24

A GROUNDED THEORY OF DISSOCIATIVE IDENTITY DISORDER

Placing DID in Mind, Brain, and Body

Lauren A. M. Lebois, Chloe S. Kaplan, Cori A. Palermo, Xi Pan, and Milissa L. Kaufman

In some ways, dissociative identity disorder (DID) is paradoxical (Loewenstein, 2020). It is both a psychiatric disorder and a marker of effective early developmental adaptation to overwhelming traumatic stress and confounding attachment dilemmas (Putnam, 1997; 2016). Historical controversies surrounding DID, and, at times, this paradoxical perspective, have impeded its empirical study and obscured the validity of existing findings. However, a burgeoning body of research has linked DID to a particular causal environment and to a variety of biological manifestations. Here we provide an innovative theory of DID that translates the phenomenology using modern models of cognition and neuroscience, and grounds DID in environmental experience, the brain and body.

What is DID?

DID is a posttraumatic psychobiological syndrome that develops in childhood to help the child survive overwhelming experiences (Putnam, 1997). It is demarcated by experiences of identity alteration and dissociative amnesia (APA, 2013). The timing of traumatic experience during childhood for individuals with DID disrupts typical identity development (Putnam, 2016). In typical development, children gradually coalesce a coherent sense of self that is perceived as stable across time, as well as emotional and behavioral contexts (Harter, 2015). Individuals with DID instead go down an alternative developmental pathway. Consequently, they experience behavioral and emotional states that subjectively feel discrete and remain unintegrated in adulthood (Putnam, 2016).

These seemingly discrete mental states are termed "personality states" or "identity states" in the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (APA, 2013) or sometimes called "alters" or "parts" in the literature. However, the term "self-states" acknowledges the subjectively discrete experience of these states without reifying them as actual "people" that inhabit the mind of someone with DID (Kluft, 1988). Furthermore, the term self-state permits a straightforward mapping of this experience in DID to current theories of cognition, and the existing empirical biological research on DID. To aid in our novel synthesis, we use the term self-states moving forward.

What is a Self-state?

To begin our discussion, it is important to note that all people have self-states – not just those with DID. A self-state is the subjective experience of a particular "way of being you" (Chefetz, 2015, p. 66). A self-state is a metaphor with "umph," a subjective experience in a living being, grounded in cognition that conceptually expands to include embodiment. Following theories of *grounded cognition*[1] (Barsalou, 2008), self-state as metaphor is grounded in the experience of the body and five senses in the world. In this section, we explain how the metaphor of the self-state is an actual embodied, dynamic construction used to help us predict how to act and be in the world. Then, by reflecting the language of DID self-states within our understanding of modern cognitive psychology and neuroscience, we hope to demystify and destigmatize the experience of identity alteration in DID. Self-states manifest and operate the way they do because that is just the way human minds and brains work according to the science of grounded cognition. We are all a collection of self-states, some collections more integrated and some more dissociative than others.

Our ability to create a variety of self-states is perhaps infinite. Indeed, we have aspects of subjective experience that may feel, for example, enthusiastic, inspired, nervous, attentive, determined, hungry, angry, ashamed, creative, loving, tense or some marvellous, messy combination thereof. A self-state is a dynamic construction that develops in memory over time. As such, it can change over time – it is not static. Given this, both learning and our unique experiences in the world play fundamental roles in the development and elaboration of self-states over time. Furthermore, our self-states can adapt to the context at hand (see Barsalou, 2010; Lebois et al., 2019 for examples of learning and context-dependence in emotion and conceptual knowledge).

Importantly, self-states are actually a dynamic network of brain activity. This brain activity is a collection of perceptions (e.g., vision), actions (e.g., proprioception), and internal states (e.g., affect) associated with that particular self-state based on our past experience (Barsalou, 2008). Given this, your subjective experience in a particular self-state might include what it feels like in your body, what you look like, attributes or skills you have, and how you feel about yourself in a particular context (and the brain activity associated with all of these perceptions, actions and internal states).

For example, in a specific self-state you might feel yourself breathing smoothly with a sense of ease in your body. You have the perception that you look solidly present and calm. You are aware of your ability to be a sensitive, listening presence. You feel a warm curiosity and sense that you are good. In another self-state, you might feel an adrenaline rush that sends tingles through your arms. You have the perception that you look calm on the outside but feel excited anticipation inside. You are aware of your ability to speak competently in front of an audience, and you feel a sense of mastery as you start talking.

Critically, when you are experiencing a particular self-state or even just thinking about that state, you are, to a certain extent, reactivating brain activity (memories) associated with past experiences linked to that state. This brain activity includes perceptions, actions, and internal states that are stored in memory (i.e., simulation, Barsalou, 2008). Having a mind and brain that works this way helps us predict what might happen in the environment and prepares our body to interact with the environment (Barsalou, 2020).

A self-state in DID, just like other self-states, is a constellation of perceptions, actions and internal states based on past experience stored in memory. In the same vein, the neural correlates of a self-state in DID are also a dynamic, distributed network of brain activity. To a certain extent, brain activity associated with past experience while in that self-state is reactivated when someone with DID is currently in that state or thinking about that self-state. Importantly, this reactivation is context-dependent, just like self-states experienced by someone without DID.

However, a self-state in DID is unique as compared to a self-state in someone without DID. In DID, one experiences shifts in subjectivity and agency such that the one self-state may become an observer while another aspect of self, another self-state, takes control of the "wheel" and drives on (R. Oxnam, personal communication, February 2016). Furthermore, in DID, self-states often feel non-autobiographical or ego-alien, that is, they feel distinctly separate as if they do not belong to the person, and often as if they belong to someone else (Brenner, 2001; Chefetz & Bromberg, 2004).

This ego-alien experience of one's sense of self is also termed a "not–me" experience (Sullivan, 2013). For example, a progression along this "not–me" continuum may include: 1. feeling like you are watching a self-state from above that belongs to you but simultaneously is somewhat separate; 2. feeling like a markedly different self-state that does not belong to you; and 3. feeling a complete loss of experiential awareness while in a different self-state (Brenner, 2001).

Often self-states in DID are dominated by a particular affect, set of memories, and behaviors (Kluft, 1988). A self-state in DID may also have a particular mental image or experience of the body associated with it (Loewenstein, 2020). In a particular self-state, you might feel numb and detached. For example, you perceive yourself to look like an adult woman (as you are), but your body does not feel real below the neck and your emotions seem far away or absent. In another self-state, you might feel like an angry young boy. In this self-state you feel that your body is smaller, like a child's, you perceive yourself to be physically strong and male (despite being an adult woman), and you feel a generalized sense that you are angry and somehow bad. In yet another self-state, you might feel even more separate, realizing only later that you went to work and competently made an important presentation in front of a large group of colleagues, leaving you confused as to how someone as anxious as you could perform in such a professional manner. Importantly, while DID self-states may have statistical regularities in their associated affect, memories and behaviors, they are not static, stable states – they are dynamic (just like any other self-state). The mind of an individual with DID is a collection of self-states or a dynamic, "self-state system" (Loewenstein, 2020).

Other Dissociative Symptoms Experienced in DID

Having a mind made up of a dynamic, self-state system is commonly associated with a number of related discontinuities and alterations in experience. These discontinuities and alterations disrupt executive functioning and one's sense of self

in a variety of ways, and span a range of experiences including general memory problems, depersonalization, derealization, posttraumatic flashbacks, somatoform symptoms, trance, voice hearing, speech/thought insertion or withdrawal, "made" experiences (i.e., feelings, emotions, impulses, actions), temporary loss of knowledge, and amnesia (Dell, 2009). Dell hypothesizes that, in DID, these experiences are actually dissociative intrusions in which a self-state intrudes into the conscious experience of another self-state.

Often individuals with DID are aware of these symptoms (e.g., depersonalization, made emotions). However, some occur outside awareness (e.g., amnesia). Importantly, these experiences largely occur internally, not overtly (Dell, 2009). Below we describe three dissociative experiences that are characteristic of DID, including depersonalization, "made" experiences, and amnesia (see Dell, 2009 for a complete review). These features are further explored in the empirical biological sections of this review.

Depersonalization is the experience of detachment or disconnection from one's sense of self or body (Dell, 2009). In DID, depersonalization experiences include prototypical examples such as feeling like you are watching yourself from outside your body or that parts of your body feel unreal (Steinberg, 1994). However, in DID depersonalization becomes so severe that it intersects with identity alteration. For example, individuals may experience a loss of agency where it feels as if their emotions, thoughts, and behaviors emerge without their control (i.e., "made" or intrusive feelings, emotions, impulses, actions; APA, 2013; Dell 2006b). As described in the previous section, sometimes this experience is coupled with a feeling that emotions, thoughts, behaviors and/or their body are not their own, and at times that they belong to someone else (APA, 2013). Importantly, individuals with DID logically understand these experiences must be their own even though they feel alien (Dell, 2009). Overall, depersonalization functions adaptively to provide distance from overwhelming affect by creating the illusion of, for example, physical distance or "not-me-ness" (Brenner, 2001).

Dissociative amnesia includes gaps in memory for autobiographical information or experiences (APA, 2013). It can be either persistent or recurrent and the content traumatic or nontraumatic in nature. The amnesia can be generalized to one's life and identity (e.g., a fugue in which someone does not remember who they are or their personal history), localized to the details of a specific time period (e.g., ages 6–10 are 'missing') and/or selective for only some details during a period of time (e.g., Sunday visits to my grandparents in childhood are just blank; Steinberg, 1994). In DID, dissociative amnesia functions adaptively to preserve attachment bonds by compartmentalizing highly conflicted thoughts and feelings about abusive caregivers (Liotti, 1992).

Observable Manifestations of Self-states

The self-state and associated dissociative intrusions reviewed so far are experienced largely internally, as subjective qualities of experience in someone with DID (Dell, 2009). This makes it challenging to find ways of confirming that someone is experiencing these symptoms beyond relying solely on their self-report. Brain and body-based measures of these experiences, however, can augment subjective self-reports. Research in DID is beginning to develop paradigms to capture the objectively observable manifestations of self-states and dissociative intrusions in biology and performance on various tasks. The rest of this review is dedicated to a novel synthesis of this empirical work – grounding DID in experience in the world and body.

Grounding DID in Environmental Risk Factors

Like other psychiatric disorders, the etiology of DID includes a combination of genetic and environmental factors. Genetic variation moderates the association between environmental risk factors and psychiatric disorders (Caspi & Moffitt, 2006). For example, an individual with a low genetic loading for DID might not develop this disorder despite chronic, severe childhood trauma. In contrast, someone with a high genetic loading for DID might develop this disorder with a less severe childhood trauma load. In this way, there could be different pathways to the development of DID. This gene by environment interaction is understudied in psychiatry, let alone in DID specifically; however, patterns are beginning to emerge for DID and pathological dissociation more generally. Given that there are currently no DID-specific molecular genetic studies, here we focus on grounding DID in common environmental risk factors.

Overwhelming Childhood Experiences, Trauma, and Abuse

Clinical reports, empirical work, epidemiological studies, and meta-analyses support a moderate relationship between trauma and dissociation, and trauma and dissociative disorders, especially DID (Dalenberg et al., 2012). This work has

been conducted in both children and adults in the general population, clinical, and DID-samples across several countries (Dorahy et al., 2014). Research specific to DID has found high rates of many different maltreatment types (Dalenberg et al., 2012), and often individuals with DID have been found to have a higher trauma load than any other psychiatric group (Loewenstein, 2020).

Given ethical constraints, the work examining the relationship between trauma and dissociation, and trauma and DID, is necessarily correlational. However, prospective, longitudinal evidence following individuals in the direct aftermath of an acute trauma has demonstrated the link between trauma and dissociation (Carlson et al., 2016; Daniels et al., 2012), further supporting the strength of this relationship and the probability that trauma is causally linked to the occurrence of dissociation and DID (cf., Dell, Chapter 14, this volume). In addition, a history of childhood trauma in individuals with DID has been verified using hospital, police, and child protection agency records, providing additional support for the direct link between trauma and DID (e.g., Chu et al., 1999; Coons, 1994).

In light of this evidence, the field has largely moved beyond simply documenting the association between maltreatment, dissociation, and/or DID to instead identifying the distinct aspects of maltreatment leading to the development of DID specifically. In particular, a pattern of severe, chronic childhood trauma has emerged in almost all systematic studies of DID (Dorahy et al., 2014). Typically, this trauma is relational and occurs between the child and an attachment figure (Liotti, 1992; Ross, Norton, & Wozney, 1989). The abuse is often physical or sexual in nature. For example, rates of childhood physical abuse were around 84% and rates of sexual abuse ranged from 74% to 90% in a sample of individuals with DID (Ross & Ness, 2010).

While the relationship between DID and childhood physical/sexual abuse is often emphasized, data also suggests verbal abuse, emotional abuse, and emotional neglect are also strongly associated with adulthood dissociative symptoms (e.g., Teicher, Samson, Polcari & McGreenery, 2006; Şar, Dorahy, & Krüger, 2017). Of the different maltreatment types, emotional maltreatment may be among the strongest predictors of dissociative symptoms (King et al., 2020). For example, in a sample of psychiatric patients, childhood emotional neglect by immediate family members had the strongest association with an adult dissociative disorder diagnosis (Krüger & Fletcher, 2017).

While the majority of research has associated the environmental etiology of DID with severe, chronic childhood relational abuse by caregivers, two variations are of note. First, chaotic family dynamics characterized by frequent mood swings, uncontrollable anger, dissociation, paranoid ideation, and subclinical features of borderline personality disorder may also contribute, and in some cases, may be sufficient environmental circumstances for the development of DID (Ozturk & Şar, 2006). Second, chronic, painful, traumatic medical procedures performed by caregivers with non-malicious intent during developmentally sensitive time periods in childhood have also been associated with dissociative symptomatology in adults (Diseth, 2006), though this association has not yet been tested in DID specifically.

Disorganized Attachment

In addition to overwhelming childhood experiences, researchers theorize that the etiology of DID includes a disorganized attachment style (Barach, 1991; Blizard, 2003; Liotti, 1992; 1999). Disorganized attachment is marked by inconsistent and conflicting attachment behaviors (also known as Disoriented or Type D attachment; Main & Solomon, 1986). Infants with this attachment style display early dissociative processes such as trance-like or contradictory behavior (Main & Hesse, 1990; Main & Solomon, 1986). For example, in the strange situation paradigm where children are briefly separated and then reunited with caregivers, the child may approach their parent and simultaneously freeze in greeting them, even if they had been screaming during their separation.

Disorganized attachment is frequently observed in children who have been abused or neglected, or whose early caregiver was unavailable, inconsistent, or insensitive (Barach, 1991; Blizard, 2003; Carlson, 1998; Carlson et al., 1989; Lyons-Ruth et al., 2006). Longitudinal research has demonstrated that disorganized attachment in infancy predicts dissociation in adulthood, and a combination of disorganized attachment and trauma predicts elevated levels of dissociation in clinical and non-clinical populations (Carlson, 1998; Coe et al., 1995; Dutra et al., 2009; Ogawa et al., 1997).

It is hypothesized that disorganized attachment may contribute specifically to the etiology of DID by facilitating the development and elaboration of self-states that feel subjectively separate (Liotti, 1992; 2004). Furthermore, it is posited that the more confounding the early attachment relationship, the higher the likelihood that the child will later develop a more severe case of DID (Blizard, 1997). If there had been secure and consistent early attachments, it is thought that later trauma would not produce self-states (Liotti, 1992, 1999; McFadden, 2011).

Although the connection between trauma, disorganized attachment and dissociation in adulthood has been established, to date, no systematic empirical developmental research has focused specifically on DID samples and the hypothesized etiological contributions of disorganized attachment. This is a critical direction for future research.

Grounding DID in Biological Patterns

Foundational work has grounded DID in biology and begun to map out the biological mechanisms that underpin the experience of DID. Both neuroimaging and psychophysiological methodologies highlight that the symptoms of DID are associated with observable biological patterns (for recent systematic reviews see Lotifina, Soorgi, Mertens & Daniels, 2020; Roydeva & Reinders, 2020; See also Nijenhuis, Chapter 38, this volume). Here we review brain activity, anatomy, and peripheral physiology associated with DID.

Neurobiology of DID

Neurobiological research in DID has focused largely on differentiating self-states on the basis of activity in the brain. This body of work has shown observable differences between self-states across various paradigms. In addition, preliminary work has examined the neural correlates of switching between states and the process of self-state integration. Furthermore, DID brain structure and activity has been compared to nonpsychiatric controls and to controls pretending to have DID. Emerging patterns place DID on a continuum with PTSD and add to the body of literature supporting DID as a trauma-spectrum disorder, not a disorder of role-playing or fantasy involvement. More recent work points toward a fingerprint of DID in brain structure and function.

Common Methodologies

Common neuroimaging modalities used in DID research span direct and indirect measures of brain activity. Direct measures of brain electrical activity include electroencephalogram (EEG) and a derivative of EEG, called event-related potentials (ERP), that ties activity to a stimulus presentation. Indirect measures of brain activity track metabolism in the brain by leveraging blood flow, blood oxygen levels, blood water protons, or exogenous tracers injected into the bloodstream. Techniques that indirectly measure brain activity through metabolism include functional magnetic resonance imaging (fMRI), arterial spin labelling, positron emission tomography (PET), and single-photon emission computerized tomography (SPECT). EEG and ERP, as direct measures of brain activity, have strong temporal resolution, but poorer spatial resolution, that is, they can better pinpoint the timing of brain activity as opposed to the specific location of that activity. In contrast, techniques like fMRI and PET imaging have high spatial resolution, but poorer temporal resolution, meaning they can better pinpoint the location of brain activity as opposed to the timing of when that activity occurred. These methodological strengths and weaknesses should be kept in mind when interpreting the findings that follow.

Neural Correlates of Self-states

Self-states have Different Activity

Most of the neurobiological work in DID has focused on testing whether different self-states exhibit distinct patterns of brain activity. The majority of evidence demonstrates that there is differential brain activity across self-states. This has been observed across several task paradigms and while the participant is at "rest" with no task or experimenter-directed external stimulus (e.g., Reinders et al., 2003; 2006; Schlumpf et al., 2014). It has also been detected using a range of imaging methodologies including EEG, ERP, SPECT, PET, arterial spin labelling fMRI, and fMRI. Only a handful of exceptions have reported no differences between self-states in at least some experimental conditions (Coons et al., 1982; Cocores, Bender & McBride, 1984; Şar et al., 2001). We will discuss why this is not problematic at the end of this section.

The pattern of differential brain activity across self-states depends on the experimental context. Contexts studied to date range from intentionally emotionally provocative tasks to more mundane tasks, to no task (i.e., resting-state). The pattern is also influenced by brain imaging modality. For example, during a masked angry and neutral face fMRI paradigm, differential activity between self-states occurred in the parahippocampal gyrus (Schlumpf et al., 2013), whereas in a PET study, listening to trauma scripts elicited differential self-state activity across the brain in cortical (e.g., lateral and medial prefrontal cortex, cingulate, superior/inferior parietal lobule, occipital regions) and subcortical areas (e.g.,

parietal operculum, insula, amygdala, caudate, globus pallidus; Reinders et al., 2003; 2006). Two memory-related tasks demonstrated different EEG alpha coherence (Hopper et al., 2002), and different P300 ERP amplitudes across self-states (Allen & Movius, 2000). Several early studies flashed different light intensities to compare signal changes in the brain across self-states (Braun, 1983; Coons et al., 1982; Larmore, Ludwig, & Cain, 1977; Ludwig, Brandsma, Wilbur et al., 1972; Putnam, 1984). Most reported qualitatively-assessed differences distributed across the brain, although one study demonstrated quantified latency and amplitude differences (Larmore et al., 1977), and another showed differences across delta, beta, and theta frequency bands when comparing self-states (Coons et al., 1982). Most differences occurred in parietal and temporal regions.

Resting-state studies also report self-state differences distributed across alpha, beta, theta, and delta frequency bands that occur most consistently in the temporal lobe region, though some studies demonstrate differences in frontal, parietal, and occipital regions (Coons et al., 1982; Flor-Henry et al., 1990; Hughes, Kuhlman, Fichtner, & Gruenfeld, 1990; Lapointe, Crayton, Devito et al., 2006; Ludwig et al., 1972; Thigpen & Cleckley, 1954; Morselli, 1953). One arterial spin labelling resting-state fMRI study localized differences in self-state brain activity to the dorsomedial prefrontal cortex, somatosensorimotor, superior parietal, middle temporal regions and thalamus (Schlumpf et al., 2014). Altogether this work demonstrates brain-based self-state activity is identifiable across emotion, memory, perception, and resting-state tasks with neural correlate patterns that vary with the experimental context.

Switching between Self-states

The process of transitioning between self-states has also received some neurobiological attention. Two fMRI case studies have begun to identify brain activity implicated in this transition, also colloquially termed "switching." One study identified differential activity in the hippocampus, parahippocampal gyrus, medial temporal regions, substantia nigra, and globus pallidus, depending on which state the individual was transitioning to (Tsai, Condie, Wu, & Chang, 1999). In another case, researchers implicated pre and postcentral gyrus, nucleus accumbens, and lateral prefrontal cortex in the switching process (Savoy, Frederick, Keuroghlian, Wolk, 2012; Wolk, Savoy, & Frederick, 2012).

Self-state Integration

Recovery from DID is associated with no longer experiencing self-states that feel distinctly separate and feel as if they do not belong to the person or seem to belong to someone else. A small body of case study work has begun to examine this process by looking at brain function at various levels of integration. For example, topographic maps of two individuals with DID pre-integration looked different compared to post integration EEG maps (Braun, 1983). Another case study demonstrated cerebral blood flow patterns were different pre versus post integration (Mathew, Jack & West, 1985). The blood flow pattern was relatively stable across experimental sessions for the individual with DID post integration – similar to stability demonstrated by nonpsychiatric controls. Two individuals with higher levels of integration demonstrated a neural marker of memory recognition (i.e., a higher P300 amplitude) for words learned by a different self-state, whereas two individuals with lower levels of integration did not demonstrate this neural marker for words learned in a different self-state (Allen & Movius, 2000). Finally, one study examined structural brain differences between individuals with DID and those who have recovered from DID. Recovered individuals had significantly larger hippocampal volume compared to individuals with current DID (Ehling, Nijenhuis & Krikke, 2008).

Summary

The research reviewed here suggests that self-states in DID are dynamic, distributed networks of brain activity and specific differences or similarities between states in any moment are context-dependent. Previous work has hypothesized that self-states have different neural correlates because they are associated with a different moods, emotional states, levels of arousal, or senses of ownership over memories. Likely all of these contribute to differential neural activity between self-states. Following theories of grounded cognition (Barsalou, 2008), the reason for this is that a self-state is made up of a constellation of perceptions, actions and internal states stored in memory and supported by a dynamic network of brain activity.

A historical critique of this work argued that if brain differences were not observed between self-states – or if differences observed could be attributed to fluctuations in mood or arousal – then this would delegitimize DID. Of course, this logic is flawed. Because self-states are embodied, dynamic constructions grounded in perception, action, and introspection based on past experience, they will be paired with different perceptions, actions, internal states, and thus, different levels of valence and arousal.

Along the same lines, states may not differ in all contexts, at least at the level discernible by current technologies. Accordingly, differential brain activity related to mood or arousal does not suggest that self-states are not "real." Furthermore, failure to show differences between states in a particular task also does not suggest that self-states are not "real." A more nuanced question is what neural activity may be unique to DID self-states? For example, someone without DID will show different neural activity in the same task if they complete it once while feeling angry, and again while feeling fearful or numb. How does self-state activity in DID look different from this? The collection of results reviewed so far does not answer this question.

DID Versus Nonpsychiatric Controls and People Simulating DID

A body of work that does start to answer the question of what is neurobiologically unique to DID contrasts both brain structure and function to nonpsychiatric controls. A subset of this work also contrasts DID self-state activity to brain activity of individuals, often hired actors, pretending to experience DID self-states. While the experimental practice of comparing individuals with DID to DID-simulating controls does shed some light on what may be neurobiologically unique to DID, this paradigm has largely been used to test alternative theories of DID that attribute this disorder to fantasy proneness, suggestibility, and factitiousness. We review each set of studies in turn.

Nonpsychiatric Controls

Brain structure. Most brain anatomy work comparing DID and control samples have focused on the hippocampus. The hippocampus is centrally involved in learning and memory processing (Squire & Zola-Morgan, 1991), and is sensitive to the neurotoxic effects of chronically activated stress hormones (Conrad, 2008). The majority of studies find that the hippocampus is smaller and abnormally shaped in DID groups compared to nonpsychiatric control groups (Chalavi et al., 2015a; Chalavi et al., 2015b; Ehling et al., 2008; Irle, Lange, Sachsse, & Weniger, 2009; Tsai et al., 1999; Vermetten, Schmahl, Lindner, Loewenstein & Bremner, 2006). In addition to group level comparisons, some work has also demonstrated a negative correlation between hippocampal size and dissociative symptom severity in DID samples or mixed samples that include some individuals with DID (Chalavi et al., 2015b; Ehling et al., 2008; Stein, Koverola, Hanna, Torchia & McClarty, 1997).

There is also evidence of structural differences in other brain regions in DID samples. For example, some studies show DID groups have smaller amygdala, smaller parahippocampal gyrus, and larger pallidum volumes compared to controls (Chalavi et al., 2015a; Ehling et al., 2008; Vermetten et al., 2006). One exception to the hippocampal and amygdala patterns was shown in a mixed DID / Dissociative Amnesia group without comorbid PTSD (Weniger, Lange, Sachsse & Irle, 2008). This sample had no difference in hippocampal or amygdala volume compared to nonpsychiatric controls.

Brain Function

Most work comparing DID to nonpsychiatric control brain activity has focused on resting-state activity, attention, memory, and spatial tasks. This work is split between studies that compare self-states and those that do not. Studies without self-state comparisons typically demonstrate differences in brain activity related to working memory, spatial navigation, and memory recognition for individuals with DID. For example, a mixed DID and dissociative disorder not otherwise specified (DDNOS, milder presentation of DID) group outperformed nonpsychiatric controls on difficult trials in a verbal working memory fMRI task and had more brain activity in typical working memory brain regions compared to controls (e.g., lateral PFC, parietal cortex; Elzinga et al., 2007).
Similarly, a mixed DID/Dissociative amnesia sample performed comparably to controls in an fMRI virtual maze navigation (Weniger et al., 2013). Brain activity looked similar across groups, except the dissociative disorder sample had less activity in the postcentral gyrus, inferior parietal lobule, insula, superior temporal gyrus, cingulate, caudate and thalamus compared to controls.
A series of SPECT studies at rest demonstrated increased regional cerebral blood flow in the temporal lobes (Şar et al, 2001), prefrontal cortex and occipital lobes, and decreased orbitofrontal cortex cerebral blood flow in a DID sample compared to controls (Şar, Unal, & Ozturk, 2007). Finally, a cohort including some individuals with DID had less EEG connectivity compared to controls after adult attachment interview provocation (Farina et al., 2014).

Studies that incorporated different self-states into the design have demonstrated differential activity related to verbal, spatial, and memory tasks, and rest conditions. For example, a DID sample showed a smaller ERP P300 amplitude, a marker of memory recognition, compared to a control group in a memory assessment (Allen & Movius, 2000). In

addition, several studies found contrasting results related to signal variability across tasks and rest. One found that brain activity in a DID group showed less variability across verbal and spatial tasks compared to control activity as measured by EEG (Flor–Henry et al., 1990). In contrast, DID self–states exhibited more EEG signal and regional cerebral blood flow variability at rest compared to controls across different sessions, but less variability than comparing controls to one another (Mathew et al., 1985; Lapointe et al., 2006).

Simulating Controls

Across numerous experimental designs and imaging methodologies no DID-simulating control groups have reproduced the differences in brain activity between self-states demonstrated by DID groups (Coons et al., 1982; Hopper et al., 2002; Hughes et al., 1990; Putnam, 1984; Reinders et al, 2014; Schlumpf et al., 2013; Schlumpf et al., 2014). Activity between groups was different even when controls were prone to deep involvement in fantasy (Reinders et al., 2016; Reinders, Willemsen, Vos, den Boer & Nijenhuis, 2012). Importantly, the simulating controls did demonstrate differences in neural activity when they were in different states, but these patterns did not correspond to the patterns observed in DID samples.

Summary

Altogether, studies comparing DID participants to controls suggest that the neural correlates of DID involve a dynamic, distributed network of brain activity and that specific differences between DID and nonpsychiatric controls are task-dependent. To date, the strongest structural evidence is for hippocampal abnormalities in DID. Additionally, there are two emerging patterns to highlight in brain function. 1) There is some evidence that under some working and spatial memory task conditions dissociative disorder samples match or outperform control performance, but exhibit different brain activity (Elzinga et al., 2007; Weniger et al., 2013). This suggests executive and visuospatial functioning may be conserved under these circumstances and differential DID versus control brain activity may either represent a compensatory mechanism or unique processing style. However, these studies employed mixed dissociative disorders samples. It is yet unclear if a DID-only sample would replicate these results. 2) Control groups simulating DID have not replicated self-state patterns exhibited by individuals with DID, suggesting DID is not a disorder of role-playing, fantasy proneness, suggestibility, fabrication, and/or factitiousness.

This collection of results also begins to answer the question of what is unique to DID and self-state activity (in comparison to simulation of these states). It does not, however, fully tease out how differential self-state activity is distinct from the differential brain activity someone without DID would display when completing a task multiple times in different states (e.g., feeling angry, fearful, or numb). More targeted studies eliciting and isolating the felt sense that feelings, thoughts, and memories are nonautobiographical would further contribute to understanding the neural correlates of DID. Moreover, a limitation of the findings discussed here is that they do not demonstrate what is specific to DID versus general psychopathology. Further insight into the neural correlates specific to DID requires psychiatric control groups.

Placing DID on a Continuum with PTSD Neurobiology – Evidence of a Posttraumatic Adaptation

Perhaps unsurprisingly, given DID is a trauma-spectrum disorder, research is beginning to show that the neural correlates of DID overlap with the neurobiological underpinnings of PTSD. The research paradigm demonstrating this overlap is termed "symptom provocation." In these studies, participants often listen to recordings of their own trauma narratives while brain activity is being measured indirectly using fMRI or PET imaging. Listening to a trauma narrative presumably activates a participant's PTSD symptoms. Brain activity during the trauma narrative is then compared to activity that occurs when participants are listening to a non-trauma or "neutral" narrative. This approach allows researchers to isolate neural activity related to posttraumatic stress symptoms.

Symptom provocation studies show that a key neurobiological mechanism of dissociation in PTSD and DID is an excess of corticolimbic inhibition (Lanius et al., 2002; 2006; Reinders et al., 2014). For example, on average, individuals with frequent depersonalization/derealization experiences and PTSD, that is, the dissociative subtype of PTSD, exhibit hyperactivity in cortical areas of the brain involved in emotion and arousal regulation (e.g., ventromedial prefrontal cortex, rostral anterior cingulate cortex). In contrast, limbic regions are often hypoactive (e.g., amygdala, insula; Lanius et al., 2002; 2006; see Schiavone & Lanius, Chapter 39, this volume). This pattern, in part, underpins the experience of numbness and detachment individuals with this PTSD subtype report.

Several resting-state functional connectivity studies support these findings. They reveal entrenched patterns of emotion and arousal over-regulation in the way brain areas communicate in individuals with significant trauma-related dissociative symptoms – even while the individual is at rest with no engagement in a task (e.g., Harricharan et al., 2016; Nicholson et al., 2015).

In contrast, on average, individuals with classic PTSD, that is, no/very few or infrequent depersonalization/derealization symptoms, exhibit a pattern of failed corticolimbic inhibition in these situations (Lanius et al., 2001; 2002; 2006). They have hyperactivity in limbic areas of the brain involved in relevance detection (amygdala) and bodily experience (insula), but hypoactivity in cortical regions involved in regulating the limbic activity (e.g., ventromedial prefrontal cortex, rostral anterior cingulate cortex). This pattern, in part, underpins the experience of hyperarousal and emotion under-regulation in PTSD.

Studies of DID using the symptom provocation paradigm mirror these contrasting PTSD findings. Specifically, when individuals with DID are in a hyperaroused self-state (also termed "trauma identity/personality state" or "emotional part"), brain patterns resemble failed corticolimbic inhibition patterns in classic PTSD (Reinders et al., 2014; Reinders et al., 2016). In contrast, when individuals with DID are in a numb, detached self-state (also termed "neutral identity/personality state" or "apparently normal part"), brain patterns resemble excessive corticolimbic inhibition patterns in the dissociative subtype of PTSD (Reinders et al., 2014; Reinders et al., 2016). These findings suggest that individuals with DID oscillate between common PTSD brain patterns when thinking about and re-experiencing their own traumatic events.

SPECT-based work supports these conclusions when an individual with DID is at rest. Şar and colleagues (2007) found increased activity in medial and lateral prefrontal areas for individuals with DID compared to control participants.

Structural Findings

In addition to brain function, several studies have demonstrated similar brain structure in DID and PTSD, with some distinguishing patterns that may be specific to DID symptomatology. Structural hippocampal findings have been central to neurobiologically connecting PTSD and DID.

One of the largest neuroimaging studies of PTSD to date definitively demonstrated smaller hippocampal volume in PTSD versus trauma exposed controls (Logue et al., 2018). As discussed earlier, smaller hippocampal volume has also been demonstrated in DID and dissociative disorder cohorts (Chalavi et al., 2015a; Chalavi et al., 2015b; Ehling et al., 2008; Irle et al., 2009; Tsai et al., 1999; Vermetten et al., 2006; however, c.f., Weniger et al., 2008 for contrasting evidence). Furthermore, hippocampal volume was negatively associated with the severity of trauma history (Chalavi et al., 2015a; Chalavi et al., 2015b). When brain structure was directly compared between DID and PTSD cohorts, both had smaller whole brain, frontal, temporal, and insular gray matter volume compared to nonpsychiatric controls (Chalavi et al., 2015a). In addition, the DID group had larger putamen and pallidum compared to the PTSD group, and the volume of these structures was positively associated with dissociative symptom severity (Chalavi et al., 2015a). Furthermore, some hippocampal subfields, CA4–dentate gyrus and subiculum, were smaller in DID compared to PTSD (Chalavi et al., 2015b).

Summary

Taken together, this functional and structural brain imaging work continues to highlight DID as a posttraumatic developmental adaptation, and it has begun to place DID on a continuum with PTSD. Corticolimbic inhibition has emerged as a mechanism of depersonalization and derealization in both PTSD and DID, and individuals with DID display either an excess or failure of corticolimbic inhibition depending on the experimental conditions and self-state. Future work is needed to identify the neural correlates specific to other forms of dissociation experienced by individuals with DID.

Furthermore, the basal ganglia are emerging as key regions that may distinguish PTSD and DID given structural findings related to the putamen and pallidum (Chalavi et al., 2015a), differential brain activity between self-states in the caudate and pallidum (Reinders et al., 2006), and activity in the nucleus accumbens and substantia nigra while transitioning between self-states (Savoy et al., 2012; Wolk et al., 2012; Tsai et al., 1999). The basal ganglia have been implicated in diverse functions including habit, skill, and reward learning, working memory, motor control, planning, reasoning, problem solving, language, and processing emotional stimuli (Packard & Knowlton, 2002; Stocco, Lebiere, & Anderson, 2010; Pierce & Péron, 2020). Overall, they may serve to gate and perhaps select or coordinate diverse, competing response patterns across cognitive, affective and motoric domains (Pierce & Péron, 2020; Stocco et al., 2010). Thus, we hypothesize that these structures may be critical to the experience of a mind made up of a self-state system with many competing and conflicting thoughts and feelings.

On a final note, while there are several studies directly comparing DID to PTSD groups in brain structure, there are currently no studies that directly make this group comparison with brain activity or connectivity. This is a major gap in the data needed to distinguish overlapping versus unique patterns between PTSD and DID.

A Neurobiological Fingerprint of DID?

Case studies aside, the work reviewed so far tells us about average patterns of brain structure and function in DID. However, this does not mean that any one individual with DID would necessarily approximate this average pattern given the substantial variability in brain structure and function across individuals. It is still an open question whether it is possible to look at an individual's brain and determine whether or not they have DID. However, recent machine learning-based work has begun to answer this question. Machine learning is a type of artificial intelligence in which computer algorithms build a model based on a set of data and use that model to make predictions. Importantly, these predictions are not explicitly programmed by the experimenter.

Using only data about brain structure, pattern classifiers could distinguish between DID and nonpsychiatric control brains with relatively high sensitivity and specificity (Reinders et al., 2018). Both DID-related increases and decreases in gray and white matter volume distributed across the brain drove the prediction in this model. Similarly, a recent study demonstrated an individual's overall pathological dissociation score, a marker of DID symptom severity (Dell, 2006a), could be estimated using only brain functional network connectivity (Lebois et al., 2021). Two networks that emerged as central to the results were the default mode and frontoparietal control network. The default mode network is implicated in internally-focused attention often related to self-related processing (Raichle, 2015), whereas the frontoparietal control network is involved in executive functioning like problem solving and working memory (Menon, 2011). These networks are coactive during internally-focused problem solving or planning (Spreng, Stevens, Chamberlain, Gilmore, & Schacter, 2010). A pattern of hyperconnectivity between the default mode network and frontoparietal control network emerged as the most predictive of DID pathology. This indicates that brain activity in these networks is synchronized in DID. Furthermore, this pattern was measured across conditions of rest, a challenging attention task, and an emotional faces paradigm, indicating synchronicity between default mode and frontoparietal control networks may be a task-independent biomarker of pathological dissociation. In aggregate, these two studies move us closer to identifying a structural and functional "fingerprint" of DID in the brain that could be used to reliably identify someone with DID on an individual basis.

Peripheral Psychophysiology of DID

A small body of work has begun to ground DID in peripheral psychophysiology. Depending on the experimental context and self-state, individuals with DID fluctuate between patterns reflective of hyper and hypoarousal in their peripheral physiology. Furthermore, psychophysiological markers demonstrate that self-states sometimes display similar or disparate patterns depending on the emotional content of the stimuli.

Common Methodologies

Most psychophysiological work in DID focuses on electrodermal activity, that is, changes in electrical activity of the skin. Increased skin conductance reflects increased arousal. Other common recordings include heart rate, heart rate variability and blood pressure. Increased heart rate variability demonstrates flexibility in autonomic response, which is typically thought to be adaptive, whereas decreased variability implies a rigid response despite changes in context. Changes in heart rate cause blood pressure to rise or fall, for example, as the heart beats more times per minute, blood pressure rises as blood vessels expand to allow more blood to flow more efficiently. Some studies also leverage electromyography (EMG) to measure electrical activity produced by muscles in various paradigms. In particular, EMG has been used to measure muscle movement near the eye when an individual startles (i.e., eye blink startle response).

Psychophysiology of Self-states

Like the neurobiological work, most peripheral psychophysiological research has focused on demonstrating different patterns across self-states within an individual with DID. All studies conducted to date have demonstrated different patterns across self-states in the peripheral psychophysiology; however, the pattern depends on the experimental

context. For example, two case studies demonstrated skin conductance response differences across states when reading emotionally provocative words (Ludwig et al., 1972; Prince & Peterson, 1908). The increased skin conductance response was only present when the words were identified as personally meaningful in a specific self-state. All states, however, had an increased skin conductance response in a conditioning task regardless of the state in which the conditioning originally occurred (Ludwig et al., 1972).

Several other case studies conceptually replicated and extended these findings. Specifically, changes in respiration, heart rate, and skin conductance response were observed across different states (Bahnson & Smith, 1975). Changes in light intensity elicited a different pattern across self-states in heart rate, skin conductance, blood pressure, and in particular, EMG (Larmore et al., 1977). In addition, skin conductance response changed across self-states, and the pattern was different depending on whether the participant was attempting to control versus express emotions (Brende, 1984). This case study work is bolstered by two group-based studies that also demonstrated, across self-states, different heart rate and respiration patterns in a rest and habituation paradigm (Putnam, Zahn, & Post, 1990), and different blood pressure, heart rate and heart rate variability in a symptom provocation paradigm (Reinders et al., 2006).

Simulating Controls

Some work compared self-states in DID to those generated by controls simulating DID. Across rest, a habituation paradigm, and a symptom provocation paradigm, no simulating controls have replicated peripheral psychophysiological patterns demonstrated across DID self-states in heart rate, heart rate variability, respiration, skin conductance, or blood pressure, even when those controls were prone to involvement in fantasy (Putnam et al., 1990; Reinders et al., 2014; Reinders et al., 2016; Reinders et al., 2012). Importantly, as in the neurobiological studies, the simulating controls often did demonstrate differences in peripheral psychophysiology when simulating different states; it just was not the same pattern as the DID samples.

Summary

Studies on the self-state psychophysiology of DID, taken together with neurobiological studies, demonstrate that self-states are dynamic, distributed networks of brain activity that prepare the body to interact with the world in a particular way. Given this, self-states are "visible" in both the central and peripheral nervous system and differences across states are context-dependent. An emerging pattern based on this work suggests that differences in self-states may be more readily discernible in more emotionally provocative contexts (e.g., Ludwig et al., 1972; Reinders et al., 2006). Additionally, the fact that controls simulating DID could not replicate DID self-state patterns again suggests DID is not a disorder of role-playing, fantasy proneness, suggestibility, fabrication, and/or factitiousness.

Placing DID on a Continuum with PTSD Psychophysiology – Evidence of a Posttraumatic Adaptation

Much like the neurobiological work on DID demonstrating overlap with PTSD, the peripheral psychophysiology is beginning to show a parallel overlap. As described earlier, the research paradigm with the strongest evidence for this overlap is symptom provocation. These studies show that an excess of corticolimbic inhibition during depersonalization and derealization is visible in the peripheral nervous system in both PTSD and DID (Lanius et al., 2002; 2006; Reinders et al., 2014). For example, on average, individuals with the dissociative subtype of PTSD show no change or a decrease in heart rate while listening to trauma narratives (Lanius et al., 2002). In contrast, on average, individuals with classic PTSD (i.e., no or infrequent depersonalization/derealization symptoms), show an increased heart rate during these conditions compared to control participants (e.g., Lanius et al., 2001).

These contrasting PTSD results are mirrored in DID using the symptom provocation paradigm (Reinders et al., 2006; Reinders et al., 2012; Reinders et al., 2014; Reinders et al., 2016). When individuals with DID are in a hyperaroused self-state, peripheral psychophysiology patterns resemble failed corticolimbic inhibition patterns in classic PTSD. Specifically, individuals with DID demonstrate higher heart rate frequency and systolic blood pressure and decreased average heart rate variability while listening to trauma narratives. In contrast, when individuals with DID are in a numb, detached self-state, peripheral psychophysiology patterns resemble excessive corticolimbic inhibition patterns in the dissociative subtype of PTSD. Specifically, individuals with DID demonstrate lower heart rate, systolic blood pressure, and more heart rate variability. A symptom provocation case study supports these conclusions, demonstrating a decrease in heart rate during a trauma narrative when the participant with DID was experiencing depersonalization (Williams, Haines & Sale, 2003). This suggests that individuals with DID oscillate between common PTSD patterns in the autonomic nervous system when thinking about and re-experiencing their own traumatic events.

Two studies have also demonstrated reduced or a trend toward reduced habituation in DID groups (Dale et al., 2008; Putnam, Zahn, & Post, 1990). For example, individuals with DID maintained a startle response in EMG signal to auditory stimuli, whereas control participants no longer startled after some time in the task (Dale et al., 2008). This reduced habituation pattern is typically found in PTSD samples during conditioning paradigms and reflects a failure of extinction and inhibitory learning (Lebois et al., 2019).

Summary

In aggregate, peripheral psychophysiology work continues to highlight DID as a posttraumatic developmental adaptation, and like the neurobiological work, it has begun to place DID on a continuum with PTSD. Corticolimbic inhibition is not only measurable in brain activity, but also visible in the peripheral physiology as a mechanism of depersonalization and derealization in both PTSD and DID. Individuals with DID display either an excess or failure of corticolimbic inhibition depending on the experimental conditions and self-state. Notably, no studies have directly compared DID and PTSD within one study to more definitively determine overlap versus physiological correlates that may be unique to DID. This is a key direction for future work.

Digging Deeper: DID Research Moving Forward

Conclusions

In this review, we have grounded DID in modern theories of cognition, and experience in the world and body. Research suggests that self-states are dynamic, distributed networks of brain activity that prepare the body to interact with the world in a particular way. This distributed network of brain activity represents a constellation of perceptions (e.g., vision), actions (e.g., movement) and internal states (e.g., affect) stored in memory based on past experience.

To a certain extent, brain activity associated with past experience while in a specific self-state is reactivated when someone is currently in that state or thinking about that self-state, and this reactivation is context-dependent. Using this grounded cognition framework demystifies seemingly contradictory evidence in biological studies of DID and self-states in which different states do not always show consistent patterns across experimental conditions. These findings are not actually conflicting – one would anticipate situation-specific manifestations given this conceptualization of self-states. Similarly, given this theory, valence, levels of arousal, emotion, or felt sense of ownership over memories could all or at various times contribute to different patterns seen across self-states in neuroimaging and psychophysiological paradigms. This helps to sharpen predictions in future work examining self-states in DID.

Work to date shows that DID is associated with childhood trauma, severely dysfunctional family dynamics, and in theory, disorganized attachment. These environmental circumstances interact with a genetic predisposition for the capacity to dissociate. An important implication of this work is that these environmental circumstances and experiences must occur during an early developmental window for DID to manifest – an acute onset adult trauma in the absence of early childhood trauma does not produce DID. In a related line of thinking, given gene by environmental interactions in other psychiatric disorders, even these environmental circumstances will likely not always produce DID if the person does not have a genetic predisposition towards dissociation or their genetic loading for DID is very low.

Finally, research demonstrates that DID is grounded in unique biological patterns. Self-states have differential brain activity and peripheral physiology across various paradigms and levels of integration. DID has distinguishable patterns in brain structure, function, and peripheral psychophysiology compared to nonpsychiatric controls and people simulating DID. Additionally, corticolimbic inhibition is a key mechanism of depersonalization and derealization in DID. Lastly, cutting edge machine learning work suggests DID and pathological dissociation can be identified in the brain on an individual basis–an early proof of concept demonstration moving us one step closer to having a neurobiological fingerprint of DID that could be used to identify someone with these experiences just by looking at their neurobiology. Altogether, this work is beginning to build a body of evidence demonstrating what many clinicians and people with lived experience have long known–DID is a valid disorder and a developmental posttraumatic adaptation.

Remaining Questions

Notwithstanding the reviewed seminal work, many gaps remain in our understanding of DID. For example, the literature linking DID with disorganized attachment and the development of self-states is currently theoretical. There are no genetic or epigenetic studies of DID, and no brain function or psychophysiological studies directly comparing DID and PTSD cohorts. Also, differing levels of self-state integration are large sources of unaccounted for variance in

research findings to date. Furthermore, while brain differences between DID and control cohorts have been identified, a remaining question is whether these differences in brain structure and function are pre-existing vulnerabilities that increase the likelihood of developing DID following severe childhood trauma or if they are changes that develop as adaptations to severe childhood trauma – or some combination thereof. These gaps point toward exciting opportunities for new research.

In light of this synthesis, we have several recommendations for future work grounding DID in world and bodily experience. First, an overwhelming amount of evidence supports DID as a valid diagnosis with strong content, criterion and construct validity (Dorahy et al., 2014). Given this pivotal work, we may be at a juncture in the field where we can move beyond research efforts whose central focus is to prove or disprove the "reality" of DID. In a similar vein, it will be helpful to diversify comparison groups beyond those that simulate DID to controls that allow us to pinpoint even more nuanced psychobiological mechanisms in DID. For example, nonpsychiatric controls in altered states of consciousness (e.g., hypnosis) or experiencing perceptual illusions (e.g., rubber hand illusions) may be fruitful comparisons. Likewise, psychiatric controls will allow us to identify mechanisms that are unique to DID as opposed to more general psycho-pathology. In particular, comparison to the different subtypes of PTSD will further elucidate common and unique characteristics of DID as a trauma-spectrum disorder, and comparison to bipolar disorder may shed light on shared versus unique correlates of mental state shifts.

Several topics that remain understudied in DID include state versus trait dissociation, self-related processing, basic science models, developmental trajectories, treatment studies and individual variability in DID presentation. For example, we have yet to identify reliable markers of state-based fluctuations in dissociation. Robust biomarkers are critical for future clinical trials of trauma and dissociation-related treatment. Likewise, nascent attempts at basic science models of dissociation have recently emerged (Meloni et al., 2016; Vesuna et al., 2020), and more work in this area may also help identify novel treatment targets down the line.

Additionally, few studies exist that systematically or longitudinally document child manifestations of DID and early intervention, and none that we are aware of measure biological mechanisms. Not only would this inform treatment modalities and developmental trajectories in DID, but it would also inform yet to be identified mechanisms of typical identity development in comparison.

Lastly, like PTSD, DID is a heterogeneous disorder (Kluft, 1999; Ross, 1997). Biological studies may help us distinguish between different DID subtypes and stratify cohorts for future clinical trials. The field is currently standing on fertile ground for future research and greater insight into DID.

Acknowledgments

Note

1 Traditional theories of cognition suggest knowledge is stored in memory separate from the brain's systems for perception (e.g., olfaction), action (e.g., movement), and introspection (e.g., mental states). Backed by neuroscience, grounded cognition instead proposes these systems underly cognition.

References

Allen, J. J., & Movius, H. L., 2nd. (2000). The objective assessment of amnesia in dissociative identity disorder using event-related potentials. *International Journal of Psychophysiology*, *38*, 21–41.

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*. Arlington, VA: American Psychiatric Publishing.

Bahnson, C. B., & Smith, K. (1975). Autonomic changes in a multiple personality. *Psychosomatic Medicine*, *37*, 85–86.

Barach, P. M. (1991). Multiple personality disorder as an attachment disorder. *Dissociation*, *4*, 117–123.

Barsalou, L. W. (2008). Grounded cognition. *Annual Review of Psychology*, *59*, 617–645.

Barsalou, L. W. (2010). Grounded cognition: past, present, and future. *Topics in Cognitive Science*, *2*, 716–724.

Barsalou, L. W. (2020). Challenges and opportunities for grounding cognition. *Journal of Cognition*, *3*, 1–24.

Blizard, R. A. (1997). The origins of dissociative identity disorder from an object relations and attachment theory perspective. *Dissociation*, *10*, 223–229.

Blizard, R. A. (2003). Disorganized Attachment, Development of Dissociated Self States, and a Relational Approach to Treatment. *Journal of Trauma & Dissociation*, *4*(3), 27–50.

Braun, B. G. (1983). Neurophysiologic changes in multiple personality due to integration: a preliminary report. *The American Journal of Clinical Hypnosis*, *26*, 84–92.

Brende, J. O. (1984). The psychophysiologic manifestations of dissociation. Electrodermal responses in a multiple personality patient. *The Psychiatric Clinics of North America*, 7, 41–50.

Brenner, I. (2001). *Dissociation of trauma: Theory, phenomenology, and technique.* Madison, CT: International Universities Press, Inc.

Carlson, E. A. (1998). A prospective longitudinal study of attachment disorganization/disorientation. *Child Development*, *69*, 1107–1128.

Carlson, E. B., Field, N. P., Ruzek, J. I., Bryant, R. A., Dalenberg, C. J., Keane, T. M., & Spain, D. A. (2016). Advantages and psychometric validation of proximal intensive assessments of patient-reported outcomes collected in daily life. *Quality of Life Research: An International Journal of Quality of Life Aspects of Treatment, Care and Rehabilitation*, *25*, 507–516.

Carlson, V., Cicchetti, D., Barnett, D., & Braunwald, K. (1989). Disorganized/disoriented attachment relationships in maltreated infants. *Developmental Psychology*, *25*, 525–531.

Caspi, A., & Moffitt, T. E. (2006). Gene–environment interactions in psychiatry: joining forces with neuroscience. *Nature Reviews. Neuroscience*, 7, 583–590.

Chalavi, S., Vissia, E. M., Giesen, M. E., Nijenhuis, E. R. S., Draijer, N., Barker, G. J., Veltman, D. J., & Reinders, A. A. T. S. (2015a). Similar cortical but not subcortical gray matter abnormalities in women with posttraumatic stress disorder with versus without dissociative identity disorder. *Psychiatry Research*, *231*, 308–319.

Chalavi, S., Vissia, E. M., Giesen, M. E., Nijenhuis, E. R. S., Draijer, N., Cole, J. H., Dazzan, P., Pariante, C. M., Madsen, S. K., Rajagopalan, P., Thompson, P. M., Toga, A. W., Veltman, D. J., & Reinders, A. A. T. S. (2015b). Abnormal hippocampal morphology in dissociative identity disorder and post-traumatic stress disorder correlates with childhood trauma and dissociative symptoms. *Human Brain Mapping*, *36*, 1692–1704.

Chefetz, R. A. (2015). Intensive Psychotherapy for Persistent Dissociative Processes: The Fear of Feeling Real. New York: WW Norton & Company.

Chefetz, R. A., & Bromberg, P. M. (2004). Talking with "me" and "not-me": A dialogue. *Contemporary Psychoanalysis*, *40*, 409–464.

Chu, J. A., Frey, L. M., Ganzel, B. L., & Matthews, J. A. (1999). Memories of childhood abuse: dissociation, amnesia, and corroboration. *American Journal of Psychiatry*, *156*, 749–755.

Cocores, J. A., Bender, A. L., & McBride, E. (1984). Multiple personality, seizure disorder, and the electroencephalogram. *Journal of Nervous and Mental Disease*, *172*(7), 436–438.

Coe, M. T., Dalenberg, C. J., Aransky, K. M., & Reto, C. S. (1995). Adult attachment style, reported childhood violence history and types of dissociative experiences. *Dissociation*, *8*, 142–154.

Conrad, C. D. (2008). Chronic stress-induced hippocampal vulnerability: the glucocorticoid vulnerability hypothesis. *Reviews in the Neurosciences*, *19*, 395–411.

Coons, P. M. (1994). Confirmation of childhood abuse in child and adolescent cases of multiple personality disorder and dissociative disorder not otherwise specified. *Journal of Nervous and Mental Disease*, *182*, 461–464.

Coons, P. M., Milstein, V., & Marley, C. (1982). EEG studies of two multiple personalities and a control. *Archives of General Psychiatry*, *39*, 823–825.

Dale, K. Y., Flaten, M. A., Elden, A., & Holte, A. (2008). Dissociative identity disorder and prepulse inhibition of the acoustic startle reflex. *Neuropsychiatric Disease and Treatment*, *4*, 653–662.

Dalenberg, C. J., Brand, B. L., Gleaves, D. H., Dorahy, M. J., Loewenstein, R. J., Cardeña, E., Frewen, P. A., Carlson, E. B., & Spiegel, D. (2012). Evaluation of the evidence for the trauma and fantasy models of dissociation. *Psychological Bulletin*, *138*, 550–588.

Daniels, J. K., Coupland, N. J., Hegadoren, K. M., Rowe, B. H., Densmore, M., Neufeld, R. W. J., & Lanius, R. A. (2012). Neural and behavioral correlates of peritraumatic dissociation in an acutely traumatized sample. *Journal of Clinical Psychiatry*, *73*, 420–426.

Dell, P. F. (2006a). The multidimensional inventory of dissociation (MID): A comprehensive measure of pathological dissociation. *Journal of Trauma & Dissociation*, 7, 77–106.

Dell, P. F. (2006b). A new model of dissociative identity disorder. *Psychiatric Clinics of North America*, *29*, 1–26.

Dell, P. F. (2009). The Phenomena of Pathological Dissociation. In P. F. Dell & J. A. O'Neil (Eds.), *Dissociation & the Dissociative Disorders: DSM-V & Beyond* (pp. 225–237). New York, NY: Routledge.

Diseth, T. H. (2006). Dissociation following traumatic medical treatment procedures in childhood: a longitudinal follow-up. *Development and Psychopathology*, *18*, 233–251.

Dorahy, M. J., Brand, B. L., Sar, V., Krüger, C., Stavropoulos, P., Martínez-Taboas, A., Lewis-Fernández, R., & Middleton, W. (2014). Dissociative identity disorder: An empirical overview. *Australian and New Zealand Journal of Psychiatry*, *48*, 402–417.

Dutra, L., Bureau, J.-F., Holmes, B., Lyubchik, A., & Lyons-Ruth, K. (2009). Quality of early care and childhood trauma: a prospective study of developmental pathways to dissociation. *Journal of Nervous and Mental Disease*, *197*, 383–390.

Ehling, T., Nijenhuis, E. R. S., & Krikke, A. P. (2008). Volume of discrete brain structures in complex dissociative disorders: preliminary findings. *Progress in Brain Research*, *167*, 307–310.

Elzinga, B. M., Ardon, A. M., Heijnis, M. K., De Ruiter, M. B., Van Dyck, R., & Veltman, D. J. (2007). Neural correlates of enhanced working-memory performance in dissociative disorder: a functional MRI study. *Psychological Medicine*, *37*, 235–245.

Farina, B., Speranza, A. M., Dittoni, S., Gnoni, V., Trentini, C., Vergano, C. M., Liotti, G., Brunetti, R., Testani, E., & Della Marca, G. (2014). Memories of attachment hamper EEG cortical connectivity in dissociative patients. *European Archives of Psychiatry and Clinical Neuroscience*, *264*, 449–458.

Flor-Henry, P., Tomer, R., Kumpula, I., Koles, Z. J., & Yeudall, L. T. (1990). Neurophysiological and neuropsychological study of two cases of multiple personality syndrome and comparison with chronic hysteria. *International Journal of Psychophysiology*, *10*, 151–161.

Harricharan, S., Rabellino, D., Frewen, P. A., Densmore, M., Théberge, J., McKinnon, M. C., Schore, A. N., & Lanius, R. A. (2016). fMRI functional connectivity of the periaqueductal gray in PTSD and its dissociative subtype. *Brain and Behavior*, *6*(12), e00579.

Harter, S. (2015). *The Construction of the Self, Second Edition: Developmental and Sociocultural Foundations*. New York: Guilford Publications.

Hopper, A., Ciorciari, J., Johnson, G., Spensley, J., Sergejew, A., & Stough, C. (2002). EEG coherence and dissociative identity disorder: Comparing EEG coherence in DID hosts, alters, controls and acted alters. *Journal of Trauma & Dissociation*, *3*, 75–88.

Hughes, J. R., Kuhlman, D. T., Fichtner, C. G., & Gruenfeld, M. J. (1990). Brain mapping in a case of multiple personality. *Clinical EEG*, *21*, 200–209.

Irle, E., Lange, C., Sachsse, U., & Weniger, G. (2009). Further evidence that post-traumatic stress disorder but not dissociative disorders are related to amygdala and hippocampal size reduction in trauma-exposed individuals. *Acta Psychiatrica Scandinavica*, *119*, 330–331.

King, C. D., Hill, S. B., Wolff, J. D., Bigony, C. E., Winternitz, S., Ressler, K. J., Kaufman, M. L., & Lebois, L. A. M. (2020). Childhood maltreatment type and severity predict depersonalization and derealization in treatment-seeking women with post-traumatic stress disorder. *Psychiatry Research*, *292*, 113301.

Kluft, R. P. (1988). The phenomenology and treatment of extremely complex multiple personality disorder. *Dissociation*, *1*, 47–58.

Kluft, R. P. (1999). An overview of the psychotherapy of dissociative identity disorder. *American Journal of Psychotherapy*, *53*, 289–319.

Krüger, C., & Fletcher, L. (2017). Predicting a dissociative disorder from type of childhood maltreatment and abuser–abused relational tie. *Journal of Trauma & Dissociation*, *18*, 356–372.

Lanius, R. A., Bluhm, R., Lanius, U., & Pain, C. (2006). A review of neuroimaging studies in PTSD: heterogeneity of response to symptom provocation. *Journal of Psychiatric Research*, *40*, 709–729.

Lanius, R. A., Williamson, P. C., Boksman, K., Densmore, M., Gupta, M., Neufeld, R. W. J., Gati, J. S., & Menon, R. S. (2002). Brain activation during script-driven imagery induced dissociative responses in PTSD: a functional magnetic resonance imaging investigation. *Biological Psychiatry*, *52*, 305–311.

Lanius, R. A., Williamson, P. C., Densmore, M., Boksman, K., Gupta, M. A., Neufeld, R. W., Gati, J. S., & Menon, R. S. (2001). Neural correlates of traumatic memories in posttraumatic stress disorder: a functional MRI investigation. *American Journal of Psychiatry*, *158*, 1920–1922.

Lapointe, A. R., Crayton, J. W., DeVito, R., Fichtner, C. G., & Konopka, L. M. (2006). Similar or disparate brain patterns? The intra-personal EEG variability of three women with multiple personality disorder. *Clinical EEG and Neuroscience*, *37*, 235–242.

Larmore, K., Ludwig, A. M., & Cain, R. L. (1977). Multiple Personality – An Objective Case Study. *British Journal of Psychiatry*, *131*, 35–40.

Lebois, L. A. M., Li, M., Baker, J. T., Wolff, J. D., Wang, D., Lambros, A. M., Grinspoon, E., Winternitz, S., Ren, J., Gönenç, A., Gruber, S. A., Ressler, K. J., Liu, H., & Kaufman, M. L. (2021). Large-Scale Functional Brain Network Architecture Changes Associated with Trauma-Related Dissociation. *American Journal of Psychiatry*, *178*, 165–173.

Lebois, L. A. M., Seligowski, A. V., Wolff, J. D., Hill, S. B., & Ressler, K. J. (2019). Augmentation of Extinction and Inhibitory Learning in Anxiety and Trauma-Related Disorders. *Annual Review of Clinical Psychology*, *15*, 257–284. https://doi.org/10.1016/j.neuropsychologia.2018.01.008

Liotti, G. (1992). Disorganized/disoriented attachment in the etiology of the dissociative disorders. *Dissociation*, 5, 196–204.

Liotti, G. (1999). Understanding the dissociative processes: The contribution of attachment theory. *Psychoanalytic Inquiry*, *19*, 757–783.

Liotti, G. (2004). Trauma, dissociation, and disorganized attachment: Three strands of a single braid. *Psychotherapy: Theory, Research, Practice, Training*, *41*, 472–486.

Loewenstein, R. J. (2020). Firebug! Dissociative identity disorder? Malingering? Or …? An intensive case study of an arsonist. *Psychological Injury and Law*, *13*, 187–224.

Logue, M. W., van Rooij, S. J. H., Dennis, E. L., Davis, S. L., Hayes, J. P., Stevens, J. S., Densmore, M., Haswell, C. C., Ipser, J., Koch, S. B. J., Korgaonkar, M., Lebois, L. A. M., Peverill, M., Baker, J. T., Boedhoe, P. S. W., Frijling, J. L., Gruber, S. A., Harpaz-Rotem, I., Jahanshad, N., … Morey, R. A. (2018). Smaller Hippocampal Volume in Posttraumatic Stress Disorder: A Multisite ENIGMA-PGC Study: Subcortical Volumetry Results From Posttraumatic Stress Disorder Consortia. *Biological Psychiatry*, *83*, 244–253.

Lotfinia, S., Soorgi, Z., Mertens, Y., & Daniels, J. (2020). Structural and functional brain alterations in psychiatric patients with dissociative experiences: A systematic review of magnetic resonance imaging studies. *Journal of Psychiatric Research*, *128*, 5–15.

Ludwig, A. M., Brandsma, J. M., Wilbur, C. B., Bendfeldt, F., & Jameson, D. H. (1972). The objective study of a multiple personality. Or, are four heads better than one? *Archives of General Psychiatry*, *26*, 298–310.

Lyons-Ruth, K., Dutra, L., Schuder, M. R., & Bianchi, I. (2006). From infant attachment disorganization to adult dissociation: relational adaptations or traumatic experiences? *The Psychiatric Clinics of North America*, *29*, 63–86.

Main, M., & Hesse, E. (1990). Parents' unresolved traumatic experiences are related to infant disorganized attachment status: Is frightened and/or frightening parental behavior the linking mechanism? *Attachment in the Preschool Years: Theory, Research, and Intervention*, *507*, 161–182.

Main, M., & Solomon, J. (1986). Discovery of an insecure-disorganized/disoriented attachment pattern. In T. B. Brazelton (Ed.), *Affective development in infancy* (pp. 95–124). Norwood, NJ: Ablex Publishing.

Mathew, R. J., Jack, R. A., & West, W. S. (1985). Regional cerebral blood flow in a patient with multiple personality. *American Journal of Psychiatry*, *142*, 504–505.

McFadden, J. (2011). The Role of Disorganized Attachment and Insecure Environment in the Development of Pathological Dissociation and Multiple Identities. *Journal of Analytical Psychology*, *56*, 348–353.

Meloni, E. G., Venkataraman, A., Donahue, R. J., & Carlezon, W. A., Jr. (2016). Bi-directional effects of pituitary adenylate cyclase-activating polypeptide (PACAP) on fear-related behavior and c-Fos expression after fear conditioning in rats. *Psychoneuroendocrinology*, *64*, 12–21.

Menon, V. (2011). Large-scale brain networks and psychopathology: a unifying triple network model. *Trends in Cognitive Sciences*, *15*, 483–506.

Morselli, G. E. (1953). Personalita alternate e patologia affectiva [Alternate personality and affective disorders]. *Archivo di Psicologia*, *14*, 579–589.

Nicholson, A. A., Densmore, M., Frewen, P. A., Théberge, J., Neufeld, R. W., McKinnon, M. C., & Lanius, R. A. (2015). The Dissociative Subtype of Posttraumatic Stress Disorder: Unique Resting-State Functional Connectivity of Basolateral and Centromedial Amygdala Complexes. *Neuropsychopharmacology*, *40*, 2317–2326.

Ogawa, J. R., Sroufe, L. A., Weinfield, N. S., Carlson, E. A., & Egeland, B. (1997). Development and the fragmented self: longitudinal study of dissociative symptomatology in a nonclinical sample. *Development and Psychopathology*, *9*, 855–879.

Ozturk, E., & Şar, V. (2006). The "Apparently Normal" Family: A Contemporary Agent of Transgenerational Trauma and Dissociation. *Journal of Trauma Practice*, *4*, 287–303.

Packard, M. G., & Knowlton, B. J. (2002). Learning and memory functions of the Basal Ganglia. *Annual Review of Neuroscience*, *25*, 563–593.

Pierce, J. E., & Péron, J. (2020). The basal ganglia and the cerebellum in human emotion. *Social Cognitive and Affective Neuroscience*, *15*, 599–613.

Prince, M., & Peterson, F. (1908). Experiments in psycho-galvanic reactions from co-conscious (subconscious) ideas in a case of multiple personality. *Journal of Abnormal Psychology*, *3*, 114–131.

Putnam, F. W. (1984). The psychophysiologic investigation of multiple personality disorder. A review. *Psychiatric Clinics of North America*, 7, 31–39.

Putnam, F. W. (1997). *Dissociation in Children and Adolescents: A Developmental Perspective*. New York: Guilford Press.

Putnam, F. W. (2016). *The way we are: How states of mind influence our identities, personality and potential for change*. New York: International Psychoanalytic Books.

Putnam, F. W., Zahn, T. P., & Post, R. M. (1990). Differential autonomic nervous system activity in multiple personality disorder. *Psychiatry Research*, *31*, 251–260.

Raichle, M. E. (2015). The brain's default mode network. *Annual Review of Neuroscience*, *38*, 433–447.

Reinders, A. A. T. S., Marquand, A. F., Schlumpf, Y. R., Chalavi, S., Vissia, E. M., Nijenhuis, E. R. S., Dazzan, P., Jäncke, L., & Veltman, D. J. (2018). Aiding the diagnosis of dissociative identity disorder: pattern recognition study of brain biomarkers. *British Journal of Psychiatry*, *215*, 1–9.

Reinders, A. A. T. S., Nijenhuis, E. R. S., Paans, A. M. J., Korf, J., Willemsen, A. T. M., & den Boer, J. A. (2003). One brain, two selves. *NeuroImage*, *20*, 2119–2125.

Reinders, A. A. T. S., Nijenhuis, E. R. S., Quak, J., Korf, J., Haaksma, J., Paans, A. M. J., Willemsen, A. T. M., & den Boer, J. A. (2006). Psychobiological Characteristics of Dissociative Identity Disorder: A Symptom Provocation Study. *Biological Psychiatry*, *60*, 730–740.

Reinders, A. A. T. S., Willemsen, A. T. M., den Boer, J. A., Vos, H. P. J., Veltman, D. J., & Loewenstein, R. J. (2014). Opposite brain emotion-regulation patterns in identity states of dissociative identity disorder: a PET study and neurobiological model. *Psychiatry Research*, *223*, 236–243.

Reinders, A. A. T. S., Willemsen, A. T. M., Vissia, E. M., Vos, H. P. J., den Boer, J. A., & Nijenhuis, E. R. S. (2016). The Psychobiology of Authentic and Simulated Dissociative Personality States: The Full Monty. *Journal of Nervous and Mental Disease*, *204*, 445–457.

Reinders, A. A. T. S., Willemsen, A. T. M., Vos, H. P. J., den Boer, J. A., & Nijenhuis, E. R. S. (2012). Fact or factitious? A psychobiological study of authentic and simulated dissociative identity states. *PloS One*, 7(6), e39279.

Ross, C. A. (1997). Dissociative Identity Disorder: Diagnosis, Clinical Features, and Treatment of Multiple Personality. New York: Wiley.

Ross, C. A., & Ness, L. (2010). Symptom patterns in dissociative identity disorder patients and the general population. *Journal of Trauma & Dissociation*, *11*, 458–468.

Ross, C. A., Norton, G. R., & Wozney, K. (1989). Multiple personality disorder: an analysis of 236 cases. *Canadian Journal of Psychiatry*, *34*, 413–418.

Roydeva, M. I., & Reinders, A. A. T. S. (2020). Biomarkers of Pathological Dissociation: A Systematic Review. *Neuroscience and Biobehavioral Reviews*, *123*, 120–202.

Şar, V., Dorahy, M. J., & Krüger, C. (2017). Revisiting the etiological aspects of dissociative identity disorder: a biopsychosocial perspective. *Psychology Research and Behavior Management*, *10*, 137.

Şar, V., Unal, S. N., Kiziltan, E., Kundakci, T., & Ozturk, E. (2001). HMPAO SPECT Study of Regional Cerebral Blood Flow in Dissociative Identity Disorder. *Journal of Trauma & Dissociation*, *2*, 5–25.

Şar, V., Unal, S. N., & Ozturk, E. (2007). Frontal and occipital perfusion changes in dissociative identity disorder. *Psychiatry Research*, *156*, 217–223.

Savoy, R. L., Frederick, B. B., Keuroghlian, A. S., & Wolk, P. C. (2012). Voluntary switching between identities in dissociative identity disorder: A functional MRI case study. *Cognitive Neuroscience*, *3*, 112–119.

Schlumpf, Y. R., Nijenhuis, E. R. S., Chalavi, S., Weder, E. V., Zimmermann, E., Luechinger, R., La Marca, R., Reinders, A. A. T. S., & Jäncke, L. (2013). Dissociative part-dependent biopsychosocial reactions to backward masked angry and neutral faces: An fMRI study of dissociative identity disorder. *NeuroImage. Clinical*, *3*, 54–64.

Schlumpf, Y. R., Reinders, A. A. T. S., Nijenhuis, E. R. S., Luechinger, R., van Osch, M. J. P., & Jäncke, L. (2014). Dissociative part-dependent resting-state activity in dissociative identity disorder: a controlled FMRI perfusion study. *PloS One*, *9*, e98795.

Spreng, R. N., Stevens, W. D., Chamberlain, J. P., Gilmore, A. W., & Schacter, D. L. (2010). Default network activity, coupled with the frontoparietal control network, supports goal-directed cognition. *NeuroImage*, *53*, 303–317.

Squire, L. R., & Zola-Morgan, S. (1991). The medial temporal lobe memory system. *Science*, *253*, 1380–1386.

Steinberg, M. (1994). *Interviewer's Guide to the Structured Clinical Interview for DSM-IV Dissociative Disorders (SCID-D)*. Washington, DC: American Psychiatric Press.

Stein, M. B., Koverola, C., Hanna, C., Torchia, M. G., & McClarty, B. (1997). Hippocampal volume in women victimized by childhood sexual abuse. *Psychological Medicine*, *27*, 951–959.

Stocco, A., Lebiere, C., & Anderson, J. R. (2010). Conditional routing of information to the cortex: a model of the basal ganglia's role in cognitive coordination. *Psychological Review*, *117*, 541–574.

Sullivan, H. S. (2013). *The interpersonal theory of psychiatry*. New York: Routledge.

Teicher, M. H., Samson, J. A., Polcari, A., & McGreenery, C. E. (2006). Sticks, stones, and hurtful words: relative effects of various forms of childhood maltreatment. *American Journal of Psychiatry*, *163*, 993–1000.

Thigpen, C. H., & Cleckley, H. (1954). A case of multiple personality. *Journal of Abnormal Psychology*, *49*, 135–151.

Tsai, G. E., Condie, D., Wu, M. T., & Chang, I. W. (1999). Functional magnetic resonance imaging of personality switches in a woman with dissociative identity disorder. *Harvard Review of Psychiatry*, 7, 119–122.

Vermetten, E., Schmahl, C., Lindner, S., Loewenstein, R. J., & Bremner, J. D. (2006). Hippocampal and amygdalar volumes in dissociative identity disorder. *American Journal of Psychiatry*, *163*, 630–636.

Vesuna, S., Kauvar, I. V., Richman, E., Gore, F., Oskotsky, T., Sava-Segal, C., Luo, L., Malenka, R. C., Henderson, J. M., Nuyujukian, P., Parvizi, J., & Deisseroth, K. (2020). Deep posteromedial cortical rhythm in dissociation. *Nature*, *586*(7827), 87–94.

Weniger, G., Lange, C., Sachsse, U., & Irle, E. (2008). Amygdala and hippocampal volumes and cognition in adult survivors of childhood abuse with dissociative disorders. *Acta Psychiatrica Scandinavica*, *118*, 281–290.

Weniger, G., Siemerkus, J., Barke, A., Lange, C., Ruhleder, M., Sachsse, U., Schmidt-Samoa, C., Dechent, P., & Irle, E. (2013). Egocentric virtual maze learning in adult survivors of childhood abuse with dissociative disorders: evidence from functional magnetic resonance imaging. *Psychiatry Research*, *212*, 116–124.

Williams, C. L., Haines, J., & Sale, I. M. (2003). Psychophysiological and Psychological Correlates of Dissociation in a Case of Dissociative Identity Disorder. *Journal of Trauma & Dissociation*, *4*, 101–118.

Wolk, P. C., Savoy, R. L., & Frederick, B. B. (2012). The Neural Correlates of Vertical Splitting in a Single Case Study. *Neuropsychoanalysis*, *14*, 157–163.