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The Neuropsychiatry of Dissociative Identity Disorder: Why Split Personality Patients Switch Personalities Intermittently?

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Abstract

Little has been known about the possible brain changes in the patients suffering from the psychiatric illness called Dissociative Identity Disorder (DID). This review suggests that the patients of DID have structural changes in the limbic system – hippocampus and amygdala -and the cortex. Blood flow is also altered in the orbito-frontal cortex. Another interesting finding of this review is that the glutamate release is found to be the cause of the dissociative symptoms, if this is true, then probably the glutamate blockers is the future for managing such patients. Furthermore, this review also highlights that in some DID patients the dorsolateral prefrontal and parietal cortex are activated. The limbic system especially the amygdala and the dorsolateral prefrontal and parietal cortex are associated with the short term and working memory, while the hippocampus is associated with long-term memory. All of these regions are somehow affected in the DID patient's brain. Thus, this explains the symptoms of the multiple personality changes and forgetting about the previous personality. Why the nature of these symptoms is temporary despite evidence for permanent structural brain changes is perhaps a question that is yet to be answered. Future studies will broaden our knowledge about the 'cyclical nature' and complete neuropsychiatry of this unique medical illness.

Keywords Neuropsychiatry; Psychiatric illness; Parietal cortex; Hippocampus

Introduction

A 17-year-old female patient was seen in a psychiatric ward at a local hospital in California, USA, she could change her voice anytime and claimed to be possessed by demons occasionally, while sometimes she believed she was a 70-year-old catholic nun, and other times she was in her usual self and sometimes she thought she was a 28-year-old man named Victor. She could speak multiple languages and while switching from one voice to another, she could totally change her personality. She was belligerent and verbally abusive toward the paramedics, doctors and the nurses. She refused to undergo any medical treatment despite being belligerent. To keep her calm, she was given the IV haloperidol, which temporarily calmed her down. She was released from the hospital when she felt a little better. While her family sought the help of a local church for a possible exorcism, and ignored medical treatment, the girl eventually died of dehydration few months later.

The above-mentioned case is a typical example of a mental disorder-what we call today, as dissociative identity disorder (DID), previously known as split personality disorder or multiple personality disorder. People suffering from DID may have two or more distinct personalities with possibly different ages, gender and culture [1]. Each personality has its own features and characteristics. DID is one of the most interesting yet one of the least understood pathologies. Modern science is expanding the knowledge to help future scientists explore more about the possible causes and mechanism behind this disorder. Regarding the involvement of brain, few studies have hinted and given us some idea about the disorder. Some similarities with schizophrenia and some with bipolar have been discussed in the previous literature [1,2].

This article, is an attempt to explore the recent literature up to the year 2017, to understand what parts of the human brain are involved in the pathogenesis and what other kinds of alterations are found associated with DID. Studying this disorder in depth and understanding the exact mechanism or causes will not only help the doctors and scientists to enhance our knowledge base, but it will also help patients.

This review article will explain some of the structural, functional and chemical changes in the brain of the DID patients. The article will also address the important question about why many DID patients switch personalities and forget about the previous personality while under the influence of one personality. In this article, we will also address the blood flow changes in these patient's brain in certain regions of the brain. In the end, we will recommend more studies to increase and fill the knowledge gap that is currently unknown.

Brain changes in DID

The quantitative evidence supporting changes in the brain of a person with DID is limited. However, some studies have clearly reflected the possibility of changes in the structural components of the brain, including the limbic system, the cerebral cortex and the blood flow to the Cerebral Cortex [1].

In 2003, Brunson et al. [2] suggested that stress is one of the leading factors affecting the volume of the hippocampus. This notion emphasizes the association of stress with DID and early exposure to

stress and changes in the volume of stress specific regulatory structures of the brain. Upon our review, numerous studies proposed that the most significant precipitating factors associated with DID is childhood trauma. Thus, leaving us with the question, does childhood trauma lead to changes in the volume of the hippocampus and the amygdala? In order to find this answer additional neurological research is needed in collaboration with radiological studies (Figure 1 and Table 1).

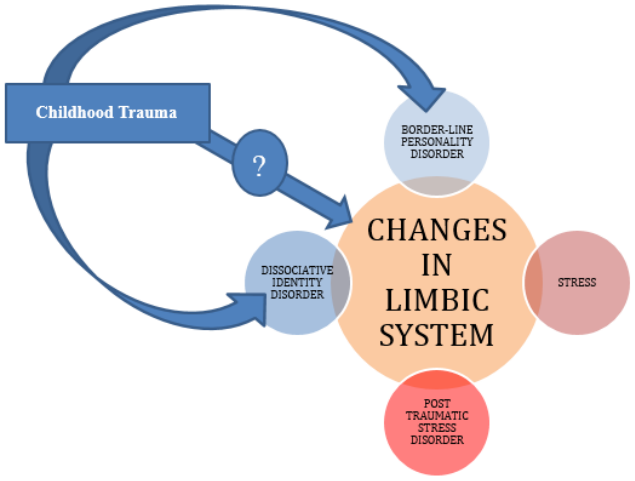

Figure 1: Relationship Diagram showing Psychological Disorders that have morphological changes in Brain. Childhood trauma is strongly associated with DID and Borderline Personality Disorder. This raises a question that "Is it possible that children who undergo trauma at some point in their lives, have some alterations in the brain morphology?"

A Magnetic Resonance Imaging (MRI) study by Vermetten, et al. compared the brain structure of female patients diagnosed with DID with healthy subjects. This study provided the evidence for changes in the limbic system. The hippocampus and amygdala in DID patients were found to be remarkably smaller (19.2% and 31.6%, respectively). The Amygdala controls emotions while the hippocampus controls long-term memory, thus Vermetten's study does provide a logical explanation for the pathophysiology of DID [3].

However, another MRI study of the hippocampus and amygdala demonstrated volume reduction in patients with Post Traumatic Stress Disorder (PTSD), while no considerable difference was seen in the hippocampus and amygdala of Dissociate Amnesia (DA), DA/DID patients or normal subjects [4]. Borderline personality disorder, on the other hand is also a known psychopathology associated with early-age trauma [5]. Investigations also indicate that patients with Borderline personality disorder have smaller hippocampal and amygdalar volumes [6-8]. There has long been a debate about the validity of DID as a mental disorder. Some scholars have suggested that DID is actually fantasy proneness while other scholars argue that all patients with DID have been exposed to early age trauma in their history [9-16]. Vermetten et al. [3] counter argued the MRI study by Weniger et al. by claiming, "patients with true dissociative identity disorder without PTSD essentially do not exist" [3]. What early age PTSD, Borderline personality disorder and DID all have in common is a disruption in early childhood development. In addition to the anatomical change in the volume of these regions of the brain, differences in the biochemical response to trauma have also been observed in the cortico-limbic system (Figure 2).

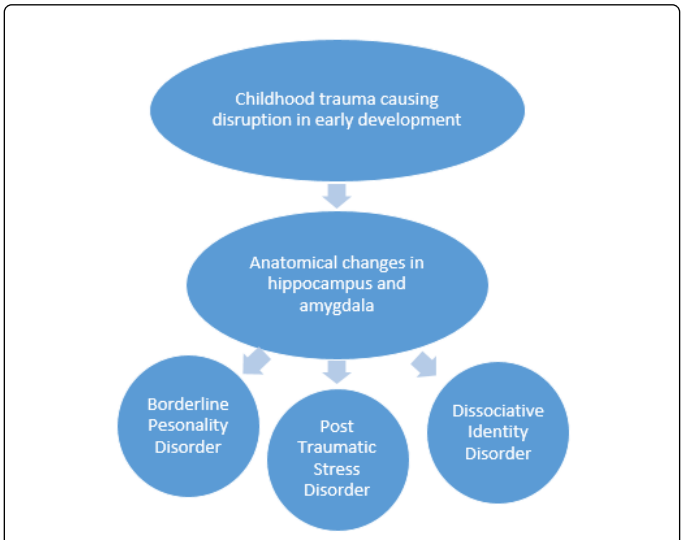

Figure 2: The Relationship of Childhood trauma, Brain Morphology and various Psychological Disorders.

Animal models have shown that glutamate is released in the cortico-limbic system during stress, and may lead to neurotoxicity and behavioral changes with transient dissociation. Glutamate release may also be responsible for inducing changes in neural plasticity. Similarly, N-Methyl-D-Aspartate (NMDA) antagonists have been used in similar animal models resulting in glutamate secretion. Investigations using NMDA antagonists such as ketamine, indeed do stimulate the release of glutamate in humans thus likely producing the associated trance-like dissociative state. Drugs that inhibit Glutamate should be investigated to elucidate its potential for early intervention in patients with traumatic events [17]. Flashbacks with vivid memories are diagnostic of PTSD. We speculate that with every vivid memory or flashback, glutamate release can result in long-term potentiation, offering a possible explanation for the biochemical mechanism of DID. This postulation further supports Vermetten et al's claim that patients with true dissociative identity disorder do not exist without PTSD. Acute Stress Disorder (ASD) follows the same diagnostic guidelines as for PTSD, except for the timeline. ASD takes place within the first month after the identifiable stressor. If the symptoms persist beyond one month, the diagnosis is considered PTSD. We believe a glutamate blocker may have the greatest effect in preventing neuroplasticity changes in the hippocampus and amygdala and lower the incidence of PTSD cases and limit the progression to DID if the patient starts treatment during the ASD period. Ketamine has become a popular medication in the study of treating acute suicidal ideation; however, a study by M. Schönenberg et al. found that Ketamine aggravates symptoms of acute stress disorder three days post-event with increased symptoms of dissociation, flashbacks, hyperarousal and avoidance relative to the comparison groups [18]. Riluzole is a glutamate blocker and currently- FDA approved in the treatment of ALS. Interestingly, a Pilot Study at Yale University, is currently investigating the safety and efficacy of the glutamate blocker in patients with PTSD. The results of this 4-year experiment are expected to be released in 2018 and we will follow these results. However, more studies are needed to explore the relationship of glutamate in the neuronal circuitry of DID patients.

This can be a potential target for intervention in DID. Depending on how glutamate levels are changed in this disorder, there does exist a possibility – that glutamate blockers administration targeted therapy may be used in the future to normalize the high levels of glutamate in the brain. However, it is still premature to recommend any such therapy until future clinical trials provide the evidence consistently regarding the efficacy of this nature of treatment.

Working memory involves certain regions in the brain, more specifically the dorsolateral prefrontal and parietal cortex. Elzinga et al. [19], studied these regions of working memory in 16 patients with DID or dissociative disorder not otherwise specified (DDNOS). He found that the patients demonstrated increased activation in these regions when compared to a control group. In addition, the patients also made fewer errors, increased task, were more anxious and had poorer concentration in comparison to the controls (Figure 3) [19].

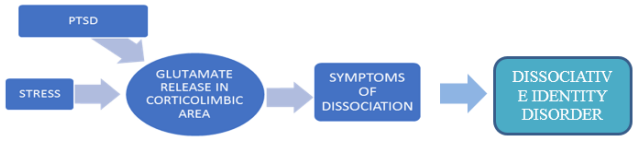

Figure 3: Glutamate is thought to cause the symptoms of dissociation. Chronic potentiation of glutamate may be underlying the pathophysiology of DID

Cerebral blood flow and DID

According to Sar et al. regional Cerebral Blood Flow (rCBF) was reduced in the bilateral orbitofrontal cortex (OFC) in the brains of DID patients. This is similar to the findings in patients with Attention Deficit Disorder (ADD). Furthermore, rCBF was augmented to the median and superior frontal regions and occipital lobes bilaterally [20]. "Decision making capacity has long been associated with the OFC. Considering this, Sar et al. stated that reduced functioning of the OFC leads to impulsivity and in patients with DID could express itself in the form of a new personality with impulsive behavior. Cerebral blood flow of DID patients has been studied by various investigations. Reinders et al. [21] calculated rCBF of DID patients in a neutral personality state (NPS) and compared it with a traumatic personality state (TPS). These subjects were then instructed to listen to a memory script. Interestingly, no differences were recorded between the NPS and TPS when listening to the neutral script. However, while listening to the traumatic script a deactivation pattern of brain areas in the NPS was present. The study concluded - that on a neurobiological level, the patients with DID possess different autobiographical selves. The medial prefrontal cortex was among the brain's region that was deactivated in the NPS versus TPS experiment. Furthermore, considering the OFC is part of the prefrontal cortex, this study points towards an idea about the involvement of the OFC in patients with DID [22]. In a previous study by Sar et al. showed that rCBF was reduced in the left and right OFC in DID patients but higher in their left (dominant) lateral temporal lobe [22]. Some other authors have also discussed the OFC involvement [22-24]. In another study by Reinder et al. [25], rCBF data demonstrated different neural networks to be linked with different processing of the neutral and trauma-related memory script by NIS and TIS. They found that patients with their own access to autobiographical trauma-related memory had variations in brain activations in distinct mental states of self-awareness [25]. It is quite clear from the discussion above that there are some alterations in the brain structure of the patients suffering from this mysterious psychiatric disorder. It is interesting to note that despite the permanent structural changes, the symptoms of DID like loss of memory and recurring personality switch overs are temporary in nature, a bewildering enigma. Much research is needed to unravel the cyclical nature of this disorder.

No.	Journal	Author	Year	Type of Study	Aim of Study	Patient population (if Applicable)	Conclusion
1	Journal of Cell Science and Therapy	Ashraf et al. [1]	2016	Mini-Review Article	To highlight the pathophysiology of DID and discuss Brain morphological changes in DID patients	-	Reduction in hippocampus size observed in DID patients. Reduced blood flow and functioning of Orbito-frontal Cortext in DID patients observed.
2	Molecular Neurobiology	Brunson et al. [2]	2003	Review	To elucidate the role of neuropeptide CRH and its long-term effects on the hippocampus in stressful episodes in early life.	-	Stress in early life affects hippocampus function acutely as well as chronically and CRH is an important contributor to this process. Abnormally high levels of neuropeptide CRH are released during pathological conditions which can promote excitotoxicity.
3	Am J Psychiatry	Vermetten et al. [3]	2006	Original Article	To compare hippocampal and amygdalar volumes in patients with dissociative identity disorder with those in	15 female patients with DID. 23 Female normal healthy patients	Hippocampal volume was 19.2% smaller and amygdalar volume was 31.6% smaller in the patients with dissociative identity disorder, compared to the healthy subjects. The ratio of hippocampal volume to amygdalar volume was significantly different between groups. T tests

					healthy subjects with no psycho-pathology.		resulted in significance differences for the left side (t=2.24, df=36, p<0.05) and the right side (t=2.07, df=36, p<0.04). The mean difference in these ratios was 15.8%.
4	Psychiatry research	Sar V et al. [4]	2007	Original Article	To investigate characteristics of regional cerebral blood flow (rCBF) in dissociative identity disorder vs control.	21 DID patients 9 healthy volunteers	The rCBF ratio was decreased among patients with dissociative identity disorder in the orbitofrontal region bilaterally. It was increased in median and superior frontal regions and occipital regions bilaterally. There was no significant correlation between rCBF ratios of the regions of interest and any of the psychopathology scale scores.
5	Am J Psychiatry	Zanarini et al. [5]	1997	Original Article	To assess pathological childhood experiences reported by Borderline Personality Disorder patients verses patients with other personality disorders	476 personality disorder patients. 358 borderline personality disorder 109 other personality disorders	Sexual abuse is not sufficient nor necessary for development of borderline personality. Other childhood experiences particularly, neglect from caregivers (both male and female) is a significant risk factor.
6	Arch Gen Psychiatry	Driessen et al. [6]	2000	Original Article	To assess if patients with borderline personality disorders (BPD), have smaller volumes of the hippocampus and the amygdala.	21 Female patients with BPD Similar group of normal women of same race handedness and similar age.	Female patients with BPD had nearly 16% smaller volumes of the hippocampus (P<.001) and 8% smaller volumes of the amygdala (P<. 05) than the healthy controls. The results for both hemispheres were nearly identical
7	Psychiatry Research	Schmahl et al. [7]	2003	Original Article	To measure hippocampal and amygdala volumes in female BPD patients	10 female patients with BPD and 23 female control subjects.	Female patients with BPD had a 21.9% smaller mean amygdala volume and a 13.1% smaller hippocampal volume, compared to controls.
8	Biological Psychiatry	Tebartz van Elst et al. [8]	2003	Original Article	To test the hypothesis that frontolimbic brain pathology might be associated with borderline personality disorder.	8 female patients with BDP 8 matched healthy controls	The patterns of volume loss of the amygdala, hippocampus, and left orbitofrontal and right anterior cingulate cortex might differentiate borderline personality disorder from other neuropsychiatric conditions. There was a significant 24% reduction of the left orbitofrontal and a 26% reduction of the right anterior cingulate cortex in borderline personality disorder. Only left orbitofrontal volumes correlated significantly with amygdala volumes.
9	Canadian Journal Of Psychiatry	Piper et al. [9]	2004	Review Article	To examine the concept of DID.	-	DID is best understood as a culture-bound and often iatrogenic condition.
10	Canadian Journal Of Psychiatry	Piper et al. [10]	2004	Review Article	The purpose of this review is to explore the illogical nature of the arguments that support DID as a medical condition	-	This review proposes that US and Canadian courts should not responsibly accept testimony in favour of DID. . It lays out why it is impossible to have a reliable diagnosis of DID due to the unsatisfactory, vague, and elastic definition of “alter personality.” It talks about how the diagnosis and treatment of DID helps encourage the person to believe in having multiple selves. It also talks about the harm which the DID treatment causes people

11	Canadian journal of psychiatry	Piper and Merskey H. [11]	2004	Review	To discuss various concepts and hypothesis about DID	NA	Lack of proof for association between childhood trauma and DID; Reliability on the diagnosis of DID; Lack of reported childhood DID cases in and 4) consistent evidence of blatant iatrogenesis appears in the practices of some of the disorder's proponents.
12	Psychological bulletin	Giesbrecht et al. [12]	2008	Review	To evaluate the research literature on cognitive processes in dissociation	NA	The review indicates that dissociation is characterized by subtle deficits in neuropsychological performance. It is associated with pseudo-memories, , fantasy proneness, and cognitive failures. Talks about lack of evidence for a link between dissociation and either memory fragmentation or early trauma.
13	Psychological bulletin (confirmmmm)	Douglas Bremner [13]	2011	Review and rebuttal of the argument of Giesbrecht et al	To show that there is no basis to not accept trauma and its potential association with dissociation		The argument of Giesbrecht et al. (2008)—that there is not a relationship between trauma and dissociation—is not correct. Literature review shows a relationship between trauma and dissociation. Counter- arguments that fantasy proneness, suggestibility, cognitive failures and associated dissociative tendencies lead to a spurious relationship with childhood trauma are not supported by a careful review of the literature.
14	Seminars in clinical neuropsychiatry	Chambers et al. [14]	1999		To highlight the effects of glutamate in dissociation and how its blockade can help attenuate symptoms in traumatized individuals with dissociative symptoms.		After stress exposure, dissociative symptoms are a predictor of the development of PTSD. Stress stimulates the cortico-limbic release of glutamate. Glutamate in animal models influences behavior, induces neural plasticity that affects brain function and behavior, and contributes to neural toxicity. NMDA antagonists may transiently stimulate glutamate release and produce symptoms resembling dissociative states in humans. A drug that reduces glutamate release also attenuates the perceptual effects of the NMDA antagonist, ketamine, in humans.
15	Journal of psychopharmacology (Oxford, England)	Schönenberg et al. [15]	2008		The aim of the present study was to examine whether ketamine administration after moderate accidental trauma modulates dissociation and other symptoms of acute stress disorder (ASD) in the direct aftermath of stressful event.		Ketamine aggravates symptoms of acute stress disorder three days' post-event with increased symptoms of dissociation, flashbacks, hyperarousal and avoidance relative to the comparison groups.
16	Psychological medicine	Bernet et al. [16]	2006		To assess working memory in dissociative patients functional magnetic resonance imaging during performance of a parametric, verbal working-memory task in patients with a dissociative		Dorsolateral and prefrontal regions of working memory in 16 patients with DID or dissociative disorder not otherwise specified (DDNOS) was studied and found that the patients demonstrated increased activation in these regions when compared to a control group. Also, the patients made fewer errors, increased task, were more anxious and had poorer concentration in comparison to the controls.

					disorder and healthy controls.		
17	Psychiatry research	Sar et al. [17]	2007		To investigate if there were any characteristics of regional cerebral blood flow (rCBF) in dissociative identity disorder.		Regional Cerebral Blood Flow (rCBF) was reduced in the bilateral orbitofrontal cortex (OFC) in the brains of DID patients just as in patients with Attention Deficit Disorder (ADD). In the median and superior frontal regions and occipital lobes bilaterally rCBF was increased.
18	NeuroImage	Reinders AA et al	2003		To show the changes in brain activity in the regions of the brain which are associated with generation of different mental states of self-awareness having its own version of trauma associated memories		Shows different cerebral blood flow patterns to the regions (the medial prefrontal cortex (MPFC) and the posterior associative cortices) for different senses of self-awareness experiences.
19	Arts and sciences writing program	Manton [19]	2016		To propose the attachment-OFC model in development of DID		Involvement of the OFC in patients with DID.Infant disorganized attachment may be a contributor to the development of DID.
20	Journal of trauma and dissociation	Sar et al. [20]	2008		To study the patterns of characteristic patterns of cerebral blood flow in DID patients		Study showed that rCBF was reduced in the left and right OFC in DID patients but higher in their left (dominant) lateral temporal lobe
21	Attachment and human development	Schore et al. [21]	2010		To study the psychological and biological mechanisms of Bowlby's attachment theory		Orbitofrontal system and its cortical and subcortical connections have been found to be the biological control system for instinctive behavior. It also talks about attachment research should focus upon the early-forming psychoneurobiological mechanisms that mediate both adaptive and maladaptive regulatory processes.
22	Seminars in clinical neuropsychiatry	Chambers et al. [22]	1999		After stress exposure, dissociative symptoms are a predictor of the development of PTSD. Glutamate neurotoxicity and its antagonism can cause its prevention.		Study talks about the hyperglutamatergic states causing acute and long-lasting consequences of traumatic stress exposure, the therapeutic and neuroprotective potential of drugs that antagonize glutamate release need to be explored in traumatized individuals with dissociative symptoms.

Table 1: Salient features of some articles reviewed.

Conclusion

The neuropsychiatry of dissociative identity disorder (DID) has long been in debate. Scientific community and the religious community have differed with each other on the exact nature of the disorder. The scientific community believes it to be a mental condition while the religious community believes it as a spiritual phenomenon. Our review suggests that DID is a pathophysiological disorder and brain structural and chemical changes are seen in the DID patients. The limbic system - Amygdala and hippocampus are affected as explained by various authors and scholars. Moreover, an increased activation in the dorsolateral prefrontal and parietal cortex is also seen in some DID

patients. The involvement of the limbic system & the dorsolateral prefrontal and parietal cortex (all these regions are associated with short-term and long term memory), possibly explains the main symptom of changing the personality and forgetting totally about the previous personality. The size of the hippocampus of DID patients is decreased in DID. While some changes in the orbitofrontal cortex are observed- with reduction in functioning and the blood flow. This suggests other possible symptoms like the changes in personality and the thought process. We also underscored the role of the neurotransmitter glutamate as a possible cause for the symptoms of dissociation. More studies need to elucidate this relationship. Depending on how this relationship unfolds, it can make a potential target for intervention in DID via glutamate blockers, if glutamate levels are found to be increased in DID as our review suggests. However, as mentioned above, it is still premature to recommend such a treatment. These kinds of findings can certainly be helpful for the psychiatrists, neuroscientists as well as the patients. Despite various studies, a challenging question still remains unanswered; like why the disorder presents with temporary amnesia rather than permanent memory loss specially when there is documented evidence for structural changes in the brain. We recommend more studies in the near future, to understand more about the neuropsychiatry and the pathophysiological mechanism for the cyclical nature of this clinical mystery.

References

1. Ashraf A, Krishnan R, Wudneh E, Acharya A, Tohid H, et al. (2016) Dissociative Identity Disorder: A Pathophysiological Phenomenon. J Cell Sci Ther 7: 251.
2. Brunson KL,Chen Y, Avishai-Eliner S, Baram TZ (2003) Stress and the developing hippocampus: a double-edged sword? Mol Neurobiol 27: 121-136.
3. Vermetten E, Schmahl C, Lindner S, Loewenstein RJ, Bremner JD (2006) Hippocampal and Amygdalar Volumes in Dissociative Identity Disorder. The Am J Psychiatry 163: 630-636.
4. Sar V, Unai SN, Oztu rk E (2007) Frontal and occipital perfusion changes in dissociative identity disorder. Psychiatry Res 156: 217-223.
5. Zanarini MC, Williams AA, Lewis RE, Reich RB, Vera SC, et al. (1997) Reported pathological childhood experiences associated with the development of borderline personality disorder. Am J Psychiatry 154: 1101–1106.
6. Driessen M, Herrmann J, Stahl K, Zwaan M, Meier S, et al. (2000) Magnetic resonance imaging volumes of the hippocampus and the amygdala in women with borderline personality disorder and early traumatization. Arch Gen Psychiatry 57: 1115–1122.
7. Schmahl CG, Vermetten E, Elzinga BM, Douglas Bremner J (2003) Magnetic resonance imaging of hippocampal and amygdala volume in women with childhood abuse and borderline personality disorder. Psychiatry Res 122: 193–198.
8. Tebartz van Elst L, Hesslinger B, Thiel T, Geiger E, Haegele K, et al. (2003) Frontolimbic brain abnormalities in patients with borderline personality disorder: a volumetric magnetic resonance imaging study. Biol Psychiatry 54: 163–171.
9. Piper A, Merskey H (2004) The persistence of folly: a critical examination of dissociative identity disorder. Part I. The excesses of an improbable concept. Can J Psychiatry 49: 592–600.
10. Piper A, Merskey H (2004) The persistence of folly: critical examination of dissociative identity disorder. Part II. The defence and decline of multiple personality or dissociative identity disorder. Can J Psychiatry 49: 678–683.
11. Coons PM (2005) Re: the persistence of folly: a critical examination of dissociative identity disorder. Can J Psychiatry 50: 813.
12. Fraser GA (2005) Re: the persistence of folly: a critical examination of dissociative identity disorder. Can J Psychiatry 50: 814.
13. Sar V (2005) Re: the persistence of folly: a critical examination of dissociative identity disorder. What are Dr Piper and Dr Merskey trying to do? Can J Psychiatry 50: 813.
14. Piper A, Merskey H (2005) The persistence of folly: A critical examination of dissociative identity disorder. Can J Psychiatry 50:814.
15. Giesbrecht T, Lynn SJ, Lilienfeld SO, Merckelbach H (2008) Cognitive processes in dissociation: an analysis of core theoretical assumptions. Psychol Bull 134: 617–647.
16. Bremner JD (2010) Cognitive processes in dissociation: comment on Giesbrecht, et al. (2008). Psychol Bull 136: 1–6; discussion 7–11.
17. Chambers RA, Bremner JD, Moghaddam B, Southwick SM, Charney DS, et al. (1999) Glutamate and post-traumatic stress disorder: toward a psychobiology of dissociation. Semin Clin Neuropsychiatry 4: 274–281.
18. Schönenberg M, Reichwald U, Domes G, Badke A, Hautzinger M (2008) Ketamine aggravates symptoms of acute stress disorder in a naturalistic sample of accident victims. J Psychopharmacol 22: 493-497.
19. Elzinga BM, Ardon AM, Heijnis MK, Ruiter MB, Dyck R, et al. (2007) Neural correlates of enhanced working-memory performance in dissociative disorder: a functional MRI study. Psychol Med 37: 235-245.
20. Sar V, Unai SN, Ozturk E (2007) Frontal and occipital perfusion changes in dissociative identity disorder.Psychiatry Res 156: 217-223.
21. Reinders A (2003) One Brain, Two Selves. Neuro Image 20: 2119-2125.
22. Manton S (2016) Disorganized Attachment and the Orbitofrontal Cortex as the Basis for the Development of Dissociative Identity Disorder. Art & Sciences Writing Program.
23. Sar V, Unal SN, Kiziltan E, Kundakci T, Ozturk E (2001) HMPAO SPECT study of regional cerebral blood flow in dissociative identity disorder. J Trauma Dissociation 2: 5-25.
24. Schore AN (2010) Attachment and the Regulation of the Right Brain. Attach Hum Dev 2: 23-47.
25. Reinders AA, Nijenhuis ER, Quak J, Korf J, Haaksma J, et al. (2006) Psychobiological characteristics of dissociative identity disorder: a symptomprovocation study. Biol Psychiatry 60: 730-740.