Psychotic-Like Symptoms and the Temporal Lobe in Trauma-Related Disorders: Diagnosis, Treatment, and Assessment of Potential Malingering

Francesca L. Schiavone[1], Margaret C. McKinnon[2,3,4], and Ruth A. Lanius[5,6]

Abstract

Objective: To overview the phenomenology, etiology, assessment, and treatment of psychotic-like symptoms in trauma-related disorders focusing on the proposed role of temporal lobe dysfunction.

Method: We describe the literature pertaining to (i) psychotic-like symptoms and temporal lobe dysfunction in trauma-related disorders and (ii) psychological testing profiles in trauma-related disorders. We define trauma-related disorders as borderline personality disorder, post-traumatic stress disorder, and the dissociative disorders. Our search terms were dissociative disorders, temporal lobe, trauma, post-traumatic stress disorder, borderline personality disorder, psychosis, and malingering.

Results: Trauma-related psychotic-like symptoms are common and can differ in phenomenology from primary psychotic symptoms. Hallucinations consist of auditory *and* nonauditory content that may or may not relate to traumatic content. Child voices are highly suggestive of complex dissociative disorders. Critically, not only do these symptoms resemble those seen in temporal lobe epilepsy, but the temporal lobe is implicated in trauma-related disorders, thus providing a plausible neurobiological explanation. Despite such evidence, these symptoms are frequently considered atypical and misdiagnosed. Indeed, common structured psychological assessment tools categorize these symptoms as possible indicators of invalid testing profiles.

Conclusion: Psychotic-like symptoms are common in trauma-related disorders, may be related to temporal lobe dysfunction, and are frequently misinterpreted. This may lead to ineffective treatment and inappropriate determinations of malingering in the forensic system.

Introduction

Dissociation is defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) as a "discontinuity in the normal integration of consciousness, memory, identity, emotions, perception, body representation, motor control, and behavior".[1] Dissociative symptoms include trance-like states, out-of-body experiences (depersonalization), and a sense of unreality (derealization). Complex dissociative disorders fall at the more severe end of this spectrum and include dissociative identity disorder (DID), which is characterized by marked identity

[1]Department of Psychiatry, University of Toronto, Toronto, Ontario, Canada
[2]Mood Disorders Program, St. Joseph's Healthcare, Hamilton, Ontario, Canada
[3]Department of Psychiatry and Behavioural Neuroscience, McMaster University, Hamilton, Ontario, Canada
[4]Homewood Research Institute, Guelph, Ontario, Canada
[5]Department of Psychiatry, University of Western Ontario, London, Ontario, Canada
[6]Department of Neuroscience, University of Western Ontario, London, Ontario, Canada

Corresponding Author:
Ruth A. Lanius, 339 Windermere Road, London, Ontario N6A2A2, Canada.
Email: Ruth.Lanius@LHSC.ON.CA

alteration (e.g., experiences of multiple personalities and dissociated self-states) and amnesia, and DID-like presentations of other specified dissociative disorder (OSDD) (previously known as DDNOS). Critically, these disorders are typically associated with a history of significant psychological trauma (post-traumatic stress disorder (PTSD) is a frequent comorbidity) and/or disruptions in early attachment relationships, which lead to disorganized attachment and dissociation.[1–3]

A recent meta-analysis by Lyssenko et al.[4] examined the severity of dissociative symptoms, as measured by the Dissociative Experiences Scale (DES), across a range of psychiatric diagnoses. Dissociative disorders were associated with the highest levels of dissociation, followed by PTSD, borderline personality disorder (BPD), and conversion disorder. These findings support a strong transdiagnostic link between trauma-related disorders and dissociation, a connection explored previously, for example, in the linkage between conversion disorder and the dissociative disorders.[5,6] Compared to healthy controls, however, mean scores on the DES were elevated across almost all diagnoses, including substance-related disorders, schizophrenia, and mood and anxiety disorders. Clearly, dissociative phenomena are transdiagnostic and limited not only to the dissociative disorders or indeed even to trauma-related disorders.

Patients with high levels of dissociation typically present with a broad range of psychopathology that includes explicitly dissociative phenomena such as depersonalization, derealization, dissociative amnesia, and identity alteration. These dissociative symptoms, however, typically occur in the context of complex, polysymptomatic presentations characterized by high morbidity, and comorbidity. For example, in one sample of 280 patients with DID or DDNOS (based on DSM-IV criteria), 89% also had PTSD, 83% had a mood disorder, 54% had a cluster B personality disorder, 30% had an eating disorder, 22% had a substance use disorder, and 22% had a somatoform disorder.[7] Similarly, in an Australian sample, 48% of patients with a dissociative disorder had been hospitalized, of which 68% had been hospitalized more than five times.[8] In the same sample, 68% reported self-harm, and 69% reported suicide attempts, with 26% endorsing more than five past attempts.[8] Psychotic-like symptoms, which are not typically considered dissociative, are also observed frequently and may present in ways that are considered unusual or inconsistent with primary psychosis.[1,9–17]

Notably, these types of symptoms are also well described in the temporal lobe epilepsy (TLE) literature and are thought to emerge as a consequence of temporal lobe dysfunction.[18] Located bilaterally and inferior to the lateral fissure, the temporal lobe is one of the major lobes of the cerebral cortex. The medial temporal lobe is comprised of structures including the hippocampus,

amygdala, uncus, dentate gyrus, and the parahippocampal gyrus that are involved in, or closely connected to, the limbic system, which is central to emotion, motivation, and memory. The lateral temporal lobe contains the primary auditory cortex as well as areas involved in visual processing, language processing, and complex object recognition.[18] The lateral temporal lobe also includes the temporoparietal junction (TPJ), which is believed crucial to self-processing, self-location, and perceived spatial unity of self and body, as well as multisensory integration of body-related information. Because of its role in "own-body" processing, the TPJ has been linked closely to dissociative symptoms such as depersonalization (e.g., out-of-body experiences) and identity disturbance.[19–21]

There is an urgent need to better understanding the occurrence of psychotic-like and dissociative symptoms in psychiatric disorders. In particular, the framing of these symptoms within a neurobiological context may facilitate not only improved assessment but also the treatment of patients across a range of psychiatric disorders presenting with dissociative and psychotic-like symptoms.

Aims of the Study

We will review the extant literature describing TLE-like symptoms (primarily psychotic-like and dissociative symptoms) observed across trauma-related disorders (PTSD, BPD, and the dissociative disorders) and the evidence for temporal lobe dysfunction in these patients. We then identify the clinical implications of this literature and discuss critical areas of future research.

Materials and Methods

The aim of this clinical overview is to discuss the phenomenology, etiology, assessment, and treatment of psychotic-like symptoms in trauma-related disorders (BPD, PTSD, and dissociative disorders) with attention to the proposed role of temporal lobe dysfunction. We have reviewed the literature via a PubMed search of articles published between 1980 and March 2018 using the following search terms: dissociative disorders, temporal lobe, trauma, post-traumatic stress disorder, borderline personality disorder, psychosis, and malingering.

Results

Dissociative Symptoms in TLE

A large body of literature has sought to examine the association between TLE and a variety of dissociative symptoms, motivated in part by Feindel and Penfield's[22] initial observation of a "dreamy state" reported by their patients with TLE and, critically, reproducible with

electrical stimulation of the temporal lobe. More recent work points toward a frequent association between TLE and dissociation. For example, in one case series, 33% of patients with TLE reported some form of interictal dissociative episodes.[23] In line with this observation, a series of large studies of patients with epilepsy found elevated scores on the DES, which is a widely used measure of pathological dissociation.[24–27]

Similarly, in a case series of patients with complex partial seizures, dissociative symptoms were reported by a significant portion of patients and included body part dissociation (19%), perception of surroundings as bizarre (11%), depersonalization (19%), and derealization (16%).[28] These observations were mirrored in a sample of 20 patients with TLE who completed the Cambridge Depersonalization Scale: 80% endorsed symptoms of depersonalization.[29] In a related review, the majority of patients with depersonalization that stemmed from an organic cause (including epilepsy, traumatic brain injury, and migraine) also showed evidence of temporal lobe dysfunction.[30] Taken together, these findings suggest a clear association between organic temporal lobe dysfunction and dissociative symptoms that are markedly similar to those seen in dissociative disorders.

Perhaps most strikingly, there have been numerous case series and reports connecting organic temporal lobe pathology (particularly TLE) to identity alteration identical to that seen in DID.[31–33] It is notable that these case series include both experiences of multiple personalities and experiences of supernatural possession, both of which are known forms of identity alteration in DID.[1] For example, in one case series of 40 patients with electroencephalogram (EEG)-verified TLE, 13 patients reported experiences of multiple personalities and/or possession, including several cases of syndromal multiple personality disorder.[23] Similarly, in Mesulam's[34] case series of 12 patients with TLE, 7 met criteria for multiple personality disorder (now called DID) and 5 reported experiences of supernatural possession. Critically, there is also mounting evidence that temporal lobe dysfunction other than TLE can produce DID-like symptoms. In one case study, a patient with traumatic brain injury and with evidence of biparietal and left temporal hypoperfusion on single photon emission computed tomography (SPECT) reported an episode of "multiple personality."[35] This literature suggests that temporal lobe dysfunction is not only associated with dissociative symptoms, in general, but that it may also underlie the complex dissociative symptoms seen in disorders like DID.

Psychotic-Like Symptoms in Trauma-Related Disorders

TLE is associated with psychiatric presentations that resemble closely both dissociative disorders and psychotic disorders. It is notable that the psychiatric disorders with the most prominent dissociative and psychotic-like symptoms, including the complex dissociative disorders, BPD, and the dissociative subtype of PTSD, have been linked not only to psychological trauma but also to alterations in temporal lobe structure and function.[14,36–40] The temporal lobe theory of dissociation provides a unifying account of these findings, pointing toward the transdiagnostic neurobiological underpinnings of these symptoms, with key implications for assessment and treatment.

Psychological Trauma. Strikingly, even in the absence of a diagnosable psychiatric disorder, psychological trauma has been associated with psychotic symptoms.[41–44] For example, in one study, individuals with auditory verbal hallucinations (but without any DSM diagnosis) reported experiencing significantly more sexual and emotional abuse than healthy controls.[41] In other samples of trauma exposed but healthy individuals, associations have been reported between experiences of bullying and of unwanted sexual contact and subthreshold psychotic symptoms (e.g., brief duration symptoms or symptoms not causing distress or impairment).[42–44] Although the relation between the nature of the trauma itself and the onset of specific psychotic symptoms remains unclear, traumatic events associated with intent to harm (i.e., perpetrated deliberately by another person) are more strongly associated with psychotic symptoms than nonintentional traumatic events (e.g., involvement in a life-threatening accident).[42,45] A number of smaller studies have associated sexual abuse specifically with hallucinations and neglect/attachment-disrupting events (e.g., separations from caregivers) specifically with paranoia.[42,46,47]

In non-Western cultures (and some Western subcultures), dissociative and/or psychotic-like symptoms may be expressed as experiences of possession (identity alteration characterized by feeling taken over by an outside force, e.g., a spirit or demon, as opposed to internal self-states) and/or paranormal phenomena (e.g., contact with ghosts or spirits, telekinesis, telepathy, or precognitive dreams). These experiences typically occur in cultures where nonpathological possession is more normative, and they can be considered culturally specific representations of known dissociative syndromes. As such, pathological possession has been included in the diagnostic criteria for DID alongside the more Western experience of internal self-states.[6,48] These symptoms, however, have been associated with trauma in general as well as specifically with DID. In one sample of women from the general population in Turkey, experiences of childhood or adulthood trauma were associated with possession and childhood abuse or neglect with paranormal phenomena.[49]

Multiple studies have also found associations between traumatic life events and the development of primary

psychotic disorders with odds ratios ranging from 2.8 to 7.3 reported in traumatized people compared to nontraumatized people.[47,50] Trauma has also been associated with progression to psychotic disorders in clinically high-risk populations (e.g., individuals with prodromal symptoms or a family history of psychotic disorder), with the presence of psychotic features in affective illnesses (e.g., bipolar disorder and major depressive disorder), and with the intensity, persistence, and severity of psychotic symptoms in patients with schizophrenia.[42,51–53] Thus, rather than being merely a specific etiological factor for the development of primary psychotic disorders, there is a transdiagnostic association between trauma and the presence and severity of psychotic symptoms (particularly abnormal sensory experiences) across a broad range of disorders and even in the absence of any diagnosable psychiatric illness.

Notably, in both clinical and nonclinical samples, dissociation has been identified as a mediator between trauma and psychotic experiences. Dissociation, as measured by the DES, has been associated both with psychotic experiences, in general, and with auditory hallucinations, particularly symptoms of depersonalization.[47,54,55] Several cognitive pathways for this relationship have been proposed, including the proposal that dissociation may increase distance from internal experiences, rendering them more likely to be perceived as external/hallucinatory.[55] Moskowitz et al.[56] further suggest that in the context of trauma, not just hallucinations, but other psychotic symptoms, that is, disorganized speech, disorganized behavior, and possibly delusions, may be conceptualized as dissociative phenomena stemming from decontextualized traumatic content or intrusions of dissociated self-states.

Post-Traumatic Stress Disorder. Even in the absence of a primary psychotic disorder, patients with PTSD frequently report psychotic-like symptoms (e.g., in one sample of 568 patients with a lifetime diagnosis of PTSD, about 19% of patients with a history of PTSD reported hallucinations) and such endorsement is associated with greater overall PTSD severity.[57,58] Indeed, "auditory pseudohallucinations" are discussed in the DSM-5 as an associated feature supporting the diagnosis of PTSD, with some trauma experts constructing these symptoms as frankly psychotic and calling for the addition of a "with psychotic features" specifier to the diagnostic criteria.[1,58] A recent review of the evidence (mainly in a military population) found some evidence for a "PTSD with secondary psychotic features" construct, with phenomenological and biological differences from both PTSD and schizophrenia.[59]

Across a series of studies, patients with PTSD, typically with complex trauma including war, childhood trauma, and refugee trauma, report a broad range of psychotic symptoms, including delusions, visual and auditory hallucinations, as well as more unusual sensory modalities, including olfactory and tactile hallucinations.[10–12] In their clinical assessments of patients, particularly those involving women with childhood trauma histories, Frewen and Lanius[9] note that pseudohallucinations are common and are typically olfactory and tactile (e.g., sensations of bugs crawling on the skin). Some, but not all, of these patients report psychotic content that is related to the trauma, and it is experienced differently from traumatic reexperiencing.[11,60,61] Moreover, compared to patients with psychotic disorders, these experiences tend to be briefer and include sensory modalities other than, or in addition to, auditory. Notably, in one study, the majority of traumatized participants reported a single voice coming from inside their head.[10,11]

In key recognition of the importance of dissociative symptoms in the psychopathology of PTSD, the DSM-5 has recently included a dissociative subtype. The dissociative subtype is characterized by primarily depersonalization and/or derealization responses to trauma-related cues, which contrast sharply with the hyperarousal and sympathetic nervous system activation observed in individuals with PTSD without the subtype.[1] Critically, psychotic-like experiences may be linked in particular to the dissociative subtype of PTSD, where psychotic symptoms have been associated with higher scores on the DES in multiple studies.[11,47,54,55,60] Notably, trauma is also associated with measures of somatoform dissociation, which encompasses symptoms that could be seen as psychotic-like or TLE-like (alterations of taste, smell, and visual perception, and seizure-like episodes).[62]

Borderline Personality Disorder. BPD is associated with traumatic life events (particularly childhood abuse) with some authors proposing that childhood trauma serves as part of a multifactorial etiology for the disorder.[36,63] Among individuals with BPD, estimates of the prevalence of childhood maltreatment typically range from 76% to 90% for childhood neglect and from 80% to 81% for childhood abuse (physical, sexual, or emotional), which is higher than the prevalence in patients with other personality disorders, although one recent review identified some heterogeneity in these rates and suggested that prevalence may range from 30% to 90% for any kind of childhood trauma.[36,63–65]

In a pattern similar to that observed in studies of patients with PTSD, BPD is associated with both dissociative and psychotic symptoms. Indeed, severe dissociative symptoms are included in the diagnostic criteria, and the prevalence of comorbid dissociative disorders in BPD has been estimated between 59% and 76%, with 10% to 27% meeting criteria for DID.[1,66,67] The most common psychotic symptoms observed in BPD

are auditory verbal hallucinations, which occur in 20% to 40% of these patients.[13,42] These hallucinations are long-standing, generally identified as negative and distressing, come from inside the head, and are sometimes (but not always) associated with traumatic memories (e.g., hearing the voice of a past abuser).[68–70] Despite their common conceptualization as "pseudohallucinations" that are clearly distinct from primary psychosis in phenomenology, when compared to the auditory hallucinations experienced by patients with schizophrenia or schizoaffective disorder, no significant difference has been found in duration, degree of distress, or location (inside vs. outside the head) of BPD-associated hallucinations, and both groups at times report content related to past traumatic experiences.[69,70] Moreover, much like in PTSD, unusual sensory modalities are reported frequently. For example, in one sample of patients with BPD, 30% reported delusions, 20% reported visual hallucinations, 13% reported tactile hallucinations, and 10% reported olfactory hallucinations.[13]

Dissociative Depression. The key literature points to a critical link between childhood trauma and the onset of increased levels of dissociation in patients with major depression.[71] Indeed, the concept of "dissociative depression" has been proposed recently in an attempt to better classify a subset of patients with major depression who also exhibit concurrent dissociative symptoms. In one sample of Turkish women with no differences in comorbidity profile from individuals with nondissociative depression (i.e., no increase in comorbid BPD, PTSD, or dissociative disorder), dissociative depression was associated with a positive trauma history, PTSD-like cognitive distortions, frequent mood swings, and impulsivity. These patients also reported psychotic-like symptoms similar to those seen in other trauma-related disorders such as hallucinations, amnesias, experiences of possession, and identity alteration symptoms.[72]

Depersonalization Disorder. Depersonalization disorder is a dissociative disorder characterized by chronic feelings of detachment from or unreality of the body or self, a common experience in TLE. Depersonalization disorder can also be associated with childhood trauma.[73] Although complex partial-seizure-like symptoms other than depersonalization/derealization themselves have yet to be explored fully in patients with diagnosed depersonalization disorder, nevertheless, in a sample of patients with BPD or PTSD, depersonalization/derealization symptoms were strongly associated with the presence of pseudohallucinations independent of diagnosis, and scores on the depersonalization subscale of the DES were the most strongly associated with psychotic symptoms.[55,74]

DID and OSDD. DID and DID-like presentations of OSDD have been related to severe and prolonged early life trauma.[14] These disorders are strongly associated with psychotic-like symptoms, including voices and, in a pattern similar to that observed in PTSD and BPD, hallucinations involving multiple sensory modalities, particularly unusual ones such as tactile, gustatory, or olfactory hallucinations.[1,15] Some of these symptoms can be difficult to distinguish from schizophrenia. For example, patients with DID frequently report voices that are both internally and externally perceived, as well as Schneiderian first-rank symptoms (most commonly voices conversing with each other or commenting on the person's actions, made thoughts, and made actions).[15–17]

Paranormal phenomena and experiences of possession are sometimes seen in dissociative disorders, particularly in cultures where nonpathological possession is culturally normative, and can be interpreted as psychotic. Epilepsy, particularly TLE, has also been associated with religious and mystic experiences.[75] The DSM-5 now includes possession as part of the diagnostic criteria for DID, in order to increase cross-cultural applicability of the diagnosis.[1,6] In one sample of women in the general population in Turkey, 33% of those with a dissociative disorder reported at least one type of paranormal experience compared to 17.3% of those without and 10.4% of those with a dissociative disorder reported experiences of possession compared to 0.2% of those without.[49] In another study, compared to unmatched samples of psychiatric outpatients and members of the general population, a sample of patients with DID reported more trance, sleepwalking, possession, and paranormal experiences.[76]

The recent literature has attempted to identify those symptoms that may be particularly useful in differentiating psychotic disorders from complex dissociative disorders. Although Schneiderian first-rank symptoms were originally thought to be pathognomonic for a primary psychotic disorder, in one study, patients with DID endorsed an average of 6.4 Schneiderian first-rank symptoms, far more than the 1.3 endorsed by patients with schizophrenia.[17] In another sample of 30 patients with multiple personality disorder by DSM-III criteria, all patients endorsed at least one first-rank symptom at some point in the course of treatment, with the average number endorsed being 3.6.[16] There are, however, some key features of psychotic symptoms (specifically auditory hallucinations) that distinguish complex dissociative disorders. For example, Dorahy et al.[15] found that compared to patients with schizophrenia, patients with DID were more likely to hear multiple voices, to hear child voices, and to have started hearing voices before the age of 18 years. Patients with DID were also more likely to experience visual, olfactory, and tactile hallucinations than were patients with schizophrenia.[15]

Temporal Lobe Dysfunction in Trauma-Related Disorders

Psychological Trauma. Recent data support a strong association between childhood psychological trauma (either in the absence of overt psychopathology or with mixed psychiatric diagnoses) and structural and functional alterations of the temporal lobe. In studies of children and adolescents with low psychopathology, a history of abuse is associated with decreased temporal cortical thickness and reduced right temporal volume.[77,78] In line with these findings, magnetic resonance imaging (MRI) studies of psychiatrically ill samples of traumatized children (with a broad range of diagnoses including PTSD) reveal increased gray matter volumes in the superior temporal gyrus.[79,80] On qEEG, children who have been abused show differences in hemispheric coherence and asymmetry, with the temporal region being among those regions most prominently affected.[81,82] Functionally, alterations in resting state activity in the superior temporal gyrus have been reported in adults without psychiatric illness exposed to early life trauma.[83] Finally, exposure to trauma-related cues may provoke temporal lobe activation in traumatized populations; a study of youth with a history of emotional neglect revealed increased activity of the medial temporal lobe on exposure to threatening cues.[84]

Post-Traumatic Stress Disorder. Related studies reveal temporal lobe abnormalities among individuals with a formal diagnosis of PTSD. For example, in one MRI study, compared to healthy controls, patients with PTSD (not separated by subtype) showed decreased gray matter volume in the right temporal lobe, including the middle and inferior gyri and the fusiform gyrus, which together are involved in perception and memory. Compared to a sample of psychiatric patients with obsessive compulsive disorder (OCD), this sample also had decreased right temporal gray matter volume.[85] Other MRI studies have corroborated the finding of decreased right temporal lobe volume and neuron density across a variety of types of index traumas[86–88] and, critically, one has linked volume loss in the right inferior temporal gyrus specifically to the dissociative subtype of PTSD.[89] In keeping with these observations, another study found an association between increased flashbacks and volume loss in the middle temporal gyrus in patients with PTSD.[90] These findings suggest a highly specific role of the temporal lobe in abnormal processing of somatosensory information (e.g., self-other differentiation errors in dissociative symptoms and decontextualized sensory material in flashbacks). Similarly, one study of sexually abused adolescents, overall those with PTSD had smaller hippocampal and amygdala volume bilaterally, and within that group, a smaller right amygdala was associated with passive influence phenomena, one of the psychotic-like symptoms seen in trauma-related disorders, further linking the temporal lobe to these symptoms.[91]

Notably, symptom provocation studies have consistently revealed an association between reexperiencing symptoms and decreased temporal lobe activation.[92,93] In a functional MRI (fMRI) study of adults with PTSD who dissociated when exposed to trauma-related cues, however, there was an increased activation in the superior and middle temporal gyri and altered right temporal connectivity.[37,38] Another study of PTSD patients' response to fearful faces also demonstrated an association between dissociative symptoms and increased temporal lobe activation.[94] Finally, in a sample of patients with lower overall dissociation proneness and primarily single-incident trauma (motor vehicle accident), dissociation in response to traumatic scripts nevertheless remained positively correlated with right temporal activation and negatively correlated with left temporal activation.[95] On balance, these data support strongly the assertion that both the dissociative subtype of PTSD and state dissociation in PTSD are associated specifically with altered temporal lobe activity in response to trauma-related cues.

These alterations in temporal lobe structure and function have been observed within the context of an emerging body of the literature that highlights altered functional connectivity in PTSD, which, importantly, is present even in the absence of overt trauma-related stimuli/reexperiencing. For example, there is a strong evidence of altered temporal connectivity and activity (particularly the right temporal lobe) on resting state fMRI across a variety of index traumas, despite the clear absence of provocation stimuli.[96–100] In the dissociative subtype specifically, increased functional connectivity has been observed between the amygdala and areas involved in consciousness, awareness, and proprioception, as well as those thought to be involved in depersonalization and derealization symptoms (the superior parietal lobe, dorsal posterior cingulate, and precuneus).[101]

Similarly, patients with and without the dissociative subtype demonstrate increased connectivity at rest between the periaqueductal gray matter in the midbrain, which is involved in defensive responses, and the fusiform gyrus, which is involved in scanning the environment, and is also connected with the amygdala.[20] There is also an evidence of increased resting state connectivity between the superior colliculus, which is involved in threat detection, and the right TPJ, which is implicated in depersonalization and derealization.[21] These patterns of connectivity suggest changes in subconscious threat processing that are evident even in the absence of overt threat or trauma-related stimuli and that together may affect day-to-day engagement with the external environment.

Borderline Personality Disorder. Structural imaging studies of male and female patients with BPD reveal strikingly similar patterns of decreased gray matter volume and density in the temporal lobes, with one study further illustrating a specific decrease in right temporal lobe volume among individuals with BPD and a history of abuse.[102–104] Consistent findings of decreased amygdala and hippocampal volume have also emerged in BPD.[105–107] Functional imaging of a sample of patients with BPD and histories of childhood abuse and severe dissociative symptoms similarly demonstrated right-sided temporal hypometabolism.[108] Finally, on neuropsychological testing, patients with BPD have show profiles suggestive of right temporal lobe dysfunction.[109]

Depression With Dissociative Features. Further research is necessary to identify specific neurobiological correlates of this depressive phenotype. However, one SPECT study on patients with DID with a 66.7% prevalence of comorbid depression (i.e., dissociative depression) noted left lateral temporal hyperperfusion in the sample as a whole.[110]

Depersonalization Disorder. In a pattern similar to that observed in other trauma-related disorders, on structural imaging, depersonalization disorder is associated with decreased right temporal cortical thickness and with right temporal hypometabolism on functional imaging.[111,112] Recent work has also demonstrated altered white matter structural connectivity between the right middle temporal gyrus, which may be involved in sensory integration, and the right supramarginal gyrus, which is a part of the somatosensory association cortex, as well as decreased structural connectivity between the left temporal pole, which is thought to be an association area, and the left superior temporal gyrus, which is involved in auditory processing.[113] Depersonalization has also been associated with temporal slowing on EEG.[114]

DID and OSDD. Studies of patients with DID and comorbid PTSD reveal decreased temporal lobe cortical volume and cortical surface area compared to healthy controls.[115,116] For example, an MRI study of 15 female patients with DID revealed a 19.2% decrease in hippocampal volume and a 31.6% decrease in amygdala volume compared to controls.[117] Case reports, along with small SPECT studies of patients with DID, have demonstrated consistently that compared to healthy controls and DID simulators, patients with DID show increased perfusion in the left temporal lobe and that temporal perfusion increases while in a dissociated self-state.[110,118–120] In a related case report, EEG differences between a series of dissociated self-states were most prominent in the left temporal area.[121] The exact neurobiological underpinnings of switching between self-states

remain under investigation. Notably, an fMRI of one patient performed during the process of voluntarily switching between self-states revealed right medial temporal inhibition, suggesting the possibility of temporal lobe involvement in this process.[122]

A growing body of recent literature has examined neurobiological differences between DID self-states. For example, one study compared two types of self-states in DID: trauma-fixated self-states that personally identified with, and were highly physiologically reactive to, the individual's traumatic memories and trauma-avoidant self-states that were amnestic for, or disowned, traumatic experiences. When exposed to masked neutral faces (that may have been perceived as threatening stimuli), trauma-fixated self-states showed increased activation in a range of areas including the occipitotemporal junction and parahippocampal gyrus when compared to DID-simulating controls.[123] Another study that differentiated the response of trauma-fixated and trauma-avoidant self-states to written traumatic scripts found that the trauma-avoidant self-states demonstrated activation patterns similar to those observed in patients with the dissociative subtype of PTSD.[40] Moreover, compared to trauma-fixated self-states, these trauma-avoidant self-states activated areas involved not only in overmodulation of emotional arousal but also areas with a proposed relationship to dissociative amnesia, including parahippocampal and right-sided temporal areas, as well as posterior association areas that included the fusiform and parahippocampal gyri.[40]

Assessment of Temporal Lobe Abnormalities: Limbic Irritability and the Limbic System Checklist-33

Limbic irritability has been identified as a key measure of altered temporal lobe function.[124] Limbic irritability may be assessed using the Limbic System Checklist (LSCL-33), which measures symptoms associated with ictal TLE, including somatic, sensory, behavioral, and mnemonic disturbances. Individuals exposed to childhood maltreatment score high on the LSCL-33 compared to controls who have not experienced maltreatment, providing further support for temporal lobe dysfunction in traumatized populations.[124]

Elevations on the LSCL-33 are also observed in samples of patients with pathological dissociation. In these studies, higher scores on the DES correlate with not only with elevated LSCL-33 scores but also with elevated scores on the Iowa Interview (a measure of complex partial seizure-like symptoms, such as dysnosmias, auditory hallucinations, anesthesias, and visual distortions) and other complex partial epileptic-like signs (visual anomalies, auditory-vibrational experiences, automatic behaviors, depersonalization, and "sense of a presence").[125–127] Notably, patients with BPD, which includes

dissociation among its diagnostic criteria, also exhibit elevated scores on the LSCL-33 compared to patients with depression and anxiety, along with scores comparable to those seen in patients with TLE.[109] On balance, the LSCL-33 appears a useful tool for quantifying the presence and severity of these symptoms among traumatized individuals.

Assessment of Malingering

The literature clearly indicates that patients with trauma-related disorders frequently experience psychotic-like symptoms, that these symptoms overlap substantially with the symptoms of TLE, and, finally, that there is a strong evidence of organic temporal lobe dysfunction among these patients. As such, clinicians should not be surprised by the presence of psychotic-like and TLE-like symptoms in patients with trauma-related disorders. Nevertheless, because these symptoms remain underexplored and often underdocumented and are atypical of the primary psychosis that clinicians may be more familiar with, these symptoms are typically misunderstood and misdiagnosed as either primary psychosis or as malingering. Indeed, these patients can be mistakenly classified as malingering not only by those instruments designed specifically to detect exaggeration of symptoms and malingering but also by the validity scales present in more general psychological tests.[128–135] As discussed earlier, these patients also typically present with a wide range of severe symptoms that span multiple diagnostic categories, which contributes further to invalid testing profiles and positive malingering screens.[7,8] For example, in one study of male combat veterans with PTSD and psychotic-like symptoms, 46% were classified as above the cutoff for malingering on the Miller Forensic Assessment of Symptoms Test (MFAST), mainly due to endorsement of unusual hallucinations (e.g., tactile hallucinations and visual hallucinations in black and white) and rare combinations of symptoms. However, when these patients were given the Structured Interview of Reported Symptoms (SIRS), only two scored within the malingering range.[11] Thus, although the presence of psychotic-like symptoms resembling those seen in TLE (i.e., tactile and visual hallucinations) was directly responsible for trauma patients being classified as malingering on the MFAST, when these symptoms were explored more fully, it was apparent that these represented false-positive classifications of malingering in the vast majority of patients, calling into question the utility of this screening tool in traumatized populations.

Structured Interview of Reported Symptoms. On the SIRS, a widely used structured interview intended to identify feigned psychological symptoms, high levels of diffuse pathology, and supposedly "rare" symptoms are taken as indicators of possible feigning. Critically, however, these so-called rare symptoms include common and expected manifestations of trauma-related disorders such as depersonalization and other dissociative symptoms. It is therefore not surprising that people with trauma-related disorders are at risk of being misidentified as feigning by the SIRS. Indeed, in one study of a DID population, 35% of patients with actual DID were misclassified by the SIRS as feigners.[128] Similarly, in a related study of severely traumatized patients, about half of whom also met criteria for DID, 31% were misclassified as feigners.[133]

In response to the limitations of the SIRS in assessing patients with trauma-related disorders, a revised version of the SIRS, the SIRS-2, was created. The SIRS-2 contains the same items as the SIRS but utilizes revised scoring and interpretation criteria.[129] In a recent study of the SIRS-2, more than half of the patients with DID scored above the total cutoff score for feigning (indicating endorsement of a wide range of severe symptoms) and higher than the genuine range on its Rare Symptoms Scale.[129] Moreover, 88% of patients with genuine DID endorsed at least one of the items on the Rare Symptoms Scale likely because this scale continues to includes dissociative symptoms (depersonalization and derealization). Ultimately, a combination of the SIRS-2 and the Trauma Index (TI) demonstrates the highest specificity in distinguishing genuine from feigned DID.[129] Taken together, this research clearly illustrates the urgent need to revise these instruments in a trauma-informed manner that allows for more accurate classification of this population and in doing so prevents potentially unjust adverse outcomes in disability and forensic proceedings.

Minnesota Multiphasic Personality Inventory. On the Minnesota Multiphasic Personality Inventory (MMPI-2), patients with DID can sometimes be mistaken for psychotic due to their reports of hearing voices (e.g., of their dissociated self-states). In some cases, DID patients score higher on the schizophrenia subscale than patients with diagnosed schizophrenia.[131,136] Patients with DID and patients with histories of complex trauma also tend to have elevated scores on the MMPI-2's validity scale, the F scale, which encompasses symptoms reported by less than 10% of the population against whom the test was normed.[130,131] F-scale items include experiencing bizarre sensations and unusual thoughts, although F-scale elevations were thought to be valid predictors of malingering. In one sample of female childhood sexual abuse survivors, F-scale elevations were associated with depression, dissociation, and posttraumatic stress, with dissociative symptoms being the best predictor for elevation on this scale.[137] Similarly, in a sample of patients with DID, F-scale elevations were associated with endorsement of known dissociative symptoms, including

blank spells, doing things without remembering them, and ideas of mind control, which are not rare in severely dissociative populations.[131,136] This research shows that in trauma-related disorders, the ability of the MMPI-2 to identify invalid profiles is severely limited by the overlap between F-scale items, which were uncommon in the normative sample on which the MMPI-2 was developed, and the dissociative and psychotic-like symptoms that are in fact common in trauma-related disorders.

Personality Assessment Inventory. The Personality Assessment Inventory (PAI) has three feigning indicators, including the *Negative Impression* (NIM) scale that is intended to measure rare or unusual symptoms, including memory loss and dissociative self-states, lack of positive memories in childhood, and the sense that others do not understand the severity of one's problems. Critically, the NIM is elevated significantly in samples of traumatized veterans, patients with PTSD, and patients with PTSD and DID, and unsurprisingly given their severe trauma exposure, the highest elevations occur in patients with DID.[134,138] In a pattern similar to that observed with the MMPI-2, these elevations in NIM appear affected by depression, dissociative symptoms, and borderline personality features.[135] Hence, it is clear that the PAI also overclassifies patients with trauma-related disorders as malingerers rather than as suffering from legitimate symptoms that are consistent with evidence-based findings about the impact of trauma.

Implications for Assessment. Across a wide range of psychological tests, including both screening malingering measures and more structured assessment tools, complex dissociative symptoms lead frequently to elevations on malingering scales.[128,129,131,133,134,136,137] These elevations are found consistently in a wide range of traumatized populations by numerous researchers in a variety of contexts, which suggests that the elevations are not due to malingering but rather due to the impact of trauma, including high levels of dissociation, and a severe, wide range of symptoms, leading to a significant burden of illness. Unfortunately, many clinicians and test developers are unaware of the high levels of legitimate psychopathology associated with trauma exposure, including seemingly rare and severe symptoms such as dissociative and "psychotic" symptoms. Moreover, the symptoms that are labeled as rare and unusual by these scales are instead typical and expected in traumatized population and are frequently part of the DSM-5 diagnostic criteria for trauma-related disorders (Table 3). For example, a common item on such subscales is experiences of leaving or feeling outside of one's body. In comparison, the DSM-5 (pp. 302–303) describes depersonalization (which is a part of the diagnostic criteria for PTSD) as "experiences of unreality, detachment, or being an outside observer" of oneself and specifically gives the example of "out of body experiences."[1] As has been previously reviewed, the literature on TLE and on temporal lobe dysfunction in patients with trauma-related disorders points strongly toward the temporal lobe hypothesis as a plausible explanation for these symptoms.

By misidentifying traumatized patients as malingerers, these instruments contribute to substantial delays in accurate diagnosis and effective treatment of trauma-related disorders. There may also be significant practical consequences. Notably, childhood trauma exposure and high levels of dissociation are common in forensic populations, and dissociative disorders are overrepresented compared to the general population. For example, one study found that 25% of a forensic sample had a dissociative disorder, which is more than twice the rate found in the general population.[139] Forensic patients, whose criminal responsibility may be assessed in part on the basis of psychological testing, and in whom dissociative disorders are overrepresented, may therefore be at particularly high risk for being misidentified as malingering. These decisions may have devastating forensic implications. For example, traumatized individuals may receive unjustly harsh sentences, including, in some countries, the death penalty, if the jury is not given the opportunity to consider fairly the impact of their genuine mental illness. Moreover, misclassifications of malingering can have serious medicolegal implications, for example, patients could have disability insurance claims unjustly denied if they are felt to be malingering, despite their legitimate and disabling symptoms. It is important to emphasize the life-altering pragmatic impact that an attribution of malingering can have in such circumstances.

If psychological testing is used in trauma-related disorders either for the purposes of diagnosis or in the forensic system in assessments of malingering, it is an ethical imperative to carefully consider the limitations of validity scales in these populations and to utilize classification rules with established validity in trauma-exposed populations (e.g., the TI from the SIRS or SIRS-2). Otherwise, assessors may draw conclusions that are not only inaccurate but have the potential to be highly damaging.

Discussion

Clinical Implications

Highly traumatized populations, who often meet criteria for multiple diagnoses including PTSD, BPD, OSDD, and DID, present frequently with psychotic-like symptoms that can be misidentified as malingering or primary psychosis. These include symptoms that overlap with those of schizophrenia (e.g., hearing voices) as well as psychotic symptoms that are not typical of schizophrenia

(e.g., tactile hallucinations) and symptom clusters that could be viewed as too bizarre, too severe, or too wide-ranging to be veracious. Clinically, there are several indicators that can assist with differential diagnosis (Table 1). For example, trauma-related psychotic-like symptoms often include nonauditory hallucinations (typically tactile, olfactory, or visual). When auditory hallucinations (voices) are present in highly traumatized individuals without a primary psychotic disorder, they are typically of an early onset (in the case of childhood trauma), and the presence of child voices is strongly suggestive of DID or OSDD.[15] It is also important that these assessments recognize (and educate patients about) the transdiagnostic nature of these symptoms, the etiologic role of trauma, and the limitations of current DSM-5 nosology in capturing the polysymptomatic presentations that are typical of trauma-related disorders.

The temporal lobe theory of dissociation provides clarification in understanding these symptoms within a neurobiological framework as well as evidence for their transdiagnostic nature. The literature shows that psychological trauma and a wide range of trauma-related disorders are associated with strikingly similar alterations in the structure and function of the temporal lobe (Table 2). Patients with complex trauma often experience psychotic-like symptoms identical to those observed in patients with organic temporal lobe dysfunction (particularly TLE), but these symptoms are frequently understood as psychotic or reflective of malingering, rather than as dissociative symptoms originating from the temporal lobe. The temporal lobe theory of dissociation therefore unifies dissociative symptoms and psychotic-like symptoms as a group of trauma-related symptoms and points strongly to trauma-related temporal lobe dysfunction as a

Table 1. Key features of primary psychosis, trauma-related disorders, and TLE.

	Primary psychosis	DID/OSDD	PTSD	TLE
Auditory hallucinations	Often	Often	Often	Possible
Internally vs. externally perceived voices	Either	Either	More likely internal	
Onset of voices	**After 18**	**Before 18**		
Number of voices	**Typically One**	**Typically Multiple**	**Typically one**	
Child voices	**Possible**	**Often**		
Nonauditory hallucinations	**Possible**	**Often**	**Often**	**Often**
Trauma-related content	Sometimes	Sometimes	Sometimes	
Schneiderian first-rank symptoms	Often	More often		
Trauma exposure	Often	Often	Necessary	
Dissociative symptoms	Possible	Necessary	Common	Common

DID: dissociative identity disorder; OSDD: other specified dissociative disorder; PTSD: post-traumatic stress disorder; TLE: temporal lobe epilepsy.
Note: bold face is to highlight key distinguishing features between disorders (versus those that do not discriminate).

Table 2. Temporal lobe dysfunction in trauma-related disorders.

	Trauma exposed	**PTSD**
Structural	Decreased volume (right)	Decreased volume (right)
	Reduced cortical thickness	
Functional	Altered resting state activity	Decreased blood flow (reexperiencing symptoms)
		Increased blood flow (dissociative symptoms)
	BPD	**Dissociative depression**
Structural	Decreased volume (right)	Unclear
Functional	Right hypometabolism	Unclear
	Depersonalization disorder	**DID/OSDD**
Structural	Decreased volume (right)	Decreased volume
Functional	Right hypometabolism	Left increased perfusion (in dissociated self-state)

BPD: borderline personality disorder; DID: dissociative identity disorder; OSDD: other specified dissociative disorder; PTSD: post-traumatic stress disorder.

neurobiological underpinning of this often misinterpreted trauma-related symptom cluster. For example, a patient with PTSD may experience episodes of depersonalization, identity alteration, olfactory hallucinations, and auditory verbal hallucinations. By applying the temporal lobe theory of dissociation, the clinician can understand that, rather than having a series of unconnected and bizarre symptoms, this patient may in fact have multiple TLE-like features that are all explained by their trauma-related disorder and by alterations in their temporal lobe structure and function.

When indicators of complex trauma and dissociation are present, a careful trauma history as well as a full review of dissociative symptoms should be undertaken to clarify the diagnosis. Tools such as the DES or Loewenstein's[140] semistructured Office Mental Status Examination for Chronic Complex Dissociative Symptoms can be invaluable in assessing these features. Complex partial seizure and TLE-like symptoms should also be specifically assessed, and, if TLE is ruled out as an etiology, these symptoms should be considered as the part of the trauma-related disorder rather than as indicative of a comorbid primary psychosis. They should therefore be managed according to the treatment guidelines for posttraumatic and dissociative disorders.[141] In the case of dissociative disorders, the prototypical example of trauma-related dissociation, we know that treatment targeted specifically at the dissociative disorder is associated with a range of improvements in symptoms and in functioning as well as with decreased psychiatric hospitalizations and decreased health-care utilization costs, making identification of these disorders even more paramount.[142,143]

Future Directions

Researchers are beginning to study, and when necessary, adapt, psychological tests to provide valid conceptualizations and classifications of patients with trauma-related disorders (e.g., the TI developed for the SIRS and SIRS-2). Much more research is needed to document and remediate existing deficiencies in the assessment of patients with trauma-related disorders, including those who present with psychotic-like symptoms (Table 3). Validity scales should be developed that do not rely on dissociative or TLE-like symptoms to identify malingering, thus allowing for more accurate assessment of malingering in this population.

Currently, there is a minimal literature on specific treatments for TLE-like symptoms in individuals who have experienced complex trauma. Expert opinion currently suggests that psychotic-like symptoms do not respond to the same treatments as primary psychotic disorders.[141] Although antipsychotics may assist with PTSD-related intrusions, thought organization, anxiety and irritability, they are not recommended for the treatment of hallucinations in patients with trauma-related disorders (either voices related to the intrusion of other dissociated self-states or complex partial seizure-like olfactory, tactile, and gustatory hallucinations).[141] Moreover, although antiepileptic mood-stabilizing medications such as lamotrigine, divalproex, and carbamazepine are sometimes used in this population to manage mood instability or comorbid bipolar disorder, the ability of antiepileptic medications to address TLE-like symptoms in nonepileptic complex trauma populations remains to be studied.[141]

Table 3. Areas of substantial overlap between validity scale items, dissociative symptoms, and temporal lobe epilepsy-like symptoms.

	LSCL-33 items	DSM-5 dissociative symptoms
SIRS rare symptoms		
Smelling strange odors	Smelling unusual or sweet odors	Olfactory hallucinations or flashbacks
Body parts look unfamiliar	Feeling of being outside oneself	Depersonalization
Feeling outside your body	Feeling you have left your body	Depersonalization/derealization
MMPI-2 infrequency items		
Feeling possessed by spirits	Feeling of being possessed	Identity alteration
Hearing voices	Hearing voices	Auditory hallucinations or flashbacks
Feeling you have left your body	Feeling you have left your body	Depersonalization/derealization
Seeing things others do not see	Seeing patterns, lights, or images	Visual hallucinations or flashbacks
Periods of memory loss		Dissociative amnesia
PAI NIM items		
Different personalities inside	Feeling of multiple personalities	Identity alteration
Inability to remember own identity		Dissociative amnesia
Complete memory loss		Dissociative amnesia

DSM: Diagnostic and Statistical Manual of Mental Disorders; LSCL: Limbic System Checklist; MMPI: Minnesota Multiphasic Personality Inventory; NIM: Negative Impression; PAI: Personality Assessment Inventory; SIRS: Structured Interview of Reported Symptoms.

Future work may also focus on nonpharmacological treatments, such as psychotherapy, or emerging adjunctive treatments, such as real-time fMRI neurofeedback to upregulate or downregulate relevant areas of the temporal lobe.[144]

Take Home Points

- Psychotic-like symptoms, particularly nonauditory hallucinations, are common across trauma-related disorders, as are dissociative symptoms.
- The psychotic-like and dissociative symptoms observed in trauma-related disorders, including in some cases experiences of possession and supernatural experiences, resemble those observed in TLE.
- The recent literature points toward the temporal lobe dysfunction as a potential unifying mechanism behind trauma-related and dissociative psychopathology in trauma-related disorders.
- These symptoms are often not assessed by clinicians or are misattributed to malingering, when instead they are core symptoms of trauma-related disorders.
- Misunderstanding of these symptoms can lead to adverse clinical outcomes, including misdiagnosis, the use of inappropriate medications, and wide-ranging medicolegal consequences.

Clinical Recommendations

1. Patients with trauma-related disorders should be screened for psychotic-like symptoms, and, if present, they should be considered part of the trauma-related disorder, provided that TLE has been ruled out.
2. Psychotic-like symptoms should be managed as per treatment guidelines for the trauma-related disorder in question, as antipsychotics are frequently not effective.
3. Psychological testing profiles should be interpreted with caution in patients with a history of trauma, and available literature on typical testing profiles and limitations should be incorporated into the interpretation. Further research and revision are necessary to reduce the inclusion of dissociative, psychotic-like, and other trauma-related symptoms on exaggeration and malingering indices.

Additional Comments

1. The literature on specific treatments for psychotic-like symptoms in trauma-related disorders is limited, thus limiting the ability to make detailed treatment recommendations.

2. There are limited neuroimaging studies that examine specifically the subpopulation of patients with psychotic-like symptoms and trauma-related disorders.

References

1. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*. Washington, DC: American Psychiatric Publishing, 2013.
2. Lyons-Ruth K, Dutra L, Schuder MR, Bianchi I. From infant attachment disorganization to adult dissociation: relational adaptations or traumatic experiences? *Psychiatr Clin North Am* 2006; 29(1): 63–86, viii.
3. Harari D, Bakermans-Kranenburg M, Van Ijzendoorn M. Attachment, disorganization, and dissociation. In: Vermetten E, Dorahy M, Spiegel D (eds) *Traumatic Dissociation: Neurobiology and Treatment*. Arlington, VA: American Psychiatric Publishing, 2007, p. 31–54.
4. Lyssenko L, Schmahl C, Bockhacker L, Vonderlin R, Bohus M, Kleindienst N. Dissociation in psychiatric disorders: a meta-analysis of studies using the Dissociative Experiences Scale. *Am J Psychiatry* 2018; 175(1): 37–46.
5. Spiegel D. Trauma, dissociation, and memory. *Ann N Y Acad Sci* 1997; 821: 225–237.
6. Spiegel D, Lewis-Fernández R, Lanius R, Vermetten E, Simeon D, Friedman M. Dissociative disorders in DSM-5. *Annu Rev Clin Psychol* 2013; 9: 299–326.
7. Brand B, Classen C, Lanins R, et al. A naturalistic study of dissociative identity disorder and dissociative disorder not otherwise specified patients treated by community clinicians. *Psychol Trauma* 2009; 1(2): 153.
8. Leonard D, Brann S, Tiller J. Dissociative disorders: pathways to diagnosis, clinician attitudes and their impact. *Aust N Z J Psychiatry* 2005; 39(10): 940–946.
9. Frewen P, Lanius RA. *Healing the Traumatized Self: Consciousness, Neuroscience, Treatment*, 1st ed. New York, NY: W W Norton, 2015.
10. Anketell C, Dorahy MJ, Shannon M, et al. An exploratory analysis of voice hearing in chronic PTSD: potential associated mechanismsz. *J Trauma Dissociation* 2010; 11(1): 93–107.
11. Lindley SE, Carlson EB, Hill KR. Psychotic-like experiences, symptom expression, and cognitive performance in

combat veterans with posttraumatic stress disorder. *J Nerv Ment Dis* 2014; 202(2): 91–96.

12. Norredam M, Jensen M, Ekstrøm M. Psychotic symptoms in refugees diagnosed with PTSD: a series of case reports. *Nord J Psychiatry* 2011; 65(4): 283–288.

13. Pearse LJ, Dibben C, Ziauddeen H, Denman C, McKenna PJ. A study of psychotic symptoms in borderline personality disorder. *J Nerv Ment Dis* 2014; 202(5): 368–371.

14. Dalenberg CJ, Brand BL, Gleaves DH, et al. Evaluation of the evidence for the trauma and fantasy models of dissociation. *Psychol Bull* 2012; 138(3): 550–588.

15. Dorahy MJ, Shannon C, Seagar L, et al. Auditory hallucinations in dissociative identity disorder and schizophrenia with and without a childhood trauma history: similarities and differences. *J Nerv Ment Dis* 2009; 197(12): 892–898.

16. Kluft RP. First-rank symptoms as a diagnostic clue to multiple personality disorder. *Am J Psychiatry* 1987; 144(3): 293–298.

17. Ross CA, Miller SD, Reagor P, Bjornson L, Fraser GA, Anderson G. Schneiderian symptoms in multiple personality disorder and schizophrenia. *Compr Psychiatry* 1990; 31(2): 111–118.

18. Filley CM. *Neurobehavioral Anatomy*, 3rd ed. Boulder, CO: University Press of Colorado, 2011.

19. Blanke O, Mohr C, Michel CM, et al. Linking out-of-body experience and self processing to mental own-body imagery at the temporoparietal junction. *J Neurosci* 2005; 25(3): 550–557.

20. Harricharan S, Rabellino D, Frewen PA, et al. fMRI functional connectivity of the periaqueductal gray in PTSD and its dissociative subtype. *Brain Behav* 2016; 6(12): e00579.

21. Olivé I, Densmore M, Harricharan S, Théberge J, McKinnon MC, Lanius R. Superior colliculus resting state networks in post-traumatic stress disorder and its dissociative subtype. *Hum Brain Mapp* 2018; 39(1): 563–574.

22. Feindel W, Penfield W. Localization of discharge in temporal lobe automatism. *AMA Arch Neurol Psychiatry* 1954; 72(5): 603–630.

23. Schenk L, Bear D. Multiple personality and related dissociative phenomena in patients with temporal lobe epilepsy. *Am J Psychiatry* 1981; 138(10): 1311–1316.

24. Devinsky O, Putnam F, Grafman J, Bromfield E, Theodore WH. Dissociative states and epilepsy. *Neurology* 1989; 39(6): 835–840.

25. Hara K, Adachi N, Akanuma N, et al. Dissociative experiences in epilepsy: effects of epilepsy-related factors on pathological dissociation. *Epilepsy Behav* 2015; 44: 185–191.

26. Ito M, Adachi N, Okazaki M, Kato M, Onuma T. Evaluation of dissociative experiences and the clinical utility of the Dissociative Experience Scale in patients with coexisting epilepsy and psychogenic nonepileptic seizures. *Epilepsy Behav* 2009; 16(3): 491–494.

27. Kuyk J, Spinhoven P, van Emde Boas W, van Dyck R. Dissociation in temporal lobe epilepsy and pseudo-epileptic seizure patients. *J Nerv Ment Dis* 1999; 187(12): 713–720.

28. Silberman EK, Post RM, Nurnberger J, Theodore W, Boulenger JP. Transient sensory, cognitive and affective phenomena in affective illness. A comparison with complex partial epilepsy. *Br J Psychiatry* 1985; 146: 81–89.

29. Sierra M, Berrios GE. The Cambridge Depersonalization Scale: a new instrument for the measurement of depersonalization. *Psychiatry Res* 2000; 93(2): 153–164.

30. Lambert MV, Sierra M, Phillips ML, David AS. The spectrum of organic depersonalization: a review plus four new cases. *J Neuropsychiatry Clin Neurosci* 2002; 14(2): 141–154.

31. Bowman ES, Coons PM. The differential diagnosis of epilepsy, pseudoseizures, dissociative identity disorder, and dissociative disorder not otherwise specified. *Bull Menninger Clin* 2000; 64(2): 164–180.

32. Benson DF, Miller BL, Signer SF. Dual personality associated with epilepsy. *Arch Neurol* 1986; 43(5): 471–474.

33. Hersh J, Chan YC, Smeltzer D. Identity shifts in temporal lobe epilepsy. *Gen Hosp Psychiatry* 2002; 24(3): 185–187.

34. Mesulam MM. Dissociative states with abnormal temporal lobe EEG. Multiple personality and the illusion of possession. *Arch Neurol* 1981; 38(3): 176–181.

35. Cantagallo A, Grassi L, Della Sala S. Dissociative disorder after traumatic brain injury. *Brain Inj* 1999; 13(4): 219–228.

36. Ball JS, Links PS. Borderline personality disorder and childhood trauma: evidence for a causal relationship. *Curr Psychiatry Rep* 2009; 11(1): 63–68.

37. Lanius RA, Williamson PC, Boksman K, et al. Brain activation during script-driven imagery induced dissociative responses in PTSD: a functional magnetic resonance imaging investigation. *Biol Psychiatry* 2002; 52(4): 305–311.

38. Lanius RA, Williamson PC, Bluhm RL, et al. Functional connectivity of dissociative responses in posttraumatic stress disorder: a functional magnetic resonance imaging investigation. *Biol Psychiatry* 2005; 57(8): 873–884.

39. Reinders AA, Willemsen AT, Vos HP, den Boer JA, Nijenhuis ER. Fact or factitious? A psychobiological study of authentic and simulated dissociative identity states. *PLoS One* 2012; 7(6): e39279.

40. Reinders AA, Willemsen AT, den Boer JA, Vos HP, Veltman DJ, Loewenstein RJ. Opposite brain emotion-regulation patterns in identity states of dissociative identity disorder: a PET study and neurobiological model. *Psychiatry Res* 2014; 223(3): 236–243.

41. Daalman K, Diederen KM, Derks EM, van Lutterveld R, Kahn RS, Sommer IE. Childhood trauma and auditory verbal hallucinations. *Psychol Med* 2012; 42(12): 2475–2784.

42. Gibson LE, Alloy LB, Ellman LM. Trauma and the psychosis spectrum: a review of symptom specificity and explanatory mechanisms. *Clin Psychol Rev* 2016; 49: 92–105.

43. Lataster T, van Os J, Drukker M, et al. Childhood victimisation and developmental expression of non-clinical delusional ideation and hallucinatory experiences: victimisation and non-clinical psychotic experiences. *Soc Psychiatry Psychiatr Epidemiol* 2006; 41(6): 423–428.

44. Scott J, Chant D, Andrews G, Martin G, McGrath J. Association between trauma exposure and delusional experiences in a large community-based sample. *Br J Psychiatry* 2007; 190: 339–343.

45. Arseneault L, Cannon M, Fisher HL, Polanczyk G, Moffitt TE, Caspi A. Childhood trauma and children's emerging

psychotic symptoms: a genetically sensitive longitudinal cohort study. *Am J Psychiatry* 2011; 168(1): 65–72.

46. Bentall RP, de Sousa P, Varese F, et al. From adversity to psychosis: pathways and mechanisms from specific adversities to specific symptoms. *Soc Psychiatry Psychiatr Epidemiol* 2014; 49(7): 1011–1022.

47. Varese F, Barkus E, Bentall RP. Dissociation mediates the relationship between childhood trauma and hallucination-proneness. *Psychol Med* 2012; 42(5): 1025–1036.

48. Lewis-Fernandez R. The proposed DSM-IV trance and possession disorder category: potential benefits and risks. *Transcul Psychiatr Res Rev* 1992; 29(4): 301–317.

49. Sar V, Alioğlu F, Akyüz G. Experiences of possession and paranormal phenomena among women in the general population: are they related to traumatic stress and dissociation? *J Trauma Dissociation* 2014; 15(3): 303–318.

50. Janssen I, Krabbendam L, Bak M, et al. Childhood abuse as a risk factor for psychotic experiences. *Acta Psychiatr Scand* 2004; 109(1): 38–45.

51. Choi JY, Choi YM, Kim B, Lee DW, Gim MS, Park SH. The effects of childhood abuse on self-reported psychotic symptoms in severe mental illness: mediating effects of posttraumatic stress symptoms. *Psychiatry Res* 2015; 229(1-2): 389–393.

52. Misiak B, Moustafa AA, Kiejna A, Frydecka D. Childhood traumatic events and types of auditory verbal hallucinations in first-episode schizophrenia patients. *Compr Psychiatry* 2016; 66: 17–22.

53. Trotta A, Murray RM, Fisher HL. The impact of childhood adversity on the persistence of psychotic symptoms: a systematic review and meta-analysis. *Psychol Med* 2015; 45(12): 2481–2498.

54. Kilcommons AM, Morrison AP. Relationships between trauma and psychosis: an exploration of cognitive and dissociative factors. *Acta Psychiatr Scand* 2005; 112(5): 351–359.

55. Perona-Garcelán S, Carrascoso-López F, García-Montes JM, et al. Dissociative experiences as mediators between childhood trauma and auditory hallucinations. *J Trauma Stress* 2012; 25(3): 323–329.

56. Moskowitz A, Read J, Farrelly S, Rudegeair T, Williams O. Are psychotic symptoms traumatic in origin and dissociative in kind? In: Dell PF, O'Neil JA (eds) *Dissociation and the Dissociative Disorders: DSM-V and Beyond*. New York, NY: Routledge, 2009, pp.521–534.

57. Sareen J, Cox BJ, Goodwin RD, J G Asmundson G. Co-occurrence of posttraumatic stress disorder with positive psychotic symptoms in a nationally representative sample. *J Trauma Stress* 2005; 18(4): 313–322.

58. Shevlin M, Armour C, Murphy J, Houston JE, Adamson G. Evidence for a psychotic posttraumatic stress disorder subtype based on the National Comorbidity Survey. *Soc Psychiatry Psychiatr Epidemiol* 2011; 46(11): 1069–1078.

59. Braakman MH, Kortmann FA, van den Brink W. Validity of 'post-traumatic stress disorder with secondary psychotic features': a review of the evidence. *Acta Psychiatr Scand* 2009; 119(1): 15–24.

60. Brewin CR, Patel T. Auditory pseudohallucinations in United Kingdom war veterans and civilians with posttraumatic stress disorder. *J Clin Psychiatry* 2010; 71(4): 419–425.

61. Ivezić S, Bagarić A, Oruc L, Mimica N, Ljubin T. Psychotic symptoms and comorbid psychiatric disorders in Croatian combat-related posttraumatic stress disorder patients. *Croat Med J* 2000; 41(2): 179–183.

62. Nijenhuis ER. Somatoform dissociation: major symptoms of dissociative disorders. *J Trauma Dissociation* 2001; 1(4): 7–32.

63. MacIntosh HB, Godbout N, Dubash N. Borderline personality disorder: disorder of trauma or personality, a review of the empirical literature. *Can Psychol* 2015; 56(2): 227.

64. Battle CL, Shea MT, Johnson DM, et al. Childhood maltreatment associated with adult personality disorders: findings from the Collaborative Longitudinal Personality Disorders Study. *J Pers Disord* 2004; 18(2): 193–211.

65. Zanarini MC, Gunderson JG, Marino MF, Schwartz EO, Frankenburg FR. Childhood experiences of borderline patients. *Compr Psychiatry* 1989; 30(1): 18–25.

66. Korzekwa MI, Dell PF, Pain C. Dissociation and borderline personality disorder: an update for clinicians. *Curr Psychiatry Rep* 2009; 11(1): 82–88.

67. Vermetten E, Spiegel D. Trauma and dissociation: implications for borderline personality disorder. *Curr Psychiatry Rep* 2014; 16(2): 434.

68. Merrett Z, Rossell SL, Castle DJ. Comparing the experience of voices in borderline personality disorder with the experience of voices in a psychotic disorder: a systematic review. *Aust N Z J Psychiatry* 2016; 50(7): 640–648.

69. Schroeder K, Fisher HL, Schäfer I. Psychotic symptoms in patients with borderline personality disorder: prevalence and clinical management. *Curr Opin Psychiatry* 2013; 26(1): 113–119.

70. Slotema CW, Daalman K, Blom JD, Diederen KM, Hoek HW, Sommer IE. Auditory verbal hallucinations in patients with borderline personality disorder are similar to those in schizophrenia. *Psychol Med* 2012; 42(9): 1873–1878.

71. Molina-Serrano A, Linotte S, Amat M, Souery D, Barreto M. Dissociation in major depressive disorder: a pilot study. *J Trauma Dissociation* 2008; 9(3): 411–421.

72. Sar V, Akyüz G, Oztürk E, Alioğlu F. Dissociative depression among women in the community. *J Trauma Dissociation* 2013; 14(4): 423–438.

73. Simeon D, Guralnik O, Schmeidler J, Sirof B, Knutelska M. The role of childhood interpersonal trauma in depersonalization disorder. *Am J Psychiatry* 2001; 158(7): 1027–1033.

74. Wearne D, Curtis GJ, Genetti A, Samuel M, Sebastian J. Where pseudo-hallucinations meet dissociation: a cluster analysis. *Australas Psychiatry* 2017; 25(4): 364–368.

75. Devinsky O, Lai G. Spirituality and religion in epilepsy. *Epilepsy Behav* 2008; 12(4): 636–643.

76. Ross CA. Possession experiences in dissociative identity disorder: a preliminary study. *J Trauma Dissociation* 2011; 12(4): 393–400.

77. Gold AL, Sheridan MA, Peverill M, et al. Childhood abuse and reduced cortical thickness in brain regions involved in emotional processing. *J Child Psychol Psychiatry* 2016; 57(10): 1154–1164.

78. Hanson JL, Chung MK, Avants BB, et al. Early stress is associated with alterations in the orbitofrontal cortex: a tensor-based morphometry investigation of brain structure and behavioral risk. *J Neurosci* 2010; 30(22): 7466–7472.

79. De Bellis MD, Keshavan MS, Frustaci K, et al. Superior temporal gyrus volumes in maltreated children and adolescents with PTSD. *Biol Psychiatry* 2002; 51(7): 544–552.

80. Tomoda A, Sheu YS, Rabi K, et al. Exposure to parental verbal abuse is associated with increased gray matter volume in superior temporal gyrus. *Neuroimage* 2011; 54(Suppl 1): S280–S286.

81. Ito Y, Teicher MH, Glod CA, Ackerman E. Preliminary evidence for aberrant cortical development in abused children: a quantitative EEG study. *J Neuropsychiatry Clin Neurosci* 1998; 10(3): 298–307.

82. Teicher MH, Ito Y, Glod CA, Andersen SL, Dumont N, Ackerman E. Preliminary evidence for abnormal cortical development in physically and sexually abused children using EEG coherence and MRI. *Ann N Y Acad Sci* 1997; 821: 160–175.

83. Philip NS, Kuras YI, Valentine TR, et al. Regional homogeneity and resting state functional connectivity: associations with exposure to early life stress. *Psychiatry Res* 2013; 214(3): 247–253.

84. Maheu FS, Dozier M, Guyer AE, et al. A preliminary study of medial temporal lobe function in youths with a history of caregiver deprivation and emotional neglect. *Cogn Affect Behav Neurosci* 2010; 10(1): 34–49.

85. Cheng B, Huang X, Li S, et al. Gray matter alterations in post-traumatic stress disorder, obsessive–compulsive disorder, and social anxiety disorder. *Front Behav Neurosci* 2015; 9: 219.

86. De Bellis MD, Keshavan MS, Shifflett H, et al. Brain structures in pediatric maltreatment-related posttraumatic stress disorder: a sociodemographically matched study. *Biol Psychiatry* 2002; 52(11): 1066–1078.

87. Freeman TW, Cardwell D, Karson CN, Komoroski RA. In vivo proton magnetic resonance spectroscopy of the medial temporal lobes of subjects with combat-related post-traumatic stress disorder. *Magn Reson Med* 1998; 40(1): 66–71.

88. Woodward SH, Schaer M, Kaloupek DG, Cediel L, Eliez S. Smaller global and regional cortical volume in combat-related posttraumatic stress disorder. *Arch Gen Psychiatry* 2009; 66(12): 1373–1382.

89. Daniels JK, Frewen P, Theberge J, Lanius RA. Structural brain aberrations associated with the dissociative subtype of post-traumatic stress disorder. *Acta Psychiatr Scand* 2016; 133(3): 232–240.

90. Kroes MC, Whalley MG, Rugg MD, Brewin CR. Association between flashbacks and structural brain abnormalities in posttraumatic stress disorder. *Eur Psychiatry* 2011; 26(8): 525–531.

91. Mutluer T, Şar V, Kose-Demiray Ç, et al. Lateralization of neurobiological response in adolescents with post-traumatic stress disorder related to severe childhood sexual abuse: the tri-modal reaction (T-MR) model of protection. *J Trauma Dissociation* 2018; 19(1): 108–125.

92. Brown S, Freeman T, Kimbrell T, Cardwell D, Komoroski R. In vivo proton magnetic resonance spectroscopy of the medial temporal lobes of former prisoners of war with and without posttraumatic stress disorder. *J Neuropsychiatry Clin Neurosci* 2003; 15(3): 367–370.

93. Lanius RA, Bluhm R, Lanius U, Pain C. A review of neuroimaging studies in PTSD: heterogeneity of response to symptom provocation. *J Psychiatr Res* 2006; 40(8): 709–729.

94. Felmingham K, Kemp AH, Williams L, et al. Dissociative responses to conscious and non-conscious fear impact underlying brain function in post-traumatic stress disorder. *Psychol Med* 2008; 38(12): 1771–1780.

95. Hopper JW, Frewen PA, van der Kolk BA, Lanius RA. Neural correlates of reexperiencing, avoidance, and dissociation in PTSD: symptom dimensions and emotion dysregulation in responses to script-driven trauma imagery. *J Trauma Stress* 2007; 20(5): 713–725.

96. Engdahl B, Leuthold AC, Tan HR, et al. Post-traumatic stress disorder: a right temporal lobe syndrome? *J Neural Eng* 2010; 7(6): 066005.

97. Miller DR, Hayes SM, Hayes JP, Spielberg JM, Lafleche G, Verfaellie M. Default mode network subsystems are differentially disrupted in posttraumatic stress disorder. *Biol Psychiatry Cogn Neurosci Neuroimaging* 2017; 2(4): 363–371.

98. Qin LD, Wang Z, Sun YW, et al. A preliminary study of alterations in default network connectivity in post-traumatic stress disorder patients following recent trauma. *Brain Res* 2012; 1484: 50–56.

99. Todder D, Levine J, Abujumah A, Mater M, Cohen H, Kaplan Z. The quantitative electroencephalogram and the low-resolution electrical tomographic analysis in posttraumatic stress disorder. *Clin EEG Neurosci* 2012; 43(1): 48–53.

100. Tursich M, Ros T, Frewen PA, Kluetsch RC, Calhoun VD, Lanius RA. Distinct intrinsic network connectivity patterns of post-traumatic stress disorder symptom clusters. *Acta Psychiatr Scand* 2015; 132(1): 29–38.

101. Nicholson AA, Densmore M, Frewen PA, et al. The dissociative subtype of posttraumatic stress disorder: unique resting-state functional connectivity of basolateral and centromedial amygdala complexes. *Neuropsychopharmacology* 2015; 40(10): 2317–2326.

102. Rossi R, Lanfredi M, Pievani M, et al. Abnormalities in cortical gray matter density in borderline personality disorder. *Eur Psychiatry* 2015; 30(2): 221–227.

103. Soloff P, Nutche J, Goradia D, Diwadkar V. Structural brain abnormalities in borderline personality disorder: a voxel-based morphometry study. *Psychiatry Res* 2008; 164(3): 223–236.

104. Völlm BA, Zhao L, Richardson P, et al. A voxel-based morphometric MRI study in men with borderline personality disorder: preliminary findings. *Crim Behav Ment Health* 2009; 19(1): 64–72.

105. Krause-Utz A, Winter D, Niedtfeld I, Schmahl C. The latest neuroimaging findings in borderline personality disorder. *Curr Psychiatry Rep* 2014; 16(3): 438.

106. Schmahl CG, Vermetten E, Elzinga BM, Douglas Bremner J. Magnetic resonance imaging of hippocampal and amygdala volume in women with childhood abuse and borderline personality disorder. *Psychiatry Res* 2003; 122(3): 193–198.

107. Schmahl C, Berne K, Krause A, et al. Hippocampus and amygdala volumes in patients with borderline personality disorder with or without posttraumatic stress disorder. *J Psychiatry Neurosci*. 2009; 34(4): 289–295.

108. Lange C, Kracht L, Herholz K, Sachsse U, Irle E. Reduced glucose metabolism in temporo-parietal cortices of women with borderline personality disorder. *Psychiatry Res* 2005; 139(2): 115–126.

109. Harris CL, Dinn WM, Marcinkiewicz JA. Partial seizure-like symptoms in borderline personality disorder. *Epilepsy Behav* 2002; 3(5): 433–438.

110. Sar V, Unal SN, Kiziltan E, Kundakci T, Ozturk E. HMPAO SPECT study of regional cerebral blood flow in dissociative identity disorder. *J Trauma Dissociation* 2001; 2(2): 5–25.

111. Sierra M, Nestler S, Jay EL, Ecker C, Feng Y, David AS. A structural MRI study of cortical thickness in depersonalisation disorder. *Psychiatry Res* 2014; 224(1): 1–7.

112. Simeon D, Guralnik O, Hazlett EA, Spiegel-Cohen J, Hollander E, Buchsbaum MS. Feeling unreal: a PET study of depersonalization disorder. *Am J Psychiatry* 2000; 157(11): 1782–1788.

113. Sierk A, Daniels JK, Manthey A, et al. White matter network alterations in patients with depersonalization/derealization disorder. *J Psychiatry Neurosci* 2018; 43(4): 170110.

114. Krüger C, Bartel P, Fletcher L. Dissociative mental states are canonically associated with decreased temporal theta activity on spectral analysis of EEG. *J Trauma Dissociation* 2013; 14(4): 473–491.

115. Chalavi S, Vissia EM, Giesen ME, et al. Similar cortical but not subcortical gray matter abnormalities in women with posttraumatic stress disorder with versus without dissociative identity disorder. *Psychiatry Res* 2015; 231(3): 308–319.

116. Reinders AATS, Chalavi S, Schlumpf YR, et al. Neurodevelopmental origins of abnormal cortical morphology in dissociative identity disorder. *Acta Psychiatr Scand* 2018; 137(2): 157–170.

117. Vermetten E, Schmahl C, Lindner S, Loewenstein RJ, Bremner JD. Hippocampal and amygdala volumes in dissociative identity disorder. *Am J Psychiatry* 2006; 163(4): 630–636.

118. Saxe GN, Vasile RG, Hill TC, Bloomingdale K, Van Der Kolk BA. SPECT imaging and multiple personality disorder. *J Nerv Ment Dis* 1992; 180(10): 662–663.

119. Schlumpf YR, Reinders AA, Nijenhuis ER, Luechinger R, van Osch MJ, Jäncke L. Dissociative part-dependent resting-state activity in dissociative identity disorder: a controlled FMRI perfusion study. *PLoS One* 2014; 9(6): e98795.

120. Semiz UB, Ebric S, Cetin M, Narin Y, Ozguven M. Regional brain blood flow changes in the 99mTc HMPAO SPECT assessment of patients with dissociative identity disorder. *Bull Clin Psychopharmacol* 2000; 10(4): 176–181.

121. Hughes JR, Kuhlman DT, Fichtner CG, Gruenfeld MJ. Brain mapping in a case of multiple personality. *Clin Electroencephalogr* 1990; 21(4): 200–209.

122. Tsai GE, Condie D, Wu MT, Chang IW. Functional magnetic resonance imaging of personality switches in a woman with dissociative identity disorder. *Harv Rev Psychiatry* 1999; 7(2): 119–122.

123. Schlumpf YR, Nijenhuis ER, Chalavi S, et al. Dissociative part-dependent biopsychosocial reactions to backward masked angry and neutral faces: an fMRI study of dissociative identity disorder. *Neuroimage Clin* 2013; 3: 54–64.

124. Teicher MH, Glod CA, Surrey J, Swett C. Early childhood abuse and limbic system ratings in adult psychiatric outpatients. *J Neuropsychiatry Clin Neurosci* 1993; 5(3): 301–306.

125. Bob P, Susta M, Pavlat J, Hynek K, Raboch J. Depression, traumatic dissociation and epileptic-like phenomena. *Neuro Endocrinol Lett* 2005; 26(4): 321–325.

126. Richards P, Persinger MA. Temporal lobe signs, the Dissociative Experiences Scale and the hemispheric quotient. *Percept Mot Skills* 1991; 72(3_suppl): 1139–1142.

127. Teicher MH, Samson JA, Polcari A, McGreenery CE. Sticks, stones, and hurtful words: relative effects of various forms of childhood maltreatment. *Am J Psychiatry* 2006; 163(6): 993–1000.

128. Brand BL, McNary SW, Loewenstein RJ, Kolos AC, Barr SR. Assessment of genuine and simulated dissociative identity disorder on the Structured Interview of Reported Symptoms. *J Trauma Dissociation* 2006; 7(1): 63–85.

129. Brand BL, Tursich M, Tzall D, Loewenstein RJ. Utility of the SIRS-2 in distinguishing genuine from simulated dissociative identity disorder. *Psychol Trauma* 2014; 6(4): 308.

130. Brand BL, Webermann AR, Frankel AS. Assessment of complex dissociative disorder patients and simulated dissociation in forensic contexts. *Int J Law Psychiatry* 2016; 49(Pt B): 197–204.

131. Brand BL, Chasson GS, Palermo CA, Donato FM, Rhodes KP, Voorhees EF. MMPI-2 item endorsements in dissociative Identity disorder vs. simulators. *J Am Acad Psychiatry Law* 2016; 44(1): 63–72.

132. Brand BL, Schielke HJ, Brams JS, DiComo RA. Assessing trauma-related dissociation in forensic contexts: addressing trauma-related dissociation as a forensic psychologist, part II. *Psychol Inj Law* 2017; 10: 298–312.

133. Rogers R, Payne JW, Correa AA, Gillard ND, Ross CA. A study of the SIRS with severely traumatized patients. *J Pers Assess* 2009; 91(5): 429–38.

134. Rogers R, Gillard ND, Wooley CN, Ross CA. The detection of feigned disabilities: the effectiveness of the Personality Assessment Inventory in a traumatized inpatient sample. *Assessment* 2012; 19(1): 77–88.

135. Stadnik RD, Brand B, Savoca A. Personality Assessment Inventory profile and predictors of elevations among dissociative disorder patients. *J Trauma Dissociation* 2013; 14(5): 546–561.

136. Welburn KR, Fraser GA, Jordan SA, Cameron C, Webb LM, Raine D. Discriminating dissociative identity disorder from schizophrenia and feigned dissociation on psychological tests and structured interview. *J Trauma Dissociation* 2003; 4(2): 109–130.

137. Klotz Flitter JM, Elhai JD, Gold SN. MMPI-2 F scale elevations in adult victims of child sexual abuse. *J Trauma Stress* 2003; 16(3): 269–274.

138. Calhoun PS, Collie CF, Clancy CP, Braxton LE, Beckham JC. Using the PAI in the assessment of posttraumatic stress disorder among help-seeking veterans. In: Blias MA, Baity MR, Hopwood CJ (eds) *Clinical Applications of the Personality Assessment Inventory*. New York, NY: Routledge, 2010. p. 93–112.

139. Spitzer C, Liss H, Dudeck M, et al. Dissociative experiences and disorders in forensic inpatients. *Int J Law Psychiatry* 2003; 26(3): 281–288.

140. Loewenstein RJ. An office mental status examination for complex chronic dissociative symptoms and multiple personality disorder. *Psychiatr Clin North Am* 1991; 14(3): 567–604.

141. International Society for the Study of Trauma and Dissociation. Guidelines for treating dissociative identity disorder in adults, third revision. *J Trauma Dissociation* 2011; 12(2): 115–187.

142. Brand BL, McNary SW, Myrick AC, et al. A longitudinal naturalistic study of patients with dissociative disorders treated by community clinicians. *Psychol Trauma* 2013; 5(4): 301.

143. Myrick AC, Webermann AR, Langeland W, Putnam FW, Brand BL. Treatment of dissociative disorders and reported changes in inpatient and outpatient cost estimates. *Eur J Psychotraumatol* 2017; 8(1): 1375829.

144. Orlov ND, Giampietro V, O'Daly O, et al. Real-time fMRI neurofeedback to down-regulate superior temporal gyrus activity in patients with schizophrenia and auditory hallucinations: a proof-of-concept study. *Transl Psychiatry* 2018; 8(1): 46.