The psychobiology of authentic and simulated dissociative personality states: The Full Monty

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Running title

Psychobiology of DID: The Full Monty

Conflict of interest

The authors declare no conflicts of interest.

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Abstract

The etiology of dissociative identity disorder (DID) remains a topic of debate. Proponents of the fantasy model and the trauma model of DID have both called for more empirical research. To this end, the current study presents new and extended data-analyses of a previously published $H_2{}^{15}O$ positron emission tomography imaging study. This study included twenty-nine subjects: 11 patients with DID, 10 high and 8 low fantasy prone DID simulating mentally healthy controls. All subjects underwent an autobiographical memory script-driven (neutral and trauma-related) imagery paradigm in two (simulated) dissociative personality states (neutral and trauma-related). Psychobiological and psychophysiological data were obtained. Results of the new post-hoc tests on the psychophysiological responses support the trauma model. New results of the brain imaging data did not support the fantasy model. This study extends previously published results by offering important new supporting data for the trauma model of DID.

Keywords

Dissociative identity disorder, neuroimaging, trauma model, fantasy model, fantasy proneness

Introduction

Despite its inclusion in the latest version of the Diagnostic Manual of Mental Disorders (DSM-5 (APA, 2013)) and a comprehensive review (Dalenberg et al., 2012) the validity of dissociative identity disorder (DID) continues to be disputed (Dalenberg et al., 2014; Lynn et al., 2014). The current Journal has relatively recently been a platform of debate between proponents of the fantasy model and trauma model regarding two original publications (Boysen and VanBergen, 2013; Paris, 2012), which were extensively commented on ((Boysen and Vanbergen, 2013; Brand et al., 2013a; Dell, 2013; Sar et al., 2013) and (Brand et al., 2013b; Martínez-Taboas et al., 2013; McHugh, 2013; Paris, 2013; Ross, 2013)).

Only one neurobiological study (by the current authors (Reinders et al., 2012)) has tested the position that DID is related to fantasy proneness (Gleaves, 1996; Lilienfeld et al., 1999; Lynn et al., 2012, 2014) by including DID patients and both high and low fantasy prone DID simulating healthy controls. The focus of that study was to test whether fantasy proneness could explain the brain imaging results from a within patient study (Reinders et al., 2006). Recently supported and extended by other neuroimaging studies in DID patients (Chalavi et al., 2015a, 2015b; Schlumpf et al., 2013, 2014), the findings were at odds with the claim that the complex phenomenology and psychobiology of DID are due to fantasy-proneness, suggestion, and motivated role-playing. The type and number of statistical tests in Reinders et al. (2012) were limited to the study's hypotheses and important additional analyses remained unexplored or unreported. The current manuscript reports the *Full Monty* by presenting results of new additional exploratory data-analyses.

Types of personality states

According to the DSM-5, DID is characterized by, among others, the presence of two or more distinct dissociative `personality states'. This terminology is different from previous DSM versions where the term `identity states' was used. In line with the latter terminology we previously have used the term `dissociative identity states' and different prototypes of dissociative identity states were indicated as *neutral identity states* (NIS) and *trauma-related identity states* (TIS) (Reinders et al., 2006, 2012, 2014; Reinders and Willemsen, 2014). However, our first publication used the terminology `neutral personality state' (NPS) and `trauma-related personality state' (TPS) (Reinders, et al., 2003), which is in line with the current DSM-5 terminology and will be used again in the remainder of this manuscript. These various indicators were derived from the terms `apparently normal part of the

personality (ANP)' and `emotional part of the personality (EP)' respectively, which are formulated in the theory of structural dissociation of the personality (TSDP) (Nijenhuis et al. 2002; Van der Hart et al. 2006) and used in recent publications (Schlumpf et al., 2013, 2014). This theory defines dissociation as a division of personality into different types of subsystems, each with their own first-person perspective, that is, their own point of view as to who they are, what the world is like, and how they relate to that world (Nijenhuis and Van der Hart, 2011; Nijenhuis, 2015).

As NPS DID patients concentrate on functioning in daily life, commonly try to hide their pathology, and avoid traumatic memories when they can. As a result, NPS has not or not sufficiently integrated these memories (Reinders, 2003). In contrast, TPS does have conscious access to these memories, recalls them as personal experiences and is bodily and emotionally affected by them. As TPS the patients are fixated in traumatic memories and engage in defensive actions such as freeze and flight, when they feel threatened (Nijenhuis et al. 2002, 2004), thereby activating fast subcortical response routes in the brain (LeDoux, 2000; Reinders et al., 2006). Patients, as TPS can either engage in active kinds of physical defence (e.g., freeze, flight, fight) indicating dominance of the sympathetic nervous system, or they can engage in death feigning primarily mediated by the dorsal vagal branch of the parasympathetic nervous system (Nijenhuis and Den Boer, 2009). Of note, alternative models exist, such as the orbitofrontal hypothesis by Forrest (Forrest, 2001). This is a neurodevelopmental model underlining deficient functionality of the orbitofrontal region in the brain. Within this model the orbitofrontal lobe is hypothesized to be affected by early trauma (Dorahy et al., 2014). This model is in line with work from Schore (Schore, 2003) and is furthermore supported by two controlled brain imaging studies that found bilateral frontal perfusion differences between DID patients and controls (Sar et al., 2001; Sar et al., 2007).

Review of perfusion and functional magnetic resonance imaging studies in DID

Imaging neuroscience has been around for more than 20 years and is by now the predominant technique in behaviour and cognitive neuroscience (Friston, 2009). However, very few neuroimaging studies have been conducted in patients with DID (Dalenberg et al., 2012; Dorahy et al., 2014; Reinders, 2008, Reinders and Willemsen, 2014). This is despite the fact that imaging neuroscience has been around for more than 20 years and is by now the predominant technique in behaviour and cognitive neuroscience

Resting state studies: The first functional brain imaging study in a single patient with DID was a positron emission tomography (PET) scan of the resting brain state (Mathew et al., 1985). The study included three mentally healthy controls and revealed hyperperfusion in the right temporal cortex of the DID patient. Four studies applied the low spatial resolution imaging technique single photon emission computed tomography (SPECT) of which two were uncontrolled case studies (Saxe et al., 1992; Sheehan et al., 2006). These case studies also found the involvement of the temporal lobe of the brain in DID. Sar et al. (Sar et al., 2001, 2007) included the largest sample of 21 DID patients in studies into DID using brain imaging techniques to date. These two studies consistently found bilateral orbitofrontal hypoperfusion differences between patients and controls.

Using arterial spin labelling perfusion MRI (ASL) two major prototypes of dissociative parts, that is an NPS and a hyperaroused TPS, were examined (Schlumpf et al., 2014). Compared to TPS, NPS showed elevated perfusion in bilateral thalamus. Compared to NPS, TPS had increased perfusion in the dorsomedial prefrontal cortex, primary somatosensory cortex, and motor-related areas. Perfusion patterns for simulated NPS and TPS were different. Fitting their reported role-play strategies, the actors activated brain structures involved in visual mental imagery and empathizing feelings.

Voluntary 'switching' studies: Two uncontrolled fMRI case studies examined brain activation patterns associated with voluntary switching between different dissociative personality states (Savoy et al., 2012; Tsai et al., 1999). Savoy et al. found involvement of the dorsolateral prefrontal cortex, the anterior prefrontal cortex, and orbitofrontal cortex, as well as bilateral activation in the nucleus accumbens, an area in the ventral striatum. Tsai et al. did not find involvement of the prefrontal cortical areas associated with voluntary switching, but observed brain activity in hippocampal areas, as well as the parahippocampus, medial temporal structures, substantia nigra, and the global pallidus, which is a part of the dorsal striatum (Tsai et al., 1999). On the other hand Sar et al. (Sar et al., 2001) found that bilateral orbitofrontal hypoperfusion during resting state is independent of personality state. This finding suggests that orbitofrontal hypoperfusion is a biomarker for DID.

Task-related brain activation studies: Using functional magnetic resonance imaging (fMRI), a relatively high temporal and spatial resolution brain imaging technique, brain activation of 16 dissociative disorder (DD) patients and 16 mentally healthy controls was studied during a working-memory task (Elzinga et al., 2007). DD patients outperformed controls despite feeling more fearful and less concentrated while activating the left anterior

prefrontal cortex, left dorsolateral prefrontal cortex, and the left parietal cortex more than controls. The prefrontal, but not the parietal activation was independent of task-difficulty.

During an fMRI task neutral and angry faces were subliminally presented to 11 individuals with DID and 15 DID-simulating mentally healthy actors and reaction times and changes in brain activation were investigated (Schlumpf et al., 2013). Abnormal reaction times were found for TPS, but not for NPS, and TPS activated different brain areas including in the parahippocampal gyrus, the brainstem, face-sensitive regions, and motor-related areas. The actors activated different neural patterns as compared to patients.

A multi-subject PET study reported that NIS and TIS are associated with different brain activation patterns when confronted with trauma-related cues (Reinders et al., 2003, 2006). They reported the involvement of mainly the cortical multimodal posterior association areas (PAA), the subcortical amygdala and subparts of the dorsal striatum (i.e., the caudate and putamen) in the psychopathology of DID. These findings were not linked to fantasy proneness (Reinders et al., 2012). Neither high nor low fantasy prone mentally healthy women instructed and motivated to simulate the involved dissociative personality states enacted the psychophysiological and neural activation patterns of the authentic dissociative personality states.

In sum: Functional differences in DID have been reported throughout the brain dependent on a variety of tasks: in the temporal (Mathew et al., 1985; Sar et al., 2001; Saxe et al., 1992; Sheehan et al., 2006; Tsai et al., 1999), frontal (Elzinga et al., 2007; Sar et al., 2001, 2007; Savoy et al., 2012) and occipital (Sar et al., 2007) cortices, the amygdala and dorsal striatum (Reinders et al., 2006, 2012), nucleus accumbens (Savoy et al., 2012), and hippocampal and pallidum structures (Tsai et al., 1999; Schlumpf et al., 2014). Hence, a convergence of findings to disseminate neurobiological markers for the psychopathology of DID is still needed.

A neurobiological model for DID: Recently, a neurobiological model for DID has been formulated (Reinders et al., 2014) combining neuroimaging research on the dissociative subtype of posttraumatic stress disorder (PTSD) (Lanius et al., 2010) and DID. This model proposes that the NPS in DID activates prefrontal and cingulate areas as well as the posterior association areas and parahippocampal gyri when confronted with trauma related information. The prefrontal and cingulate are core in the overmodulation of emotion (Lanius et al., 2010), whereas the posterior association areas and (para)hippocampal regions are thought to be involved in the suppression of unwanted autobiographical memories (Anderson et al., 2004). It further proposes that the TPS in DID activates the

insula and amygdala, as well as the dorsal striatum, while reacting to trauma-related stimuli/material. The insula and amygdala are activated during undermodulation of emotion (Lanius et al., 2010), whereas the dorsal striatum has been proposed to play an important role in the switching between identity states (Tsai et al., 1999), as well as in maintaining state stability of a dissociative identity state (Reinders et al., 2006; Reinders et al., 2012; Schlumpf et al., 2013).

Trauma and fantasy models of DID

Supporters of the opposed trauma and fantasy models (Dalenberg et al., 2012) of DID are engaged in a debate regarding the validity of DID as a mental disorder, and its causes (i.e., traumatization or fantasy proneness, suggestibility, suggestion, and simulation) (Bremner, 2010; Coons, 2005; Fraser, 2005; Giesbrecht et al., 2008; Giesbrecht et al., 2010; Gleaves, 1996; Piper and Merskey, 2004a, 2004b; Sar, 2005; Spanos, 1994). The fantasy model of DID entails the idea that this disorder can be easily and readily created in motivated suggestible individuals and that few suggestions suffice to generate the symptoms of DID (Spanos, 1994). This model (Giesbrecht et al., 2008; Merckelbach and Muris, 2001; Merckelbach et al., 2002; Piper and Merskey, 2004a, 2004b; Pope et al., 2006) is also referred to as the sociocognitive model of DID (Lilienfeld et al., 1999; Spanos, 1994), or non-trauma-related model (Reinders et al., 2012) and involves the idea that DID is a simulation caused by high suggestibility and/or fantasy proneness (Giesbrecht and Merckelbach, 2006; Giesbrecht et al., 2007; Merckelbach et al., 2000; Merckelbach and van de Ven, 2001), suggestive psychotherapy and other suggestive sociocultural influences (e.g., the media and/or the church (Spanos, 1994; Lilienfeld et al., 1999)). Although fantasy proneness and suggestibility refer to different concepts, they are highly correlated (Braffman and Kirsch, 1999; Levin and Spei, 2004; Merckelbach and van de Ven, 2001). Of note: people who argue against the DID trauma model do not solely talk about fantasy proneness, but also suggest the possibility of mild cognitive impairment (Giesbrecht et al., 2008) or sleep deprivation (van Heugten-van der Kloet et al., 2014) as an alternative explanation. To date, proponents of the fantasy model of DID have not studied individuals with DID using brain imaging techniques.

The trauma model (Dalenberg et al., 2012; Reinders et al., 2012) entails that DID is related to a combination of factors that include chronic emotional neglect as well as emotional, physical, and/or sexual abuse from early childhood, insufficient integrative capacity, attachment disorder, and lack of affect-regulation by caretakers (Dell and O'Neil,

2010; Gleaves, 1996; Spiegel, 2006; Van der Hart et al., 2006). In this view DID is thought to be at the far end of the spectrum of trauma-related psychiatric disorders (Chalavi et al., 2015a, 2015b). Proponents of the trauma model acknowledge that: some features of dissociative personality states can be influenced by sociocultural factors, that false positive cases of DID have evolved in a treatment setting, and that some psychiatric patients imitate DID (Draijer and Boon, 1999). They also note that there are differences between authentic and imitated DID (Draijer and Boon, 1999) and that there is no evidence that DID can (sub-)consciously be created by sociocultural factors (Gleaves, 1996). Furthermore, even if DID symptoms can be created iatrogenically or can be enacted this does not mean that genuine trauma-related DID does not exist (Elzinga et al., 1998).

The Full Monty

The current study presents new and extended data-analyses on the basis of previously published data (Reinders et al., 2012), which includes *within* NPS and *between personality state* (TPS versus NPS) comparisons and *conjunction analyses.* Brain activation patterns of DID patients and high and low fantasy prone DID simulating controls are compared. On basis of the Trauma model's predictions and the newly delineated neurobiological models of dissociation (Lanius et al., 2010; Reinders et al., 2014) we hypothesize: i) more brain activation in the prefrontal regions and the anterior cingulate in response to the trauma-related text as compared to neutral text for NPS to establish overmodulation of emotions, ii) activation in the dorsal striatum for maintaining state stability of a dissociative personality state (Reinders et al., 2014) for the comparison of NPS' and TPS' reactivity to the neutral text, iii) differences between high (CH) and low (CL) fantasy prone DID simulating subjects do not involve brain regions from the neurobiological models of dissociation, and (iv) no or little overlap between brain activation patterns for DID and CH and/or CL for the conjunction analyses (Friston et al., 1999; Price and Friston, 1997).

Finally, authors of a relatively recent paper reviewing simulation protocols in studies involving subjects with (simulated) DID remarked that for the psychophysiological data in Reinders et al. (2012) "the authors do not report specific post hoc test results" (Boysen and VanBergen, 2014) (p52). In reply to this we also report the results of these specific post hoc test on the psychophysiological data.

In sum, the current study aims to inform on the neurobiology of DID concerning the differential processing of trauma and neutral text within the NPS, the differential processing of the neutral text between NPS and TPS, and concerning overlap in brain

activation between DID patients and DID simulating controls.

Methods

Participants

Twenty-nine subjects participated in the PET study, which was approved by the Medical Ethical Committee of the University Medical Center Groningen: 11 patients with dissociative identity disorder (DID), 10 high fantasy prone DID simulating mentally healthy controls (CH), and 8 low fantasy prone DID simulating mentally healthy controls (CL). Controls were carefully matched for gender (all female) and age. Differences in age were not significant (DID vs. CH: $F(1,18)=0,499$ $p = 0.489$, n.s. and DID vs. CL: $F(1,16)=0.153$; $p = 0.701$, n.s.). A detailed description of the mentally healthy controls included in this study and the DID enactment procedure can be found in a previous publication (Reinders et al., 2012; Reinders and Willemsen, 2014). In short, the controls were recruited by local newspaper advertisements, did not report potentially traumatizing events such as physical abuse and emotional neglect and completed the Traumatic Experience Checklist (TEC (Nijenhuis et al., 2002)), Somatoform Dissociation Questionnaire (SDQ-20 (Nijenhuis et al., 1996)), and the Creative Experiences Questionnaire (CEQ) (Merckelbach et al., 2001) which measures fantasy proneness. A CEQ cut-off for high fantasy proneness of 10 was used, which the developers of the CEQ recommended for the current sample (personal communication by email). This resulted in two groups of controls: a high fantasy prone group and a low fantasy prone group (see Table 1). The controls received written and oral information on NPS and TPS and were instructed to simulate these different dissociative personality states. Controls were asked to provide their most painful memory to serve as an analogue for the patients' personal trauma memories, as well as a neutral personal episodic memory. They were subsequently instructed how to write the autobiographical analogue “neutral” and "trauma" memory scripts. For the experiment they had to train themselves in being an NPS who is unresponsive or under-responsive to the painful experience, and in being a TPS, a dissociative personality state in which they are stuck in and tend to recurrently re-experience the painful memory.

A detailed description of the DID patients can be found elsewhere (Reinders et al., 2003, 2006). In short, 11 patients (all female) participated (i) whose treatment had progressed to include therapeutic exposure to trauma-related memories, (ii) who met criteria for DID, as operationalized in the Structured Clinical Interview for DSM-IV Dissociative Disorders (SCID-D (Steinberg, 1993)), and (iii) who had at least one TPS and one NPS that they could activate on demand in an experimental setting and (iv) whose selected TPS had displayed signs of sympathetic nervous system dominance under

perceived threat in clinical situations. H.V. or the patient's therapist structurally evaluated if the intended NPS or TPS had been present during the experimental condition. This was done by debriefing the presence of the dissociative personality state under investigation and by checking potential interference among personality states during the execution of the experimental tasks.

Image acquisition and data processing

Cerebral blood flow PET (Siemens/CTI ECAT HR+) data, autonomic (systolic and diastolic blood pressure, discrete heart rate and heart rate variability (HRV)) and subjective (controls' subjective sensorimotor and emotional experiences) reactions were obtained (see for details, statistical analyses and results: (Reinders 2003, 2006, 2012) (main paper and supplementary materials, S2). DID patients, as well as high and low fantasy prone controls were studied in the two different types of personality states during a memory script (MS) driven imagery paradigm. Four conditions were obtained in a repeated measures design: NPSn, NPSt, TPSn, TPSt, where the last minor character (n or t) denotes the content of the memory script (MS: neutral or trauma-related). Data acquisition, reconstruction, attenuation correction, spatial transformation, spatial smoothing (isotropic Gaussian kernel of 12 mm) were performed as usual (Reinders et al., 2012).

Data analyses

The brain imaging data of the three groups was pre-processed and statistically analyzed in SPM5 (www.fil.ion.ucl.ac.uk/spm) in a three-by-two-by-two factorial design, which allows for the assessment of within and between personality state effects within and between the three groups. The subjective reactions and the autonomic reactions were included as group specific covariates in the general linear model (GLM: three factor main effects (subject, condition and group), four conditions and the group by condition interaction) of SPM5 after principal component (PC) analysis (Reinders et al., 2003, 2006, 2012). Global cerebral blood flow (CBF) was included as a nuisance covariate (AnCova by subject).

Comparisons of interest included *within personality state effects and between personality state effects*. *Within* personality state effects refer to different patterns of brain activity associated with reactions to the trauma-related and neutral text within the trauma-related or neutral personality state in DID, CH, and CL (e.g., DID(TPSt-TPSn)-CL(TPSt-TPSn)). *Between* personality state effects refer to different patterns of brain activity associated with reactions to the trauma-related text and the neutral text, respectively,

between different types of personality states in DID, CH, and CL (e.g., DID(TPSt-NPSt)-CH(TPSt-NPSt)). Conjunction analyses (Friston et al., 1999; Price and Friston, 1997) were conducted on both the between and within personality state effects between DID and CH, DID and CL and DID, CH and CL.

Conjunction analyses test for conjoint activation patterns between conditions and/or groups therewith allowing for the assessment of overlap in brain activation between patients and controls. The current study comprises three between group comparisons, namely DID vs. CH, DID vs. CL, and CH vs. CL. Hence, conjoint brain activation between DID and CH, DID and CL, and DID and CL and CH can be investigated. Some overlap of brain activity between groups is expected as experimental settings were very similar.To investigate whether conjoint activation between the DID group and the controls groups exists statistical parametric maps were thresholded at a whole brain corrected threshold of $p < 0.05$. The statistical parametric maps obtained from the comparisons between DID and controls were thresholded using an uncorrected threshold of $p < 0.001$ (Reinders et al., 2006) and explored for *a priori* hypothesized brain areas. Multiple comparisons correction was performed, using false discovery rate statistics (Genovese et al., 2002), for whole brain and for the *a priori* regions of interest (ROI). In the latter case a small volume correction was applied using a sphere with radius of 9 mm (Reinders et al., 2005). *A priori* hypothesized regions of interest (ROI) were areas reported in Reinders et al. (2006) (note that most of these are also reported in Reinders et al. (2012) in the '*Within group*: DID only' columns) and areas included in the neurobiological model for DID (Reinders et al., 2014), independent of lateralisation (hence, both hemispheres were explored). Activation localization was performed as usual (Reinders et al., 2012). Only clusters larger than eight voxels are reported taking into account the spatial resolution of the PET camera. Only the first peak voxel of a cluster is reported (note that this differs from Reinders et al., 2012, where multiple peak voxels of a cluster were investigated). Brain regions and Brodmann areas (BA) were defined using both the Talairach atlas (Talairach and Tournoux, 1988) and Deamon (Lancaster et al., 2000). Activations in sulci were defined using Brain Tutor (www.brainvoyager.com).

For the psychophysiological data the *F* and *p* values are reported as well as the mean and standard deviation. Bonferroni correction was applied to correct for the number of tests per psychophysiological measure. Values with a $p < 0.0042$ were reported as significant after the correction for multiple comparisons, results uncorrected for multiple comparisons are reported with $p < 0.05$, and trends are reported for values $0.05 < p < 0.10$

uncorrected for multiple comparisons.

Results

Within personality state effects

Different neural reactivity to the neutral and trauma-related text within both TPS (top part) and NPS (bottom part) are listed in Table 2. Results of the psychophysiological measures are listed in Table 3.

In contrast to the high fantasy prone controls, the NPS of DID activated the bilateral superior frontal gyrus while listening to the trauma-related text as compared to listening to the neutral text (see Figure 1A). Comparing listening to the trauma-related text and listening to the neutral text, there were trends for NPS of DID to have higher heart rate and more diastolic blood pressure than either CH or CL. These comparisons did not yield differences between these groups for HRV or systolic blood pressure.

When comparing the NPS of CH to the NPS of CL only right hippocampus de-activation was found for CH while listening to the trauma-related text as compared to listening to the neutral text (see Figure 1B). None of these groups' psychophysiological reactions were significantly different for this comparison.

Between personality states effects

Differences in neural responses in relation to text effects between TPS and NPS are given in Table 4 (trauma-related text effects at the top, neutral text effects at the bottom). Results of the psychophysiological measures are listed in Table 5.

DID as compared to CH and DID as compared to CL activated the left and bilateral caudate nucleus respectively in response to the neutral text in the TPS versus NPS (see Figure 1C). None of the post-hoc tests on the psychophysiological measures reached significance.

The left amygdala and cerebellum were activated when comparing the processing of the trauma-related text in TPS versus NPS in CH to the CL group, which were also found when comparing the DID to the CH group. Of the psychophysiological measures only the systolic blood pressure was found to be significantly higher in the CH as compared to the CL group when processing the trauma-related text in the TPS.

Conjunction analyses

The conjunction analyses are presented in Table 6. There was no significant overlap found in brain activation patterns between the DID group and CH for both the within and between personality state comparisons. When adding the CL group we found conjoint activation in

the three groups in the right primary auditory cortex, the right frontal and bilateral temporal regions for the within NPS comparison only. No conjoint (de-)activation was found for the within TPS comparisons or the between personality states comparisons.

Conjoint de-activation for processing of the trauma-related text between the DID group and CL group was found for the within NPS comparison in the bilateral temporal gyrus and the right occipitotemporal gyrus. No conjoint (de-)activation was found for the within TPS comparisons. Conjoint activation for the between personality state comparisons of the processing of the trauma-related text were found in the left orbitofrontal, temporal gyrus, amygdala and cerebellum, and right occipitotemporal gyrus. A conjoint de-activation was found in the right precentral gyrus.

Discussion

Proponents of the trauma and fantasy models of dissociative identity disorder (DID) have called for more neurobiological data on this disorder. Conducted in this light, the present study provides extended results from our PET study involving patients with DID and DID simulating high and low fantasy prone mentally healthy controls (Reinders et al., 2012; Reinders and Willemsen, 2014). New results were found, such as bilateral activation of the superior frontal gyrus within the neutral personality state (NPS) of the DID patients in response to the trauma-related text as compared to the high fantasy prone controls, caudate nucleus activation in the TPS as compared to NPS when processing the neutral text in DID patients as compared to high or low fantasy prone controls, and hippocampal activation differences between the DID simulating high and low fantasy prone mentally healthy control groups. The results of the new conjunction analyses confirm our previous findings that DID is not due to high levels of fantasy proneness. Furthermore, in response to requests by Boysen and Vanbergen (2013, p. 52), we performed post-hoc tests of the original psychophysiological data and found that for most measures the DID patients have significantly higher scores as compared to high or low fantasy prone DID simulating mentally healthy controls. Neither high nor low fantasy prone DID simulating mentally healthy controls were able to simulate this psychophysiological hyperarousal, which is inconsistent with the fantasy model of DID. The inability of simulators to imitate DID on physiological measures, regardless of the level of suggestibility, refutes the Fantasy Model's propositions.

Our most important finding is consistent with our neurobiological model that NPS engages prefrontal regions when listening to the trauma-related text as compared to listening to the neutral text. New research concerning trauma and dissociation (Lanius et al., 2010, 2012; Reinders et al., 2014) allowed us to perform a region of interest analysis, which revealed bilateral activation of the superior frontal gyrus in the comparison of DID patients to high fantasy prone controls (CH). This activation is hypothesized in the neurobiological model for dissociative PTSD (Lanius et al., 2010) and DID (Reinders et al., 2014). Hyperactivation of the superior frontal gyrus suppresses the sympathetic nervous system, which in turn leads to hyporesponsiveness of the psychophysiological system. This is evidenced by the lack of significant results in the psychophysiological data (Table 3). Interestingly, the activation of the superior frontal gyrus only differs between DID and CH and therefore this area seems to be similarly activated for DID and low fantasy prone controls (CL). For this finding we have three possible explanations: 1) We included more

high CH than CL subjects, so that the absence of a significantly different effect might be due to limited statistical power. This idea is supported by a recent study (Reinders et al., 2014) which compared the DID patients to a large set of control subjects (fantasy prone independent) and which reported bilateral superior, middle and medial frontal gyrus activations. It seems that more statistical power confirms the involvement of the frontal brain regions in dissociation; 2) A trait characteristic of subjects with low fantasy proneness might be less emotional reactivity; 3) CL can simulate the emotion undermodulation of DID. However, if these latter two features of CL would apply, then a difference between the two control groups should have been found in the neurobiological and psychophysiological data. As neither was the case, future studies should investigate the fantasy prone dependent activation of bilateral superior frontal regions.

Results of the within TPS comparison of brain reactivity on the differential processing of trauma-related and neutral MS and between personality state dependent processing of the trauma-related text have been discussed before (Reinders et al., 2012). The findings were only reported here to inform on the brain activation patterns entering the conjunction analyses. Most of the (de-)activated brain regions were independent of fantasy proneness as they were found in both the DID vs. CH and the DID vs. CL comparisons. Because brain activation in DID is independent of fantasy proneness these results do not support the fantasy model of DID.

Our model proposes that the dorsal striatum, which includes the caudate nucleus, plays an important role in maintaining state stability of a dissociative personality state (Reinders et al., 2014). In the *between personality states* comparison we found caudate activation in TPS as compared to NPS in DID as compared to CH and CL during the processing of the neutral text. This finding is independent of emotional reactivity in TPS because no significant differences were found for either of the subjective or the autonomic measures. We therefore propose that the caudate nucleus plays an important role in maintaining state stability of a dissociative personality state. A recent neurostructural study (Chalavi et al., 2015) reported a positive correlation between dorsal striatal volume and clinical measures of dissociation, which also indicates the involvement of the dorsal striatum in the psychopathology of DID.

As a third new line of analyses we compared both control groups to each other to inform on the neural correlates of fantasy proneness. We found the right hippocampus and the left amygdala and cerebellum. Of these findings the hippocampus is the most interesting because when mentally healthy individuals recollect autobiographical

experiences, the hippocampus becomes involved (Rugg and Vilberg, 2013). Our neurobiological model for DID proposes that acute stress can be associated with a shift from hippocampal involvement to caudate nucleus involvement (Reinders et al., 2014). Thus, acute stress is linked with a caudate nucleus-dependent stimulus-response at the expense of hippocampal dependent spatial learning and memory (Schwabe et al., 2008; White, 2009). The current analysis did show differential hippocampal activation between the control groups, but not caudate activation. Bilateral caudate nucleus activation was found for DID patients who listened to the trauma-memory scripts as TPS when compared to CH or CL. These new findings support our model that the caudate and not hippocampus plays an important role in trauma-related memory retrieval in DID. Furthermore, we found caudate activation in TPS as compared to NPS in DID as compared to CH and CL during the processing of the neutral text and proposed a role for the caudate nucleus in maintaining state stability of a dissociative personality state. This dual involvement of the caudate nucleus in the neurobiology DID needs to be further studied in future research.

Testing for commonalities in brain activation between DID and CH did not reveal any overlap in brain activation patterns for both the within and between personality state comparisons, which opposes the fantasy model. In our previous paper (Reinders et al., 2012) we proposed that CL simulated DID slightly better on a neural level than CH. The new conjunction analyses confirm this suggestion because the conjunction analyses between DID and CL revealed some conjointly activated brain regions. It seems that fantasy proneness is not a major factor in the etiology of DID because our results are in the opposite direction as predicted by the fantasy model. Hence, the results of these new conjunction analyses provide an important contribution to the etiology discussion.

Furthermore, it is important to note (top section of Table 4) that the controls are unable to simulate TPS in that there is no overlap in brain activation, but that they are able to partly simulate NPS. Conjoint activation in the three groups was found in the right primary auditory cortex, the right frontal and bilateral temporal regions for the within NPS comparison only. This is consistent with the notion that NPS functions in some regards as ‘normal.’ Taking further into account the second half of the Table we note that the majority of the conjointly (de-)activated regions between DID and CL (bilateral temporal gyrus, right occipitotemporal gyrus, left orbitofrontal, left amygdala, left cerebellum, and right precentral gyrus) are not included in the neurobiological model of DID. In addition, pivotal key regions involved in the regulation of hypoarousal in NPS (such as the prefrontal cortex, cingulate, the posterior association areas and the parahippocampal gyri) and of

hyperarousal in TPS (such as the insula as well as the dorsal striatum) in DID were not found in the control groups. These results do not support the fantasy model. We propose that the conjointly (de-)activated areas do not play a role in the DID symptomatology or emotion modulation/regulation. The conjointly (de-)activated regions may be assigned to general task performance, such as listening to, and early processing of, the auditory presented information, as well as functioning relatively 'normal'.

Limitations are: no validated quantitative trait and state dissociative symptom measures were obtained, the data were used for previous publications, information on specific psychiatric co-morbidities of the DID patients is not available, practice of DID simulation was relatively brief, and only a limited the number of patients was included. Nevertheless, to date our study is the only study investigating personality-state-dependent brain activation in response to autobiographical texts, while controlling for motivated role-playing. We recommend that future studies include larger sample sizes. Furthermore, our findings cannot be extended to DID populations in general as only female patients and controls participated in the study, even though the DID population consists mainly of females. Of note, a same-gender study does not suffer from gender differences (Bell et al., 2006). Despite these limitations our findings concur with the study's initial a priori hypothesis that high fantasy prone mentally healthy controls are unable to simulate DID. Overall our results contradict the fantasy model.

In conclusion: By presenting results of new exploratory analyses we answer to calls for more neurobiological information concerning dissociation from proponents of both the fantasy and trauma models. The results offer new information concerning the etiology of DID. This is important because empirical research into DID is still in an early phase. Results of the new post-hoc t-tests on the psychophysiological measures confirm the trauma model of DID. Results obtained from the brain data do not support the fantasy model of DID.

Conflict of interest

None declared.

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Figure and Tables

Figure 1: ‘‘Glass brain’’ renderings of significant effects

Figures 1A and 1B show differences in the processing of the trauma-related text (indicated with a small ‘t’) and the neutral text (indicated with a small ‘n’) within the neutral personality state (NPS). For statistical values and coordinates see Table 1. Figure 1A shows brain activation in the bilateral (B.) superior (S.) frontal gyrus for the dissociative identity disorder (DID) group as compared to the high fantasy prone mentally healthy DID simulating controls ($p < 0.001$, uncorrected for multiple comparisons, clusters larger than 8 voxels are depicted). Figure 1B shows a de-activation in the right (R.) hippocampus for high fantasy prone mentally healthy DID simulating controls as compared to low fantasy prone mentally healthy DID simulating controls ($p < 0.001$ uncorrected for multiple comparisons, clusters larger than 8 voxels are depicted).

Figure 1C shows differential processing of the neutral text (indicated with a small ‘n’) between the neutral personality state (NPS) and trauma-related personality state (TPS). For statistical values and coordinates see Table 3. Figure 1C shows brain activation in the bilateral (B.) caudate nucleus for the DID group as compared to the low fantasy prone mentally healthy DID simulating controls ($p < 0.001$, uncorrected for multiple comparisons, clusters larger than 8 voxels are depicted).

DID = dissociative identity disorder patient group.
CH = high fantasy prone DID simulating control group.
CL = low fantasy prone DID simulating control group.
NPSn = neutral personality state exposed to the neutral memory script.
NPSt = neutral personality state exposed to the trauma-related memory script.
TPSn = trauma-related personality state exposed to the neutral memory script.
B. = bilateral
S. = superior
R. = right

Table 1: Age and clinical measures for the controls

Mean and standard deviations (SD) for the high fantasy prone group and low fantasy prone group of the following questionnaires: Traumatic Experience Checklist (TEC (Nijenhuis et al., 2002)), Somatoform Dissociation Questionnaire (SDQ-20 (Nijenhuis et al., 1996)), and the Creative Experiences Questionnaire (CEQ) (Merckelbach et al., 2001) which measures fantasy proneness.

Table 2: Neurobiological results: Memory script effects within dissociative personality state

Overview of brain areas with statistically significant cerebral blood flow changes when comparing DID patients to high or low DID simulating controls (CH and CL respectively) and high to low fantasy prone controls (CH versus CL) for the trauma-related memory script effects within neutral or trauma-related dissociative personality states. Results for TPS are shown at the top and NPS at the bottom. Results for the TPSt – TPSn and vice versa have been published previously (Reinders et al., 2012) and are shown here for convenience as the conjunction analyses in Table 5 are dependent on these comparisons.

Table 3: Psychophysiological results: Memory script effects within dissociative personality state

This table shows the results for the post-hoc tests between the three groups for the within dissociative personality state processing of the trauma-related text as compared to the processing of the neutral text. As hypotheses were one-sided only one directional tests were performed and reported here.

Table 4: Neurobiological results: Memory script effects between dissociative personality states

Overview of brain areas with statistically significant cerebral blood flow changes when comparing DID patients to high or low DID simulating controls (CH and CL respectively) for the neutral and trauma-related memory script effects between trauma-related personality state (TPS: top) and neutral personality states (NPS: bottom). Results for the TPSt – NPSt and vice versa (top part) have been published previously (Reinders et al., 2012) and are shown here for convenience as the conjunction analyses in Table 5 are dependent on these comparisons.

Table 5: Psychophysiological results: Memory script effects between dissociative personality states

This table shows the results for the post-hoc tests between the three groups for the between dissociative personality state processing of the trauma-related text and the processing of the neutral text. As hypotheses were one-sided only one directional tests were performed and reported here.

Table 6: Neurobiological results: Conjunction analyses of the within and between dissociative personality state comparisons

Overview of brain areas with statistically significant cerebral blood flow changes when investigating conjoint activations in the DID patients and the high and/or low DID simulating controls (CH or/and CL) for the neutral and trauma-related memory script effects within and between neutral and trauma-related dissociative personality states.

Figure

Figure 1A

DID(NPSt–NPSn) – CH(NPSt–NPSn)

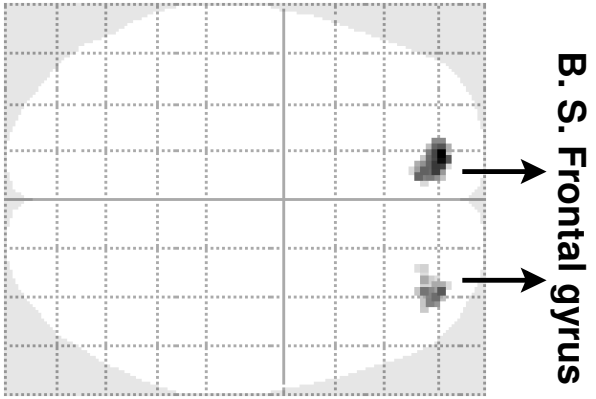


Figure 1B

CH(NPSn–NPSt) – CL(NPSn–NPSt)

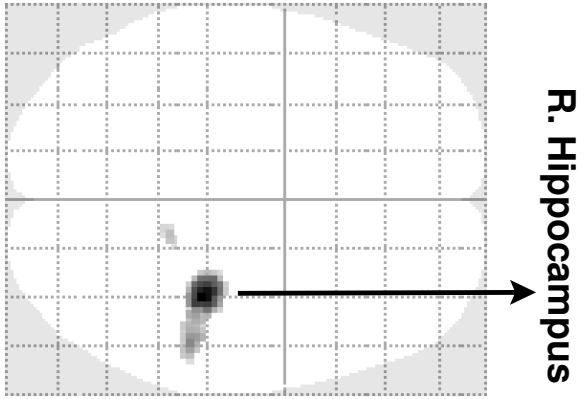
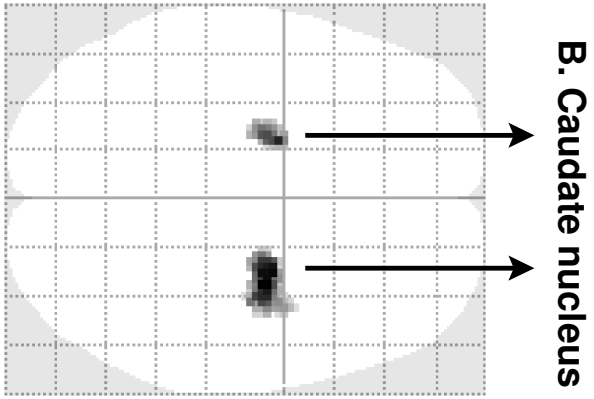


Figure 1C

DID(TPSn–NPSn) – CL(TPSn–NPSn)



Table

Table 6: Neurobiological results: Conjunction analyses of the within and between dissociative personality state comparisons

					Conjunction 2 groups										***Conjunction 3 groups***				
					DID & CH					**DID & CL**					**DID & CH & CL**				
		L/R	Brain region	BA	x	y	z	T**	kE	x	y	z	T**	kE	x	y	z	T**	kE
Within personality states																			
	TPSt - TPSn																		
	Cortical areas							n.s.					n.s.					n.s.	
	Subcortical areas							n.s.					n.s.					n.s.	
	Cerebellum							n.s.					n.s.					n.s.	
	TPSn - TPSt																		
	Cortical areas							n.s.					n.s.					n.s.	
	Subcortical areas							n.s.					n.s.					n.s.	
	Cerebellum							n.s.					n.s.					n.s.	
	NPSt – NPSn																		
	Cortical areas							n.s.					n.s.						
		R	Primary auditory cortex	BA 41/42											42	-18	26	2.47	637
		R	M. Frontal Gyrus	BA 9											24	30	20	2.45	212
	Subcortical areas							n.s.					n.s.					n.s.	
	Cerebellum							n.s.					n.s.					n.s.	
	NPSn – NPSt																		
	Cortical areas							n.s.											

		R	M. Temporal gyrus	BA 21						58	-54	-12	3.14	427	62	-50	-12	2.30	431
		L	I. Temporal gyrus	BA 37						-56	-52	-28	2.83	104	-56	-54	-32	2.38	307
		R	Occipitotemporal sulcus	BA 20						38	-30	-24	2.75	190					
	Subcortical areas							n.s.					n.s.					n.s.	
	Cerebellum							n.s.					n.s.					n.s.	
Between personality states																			
	TPSt-NPSt																		
	Cortical areas							n.s.										n.s.	
		L	Orbitofrontal cortex	BA 11						-34	30	-24	3.14	175					
		L	M. Temporal gyrus	BA 21						-42	0	-22	2.48	107					
		L	S. Temporal gyrus	BA 38						-52	18	-16	2.30	34					
		R	Occipitotemporal sulcus	BA 20						38	-14	-24	2.34	24					
	Subcortical areas							n.s.										n.s.	
		L	Amygdala							-12	4	-24	3.52	1281					
	Cerebellum																		
		L	Cerebellum					n.s.		-10	-48	-32	3.18	253				n.s.	
	NPSt-TPSt																		
	Cortical areas							n.s.										n.s.	
		R	Precentral gyrus	BA 6						44	0	50	3.22	414					
	Subcortical areas							n.s.						n.s.				n.s.	
	Cerebellum							n.s.						n.s.				n.s.	
	TPSn-NPSn																		

	Cortical areas						n.s.					n.s.					n.s.	
	Subcortical areas						n.s.					n.s.					n.s.	
	Cerebellum						n.s.					n.s.					n.s.	
	NPSn-TPSn																	
	Cortical areas						n.s.					n.s.					n.s.	
	Sub-cortical areas						n.s.					n.s.					n.s.	
	Cerebellum						n.s.					n.s.					n.s.	

DID = dissociative identity disorder patients
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BA = Brodmann area
L/R = Left/Right
** $p < 0.05$, corrected for multiple comparisons for the whole brain

I. = Inferior
M. = Middle
S. = superior

Table

Table 5: Psychophysiogical results: Memory script effects between dissociative personality states

	Processing of the trauma-related text			Processing of the neutral text		
	DID(TPSt-NPSt) > CH(TPSt-NPSt)	DID(TPSt-NPSt) > CL(TPSt-NPSt)	CH(TPSt-NPSt) > CL(TPSt-NPSt)	DID(TPSn-NPSn) > CH(TPSn-NPSn)	DID(TPSn-NPSn) > CL(TPSn-NPSn)	CH(TPSn-NPSn) > CL(TPSn-NPSn)
Subjective ratings						
Sensory rating	***F(1,20)=20.33, P<0.001****	***F(1,18)=13.06, P=0.002****	n.s.	n.s.	n.s.	***F(1,17)=6.95, P=0.018****
	DID(M=4.10, SD=2.38)	DID(M=4.10, SD=2.38)	CH(M=0.62, SD=0.56)	DID(M=0.30, SD=0.64)	DID(M=0.30, SD=0.64)	CH(M=0.01, SD=0.33)
	CH(M=0.62, SD=0.56)	CL(M=0.96, SD=0.65)	CL(M=0.96, SD=0.65)	CH(M=0.01, SD=0.33)	CL(M=0.47, SD=0.41)	CL(M=0.47, SD=0.41)
Emotional rating	n.s. ^	***F(1,18)=4.61, P=0.046****	n.s.	n.s.	n.s.	n.s.
	DID(M=4.21, SD=3.05)	DID(M=4.21, SD=3.05)	CH(M=2.07, SD=1.45)	DID(M=0.11, SD=0.31)	DID(M=0.11, SD=0.31)	CH(M=0.45, SD=0.89)
	CH(M=2.07, SD=1.45)	CL(M=1.67, SD=1.59)	CL(M=1.67, SD=1.59)	CH(M=0.45, SD=0.89)	CL(M=0.33, SD=0.52)	CL(M=0.33, SD=0.52)
Autonomic reactions						
Heart rate frequency	***F(1,20)=11.55, P=0.003****	***F(1,18)=9.95, P=0.006****	n.s.	n.s.	n.s.	n.s.
	DID(M=9.64, SD=8.39)	DID(M=9.64, SD=8.39)	CH(M=-0.08, SD=3.50)	DID(M=1.18, SD=4.71)	DID(M=1.18, SD=4.71)	CH(M=-1.17, SD=2.55)
	CH(M=-0.08, SD=3.50)	CL(M=-0.21, SD=3.01)	CL(M=-0.21, SD=3.01)	CH(M=-1.17, SD=2.55)	CL(M=-0.29, SD=2.96)	CL(M=-0.29, SD=2.96)
Systolic blood pressure	***F(1,20)=9.11, P=0.007****	***F(1,18)=4.86, P=0.042****	n.s.	n.s.	n.s.	n.s.
	DID(M=10.45, SD=11.92)	DID(M=10.45, SD=11.92)	CH(M=-1.25, SD=2.89)	DID(M=2.00, SD=6.23)	DID(M=2.00, SD=6.23)	CH(M=-0.48, SD=4.14)
	CH(M=-1.25, SD=2.89)	CL(M=0.63, SD=4.54)	CL(M=0.63, SD=4.54)	CH(M=-0.48, SD=4.14)	CL(M=-1.96, SD=4.43)	CL(M=-1.96, SD=4.43)
Diastolic blood pressure	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
	DID(M=3.59, SD=7.59)	DID(M=3.59, SD=7.59)	CH(M=0.47, SD=3.66)	DID(M=1.14, SD=4.17)	DID(M=1.14, SD=4.17)	CH(M=1.12, SD=2.66)
	CH(M=0.47, SD=3.66)	CL(M=1.38, SD=1.70)	CL(M=1.38, SD=1.70)	CH(M=1.12, SD=2.66)	CL(M=-0.71, SD=2.33)	CL(M=-0.71, SD=2.33)
HRV-average	***F(1,19)=5.66, P=0.029****	***F(1,17)=4.65, P=0.047****	n.s.	n.s.	n.s.	n.s.
	DID(M=-70.79, SD=86.37)	DID(M=-70.79, SD=86.37)	CH(M=2.20, SD=34.11)	DID(M=12.67, SD=43.26)	DID(M=12.67, SD=43.26)	CH(M=8.46, SD=34.03)
	CH(M=2.20, SD=34.11)	CL(M=2.19, SD=24.99)	CL(M=2.19, SD=24.99)	CH(M=8.46, SD=34.03)	CL(M=-2.65, SD=32.57)	CL(M=-2.65, SD=32.57)

DID = dissociative identity disorder patients
CH = High fantasy prone DID simulating healthy controls
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TPSt = trauma-related personality state exposed to the trauma-related memory script
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** = corrected for multiple comparisons
* = $p < 0.05$ uncorrected for multiple comparisons
n.s. ^ = trend: $0.05 < p < 0.10$ uncorrected
n.s. = not significant
M = mean
SD = standard deviation

Table

Table 4: Neurobiological results: Memory script effects between dissociative personality states

				Between group														
				DID - CH					**DID - CL**					**CH – CL**				
	L/R	Brain region	BA	x	y	z	T*	kE	x	y	z	T*	kE	x	y	z	T*	kE
TPSt-NPSt																		
Cortical areas																	n.s.	
	R	Postcentral gyrus	BA 43	68	-14	14	3.44	12	68	-14	18	4.45**	106					
	R	Precentral gyrus	BA 6						66	8	20	3.87	41					
	R	Insula	BA 13						46	-12	26	3.41	16					
Subcortical areas																		
	L	Amygdala		-6	-6	-26	3.75	129						-16	-2	-20	3.51	43
	L	Caudate nucleus (dorsal part)/ Insula	_ / BA 13	-12	2	18	3.90	34	-12	4	16	4.71**	568					
	R	Caudate nucleus (dorsal part)/ Putamen		24	-2	14	4.38	352	26	0	18	5.09**	490					
	L	Putamen		-24	-18	14	3.55	29										
	R	Parietal operculum	BA 40/43						48	-2	22	3.40	37					
Cerebellum																		
	L	Cerebellum (dorsal-medial part)		-6	-46	-34	3.63	47						-8	-36	-10	3.54	38
NPSt-TPSt																		

Cortical areas																	n.s.	
	R	Cingulate gyrus	BA 32	8	14	36	3.74	89										
	L	Cuneus/ Precuneus	BA 7/18/19/31	-8	-66	26	4.72	921	-12	-72	30	3.64	92					
			BA 18/19						-16	-90	36	3.56	20					
	R	Cuneus	BA 18/19	12	-80	22	3.86	252										
	L	S. Frontal sulcus	BA 6						-34	-2	52	3.64	36					
	R	S. Frontal sulcus/ Cingulate sulcus	BA 4/6	28	-16	44	3.61	33	20	-10	46	3.53	72					
	R	Fusiform gyrus/ Lingual gyrus	BA 18						26	-96	-20	3.34	34					
	L	Lingual gyrus	BA 18						-4	-90	-10	4.33**	616					
	L	S. Occipital gyrus/ Angular gyrus	BA 19/39	-38	-82	30	3.72	82	-42	-78	32	4.27**	128					
	R	Occipitotemporal sulcus	BA 20/37	48	-40	-12	4.53	92	46	-36	-14	5.24**	294					
	L	Parahippocampal gyrus	BA 35	-40	-46	-4	3.75	18	-40	-46	-6	4.73**	780					
	R	Parahippocampal gyrus	BA 36	20	-52	2	3.46	52	22	-52	0	4.26**	482					
	L	Intra-Parietal sulcus	BA 7/40	-34	-50	34	4.36	117										
	R	Intra-Parietal sulcus	BA 7/40	30	-38	40	4.36	249	34	-34	38	3.52	30					
	R	S. Parietal lobule/	BA 7	24	-64	30	3.95	108	24	-64	36	3.80**	48					

		Precuneus																
	X	Rectal gyrus	BA 11						0	28	-12	3.82**	85					
	L	M. Temporal gyrus	BA 21						-54	-24	-10	3.71**	63					
	R	M. Temporal gyrus	BA 21	62	-6	-14	3.64	15	62	-6	-14	4.16**	120				n.s.	
Subcortical areas							n.s.					n.s.					n.s.	
Cerebellum							n.s.					n.s.						
TPSn-NPSn																		
Cortical areas							n.s.					n.s.					n.s.	
Subcortical areas							n.s.					n.s.					n.s.	
	L	Caudate nucleus (dorsal part)		-18	-6	18	3.65	28	-20	-2	18	3.86	65					
	R	Caudate nucleus (dorsal part)					n.s.		30	-6	18	4.02	225					
Cerebellum							n.s.					n.s.					n.s.	
NPSn-TPSn																		
Cortical areas							n.s.					n.s.					n.s.	
Subcortical areas							n.s.					n.s.					n.s.	
Cerebellum							n.s.					n.s.					n.s.	

DID = dissociative identity disorder patients
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TPSt = trauma-related personality state exposed to the trauma-related memory script
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BA = Brodmann area
L/R = Left/Right
* $p < 0.05$, corrected for multiple comparisons
** $p < 0.05$, corrected for multiple comparisons for the whole brain

M. = Middle
S. = superior

Table

Table 3: Psychophysiogical results: Memory script effects within dissociative personality state

	Trauma-related personality state: differential processing of the trauma-related and neutral text			**Neutral personality state: differential processing of the trauma-related and neutral text**		
	DID(TPSt-TPSn) > CH(TPSt-TPSn)	DID(TPSt-TPSn) > CL(TPSt-TPSn)	CH(TPSt-TPSn) > CL(TPSt-TPSn)	DID(NPSt-NPSn) > CH(NPSt-NPSn)	DID(NPSt-NPSn) > CL(NPSt-NPSn)	CH(NPSt-NPSn) > CL(NPSt-NPSn)
Subjective ratings						
Sensory rating	***F(1,20)=33.71, P<0.001***	***F(1,18)=27.15, P<0.001***	n.s.	n.s.	n.s.	n.s.
	DID(M=4.39, SD=2.04)	DID(M=4.39, SD=2.04)	CH(M=0.51, SD=0.57)	DID(M=0.60, SD=1.24)	DID(M=0.60, SD=1.24)	CH(M=-0.10, SD=0.28)
	CH(M=0.51, SD=0.57)	CL(M=0.46, SD=0.69)	CL(M=0.46, SD=0.69)	CH(M=-0.10, SD=0.28)	CL(M=-0.03, SD=0.16)	CL(M=-0.03, SD=0.16)
Emotional rating	***F(1,20)=16.24, P=0.001***	***F(1,18)=23.97, P<0.001***	n.s.	n.s.	n.s.	n.s.
	DID(M=5.42, SD=1.88)	DID(M=5.42, SD=1.88)	CH(M=2.21, SD=1.76)	DID(M=1.32, SD=1.74)	DID(M=1.32, SD=1.74)	CH(M=0.59, SD=1.43)
	CH(M=2.21, SD=1.76)	CL(M=1.63, SD=1.30)	CL(M=1.63, SD=1.30)	CH(M=0.59, SD=1.43)	CL(M=0.29, SD=0.47)	CL(M=0.29, SD=0.47)
Autonomic reactions						
Heart rate frequency	***F(1,20)=24.42, P<0.001***	***F(1,18)=18.53, P<0.001***	n.s.	n.s. ^	n.s. ^	n.s.
	DID(M=11.45, SD=7.00)	DID(M=11.45, SD=7.00)	CH(M=0.20, SD=1.72)	DID(M=3.00, SD=3.46)	DID(M=3.00, SD=3.46)	CH(M=-0.88, SD=5.20)
	CH(M=0.20, SD=1.72)	CL(M=0.46, SD=1.84)	CL(M=0.46, SD=1.84)	CH(M=-0.88, SD=5.20)	CL(M=0.38, SD=1.27)	CL(M=0.38, SD=1.27)
Systolic blood pressure	***F(1,20)=11.37, P=0.003***	***F(1,18)=5.53, P=0.031 ****	***F(1,17)=7.04, P=0.017 ****	n.s.	n.s.	n.s.
	DID(M=12.95, SD=12.46)	DID(M=12.95, SD=12.46)	CH(M=-0.47, SD=1.66)	DID(M=4.50, SD=7.16)	DID(M=4.50, SD=7.16)	CH(M=0.30, SD=2.92)
	CH(M=-0.47, SD=1.66)	CL(M=2.33, SD=2.79)	CL(M=2.33, SD=2.79)	CH(M=0.30, SD=2.92)	CL(M=-0.25, SD=3.90)	CL(M=-0.25, SD=3.90)
Diastolic blood pressure	***F(1,20)=12.95, P=0.002***	***F(1,18)=8.49, P=0.010 ****	n.s.	n.s. ^	n.s. ^	n.s.
	DID(M=7.36, SD=5.37)	DID(M=7.36, SD=5.37)	CH(M=-0.20, SD=4.11)	DID(M=4.91, SD=7.15)	DID(M=4.91, SD=7.15)	CH(M=0.45, SD=3.59)
	CH(M=-0.20, SD=4.11)	CL(M=1.71, SD=1.11)	CL(M=1.71, SD=1.11)	CH(M=0.45, SD=3.59)	CL(M=-0.38, SD=2.11)	CL(M=-0.38, SD=2.11)
HRV-average	***F(1,19)=11.06, P=0.004***	***F(1,17)=8.58, P=0.010 ****	n.s.	n.s.	n.s.	n.s.
	DID(M=-107.94, SD=95.30)	DID(M=-107.94, SD=95.30)	CH(M=-0.62, SD=15.84)	DID(M=-24.48, SD=54.63)	DID(M=-24.48, SD=54.63)	CH(M=5.64, SD=17.36)

	CH(M=-0.62, SD=15.84)	CL(M=-0.78, SD=11.32)	CL(M=-0.78, SD=11.32)	CH(M=5.64, SD=17.36)	CL(M=-5.62, SD=14.87)	CL(M=-5.62, SD=14.87)

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TPSt = trauma-related personality state exposed to the trauma-related memory script
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** = corrected for multiple comparisons
* = $p < 0.05$ uncorrected for multiple comparisons
n.s. ^ = trend: $0.05 < p < 0.10$ uncorrected
n.s. = not significant
M = mean
SD = standard deviation

Table

Table 2: Neurobiological results: Memory script effects within dissociative personality state

				Between group														
				DID - CH					**DID - CL**					**CH – CL**				
	L/R	Brain region	BA	x	y	z	T*	kE	x	y	z	T*	kE	x	y	z	T*	kE
TPSt - TPSn																		
Cortical areas																		
	L	Insula	BA 13	-38	-14	14	3.61	48	-38	-14	14	4.61	327				n.s.	
	R	Postcentral gyrus	BA 43						68	-14	18	3.58	19				n.s.	
	R	I. Temporal gyrus	BA 20	32	-12	-44	3.57	9									n.s.	
Subcortical areas																	n.s.	
	L	Amygdala		-12	-4	-26	4.05	132									n.s.	
	L	Caudate nucleus (dorsal part)							-12	4	16	4.13	39				n.s.	
	R	Caudate nucleus (dorsal part)		24	-2	14	3.64	53	26	2	20	3.76	56				n.s.	
	L	Caudate nucleus (tail)		-22	-24	16	3.60	13									n.s.	
Cerebellum																	n.s.	
TPSn - TPSt																		

Cortical areas																		
	R	Cingulate sulcus	BA 6/24						20	-10	52	3.76	101				n.s.	
	L	(Pre-)Cuneus	BA 7/31/18/19	-12	-66	26	4.25	210	-16	68	28	4.26	181				n.s.	
	R	Fusiform gyrus	BA 19/37						34	58	-20	4.11	478				n.s.	
	L	S. Occipital gyrus/ Angular gyrus	BA 19/39						-42	80	28	3.59	29				n.s.	
	R	Occipitotemporal sulcus	BA 20/37	48	-38	-12	3.65	23	46	-36	-14	4.31	89				n.s.	
	R	Intra-Parietal sulcus	BA 7/40	34	-36	36	3.72	62	34	-34	38	4.13	122				n.s.	
	R	S. Parietal lobule/ Precuneus	BA 7						28	-66	32	3.49	34				n.s.	
	R	M. Temporal gyrus	BA 21	60	-2	-16	3.33	11									n.s.	
Cerebellum																	n.s.	
	L	Cerebellum (anterior lobe)							-4	-44	-14	3.62	48				n.s.	
NPSt – NPSn																	n.s.	
Cortical areas	L	S. Frontal Gyrus	BA 9	-16	56	34	3.80	100				n.s.					n.s.	
	R	S. Frontal Gyrus	BA 9	32	56	34	3.53	40									n.s.	

Subcortical areas							n.s.										n.s.	
Cerebellum							n.s.										n.s.	
NPSn – NPSt																		
Cortical areas																		
	R	Hippocampus					n.s.					n.s.		34	-30	-10	4.78	335
Subcortical areas							n.s.					n.s.						
Cerebellum							n.s.					n.s.						

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L/R = Left/Right
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I. = Inferior
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Table

Table 1: Age and clinical measures for the controls

	Mean (SD)	
	CH (n=10)	CL (n=8)
Age^	38.2 (10.9)	42.5 (10.1)
Creative Experiences Questionnaire (CEQ)	13.7 (3.2)	3.9 (1.6)
Traumatic Experience Checklist (TEC)	0.7 (1.3)	0.4 (0.5)
Somatoform Dissociation Questionnaire (SDQ-20)	22 (2.4)	20.9 (1.5)

^ Age of dissociative identity disorder patients: 41.0 (6.1)

Abbreviations: CH = high fantasy prone controls; CL = low fantasy prone controls