	-.0214	-.1528	.1136

* Uncorrected $p < .05$, ** Uncorrected $p < .005$, † Significant Indirect Effect (bootstrapped confidence interval did not contain zero)

Table 12. Partial correlations between Multiscale Dissociation Inventory scores and Letter N-Back Task Performance and CAPS-5 item D1 severity with age as a covariate

		Multiscale Dissociation Inventory						
		Total	Disengagement	Depersonalization	Derealization	Emotion	Memory	Identity
LNB True Positives[a]	*r*	.07	.12	.15	.05	-.10	.04	.11
	p	.45	.19	.12	.58	.29	.70	.24
LNB False Positives[a]	*r*	-.15	**-.25 ****	-.14	-.09	-.06	-.12	-.03
	p	.11	**.01**	.14	.32	.53	.20	.78
LNB Median Response Time[a]	*r*	-.02	-.03	-.05	-.05	.10	-.13	.05
	p	.81	.77	.56	.58	.30	.17	.60
LNB-0 True Positives[a]	*r*	.05	.08	.13	.02	-.06	.05	.06
	p	.60	.41	.17	.83	.55	.62	.56
LNB-0 False Positives[a]	*r*	-.15	**-.24 ***	-.12	-.11	-.08	-.08	-.02
	p	.12	**.01**	.21	.24	.39	.38	.81
LNB-0 Median Response Time[a]	*r*	-.01	-.01	-.04	-.05	.09	-.12	.07
	p	.91	.92	.71	.62	.35	.19	.48
LNB-1 True Positives[a]	*r*	.12	.15	.11	.01	-.002	.16	.18
	p	.19	.11	.24	.91	.98	.08	.05
LNB-1 False Positives[a]	*r*	-.08	-.12	-.06	-.13	-.10	.07	-.14
	p	.37	.21	.52	.17	.28	.48	.15

LNB-1 Median Response Time[a]	*r*	-.07	-.06	-.13	-.10	.07	-.14	.04
	p	.47	.50	.17	.28	.48	.15	.65
LNB-2 True Positives[a]	*r*	.01	.06	.09	.05	-.11	-.05	.03
	p	.90	.55	.32	.56	.25	.60	.72
LNB-2 False Positives[a]	*r*	-.06	-.12	-.09	-.02	.02	-.07	-.03
	p	.51	.22	.35	.81	.81	.49	.74
LNB-2 Median Response Time[a]	*r*	-.02	-.03	-.01	-.004	.02	-.09	.02
	p	.85	.76	.94	.96	.81	.36	.87
CAPS-5 D1[b]	*r*	**.25 ** **	**.25 ***	**.25 ***	**.24 ***	.13	.10	**.25 ***
	p	**.01**	**.01**	**.01**	**.01**	.21	.30	**.01**

* Uncorrected $p < .05$, ** Uncorrected $p < .01$, *** Uncorrected $p < .005$

LNB = letter n-back task

LNB-0 = 0-back trial for letter n-back task

LNB-1 = 1-back trial for letter n-back task

LNB-2 = 2-back trial for letter n-back task

CAPS-5 = PTSD Checklist for *DSM-5*

[a] = 116 participants

[b] = 105 participants