

The Correlation Between Stress and the Development of Dissociative Identity Disorder

Aviva Soibelman

Aviva Soibelman graduated June 2017 with a BS in biology

Abstract

Stress is a huge part of life. Our body responds to stress in different ways and most of the times the body overcomes the stress. There are a few incidents when the body is not capable of dealing with the stress and the toll it takes on the brain is undeniable. One result of intolerable stress is Dissociative Identity Disorder (DID) in which biopsychology comes to life, as we see how the body is affected by psychology, and vice versa. The mediators that help the body adapt to stressors become detrimental when a person faces trauma or chronic stress. Glucocorticoids, cortisol, and glutamate are all involved in either helping the body endure stress or causing plasticity in parts of the brain that are essential to being mentally healthy. This paper delves into the nature of DID, and how stress creates changes in the hippocampus and amygdala, the two parts of the limbic system that are smaller in patients with DID.

Introduction

In 1957 the world was exposed to what was then known as Multiple Personality Disorder (MPD) through the storyline of the movie, *Three Faces of Eve*. A classic example of MPD, a woman named Eve White was having symptoms of headaches and blackouts while being unaware of her disorder. One of the three identities, known as Black, is the reason her husband abandons her and is also why she tries to kill her own daughter. Black knows about White, but White does not know about Black. Through therapy the third identity emerges, known as Jane. Jane knows about all three identities and eventually all three identities merge (Lehman, 2014). This story of the *Three Faces of Eve* is a glimpse into the world of MPD. As time progressed new research has been developed and continues to develop regarding MPD, currently referred to as Dissociative Identity Disorder (DID), broadening the subject in all areas.

DID is a psychophysiological disorder that affects about 1-3% of the world's population (Vermetten et.al., 2006). DID diagnosis requires a minimum of two identities residing in one body. Each of these identities have their own biology, memories, perceptions and preferences. Common symptoms of DID include hallucinations and amnesic periods. An average of 8-13 identities develop and Daniel Goleman (1923) states that it is possible for one to be living with up to 60 different identities within oneself. The different identity types include the childlike personality type, the protector, and the persecutor. A misconception that the host personality is in fact the patient's original identity since it is the identity or alter ego will commonly seek treatment. The split in identities originates from the childhood of the patient. People with DID have a history of either sexual or physical abuse typically between the ages 3-8. Coherent with the common identity types, the childlike personality is the age in which the abuse took place. The persecutor is the identity that mirrors the abuser. The protector is the identity that feels responsible to protect the rest of the personalities from any abuse or trauma (Goleman, 1985). Each identity can be categorized as either an emotional personality or a normal personality. The emotional personalities are the ones that react and respond to the trauma they experienced. The normal personalities are the ones that deal with normal and daily functions (Nijenhuis, Steele, 2010). DID is often misdiagnosed as other personality disorders due to the complexity of the various identities. An identity in itself may have a personality disorder

such as Obsessive Compulsive Disorder and be treated for that. From an outsider's perspective what might seem like a suicide attempt may actually be one identity trying to kill another and thus must be dealt with appropriately. Additionally, DID is very similar to Post Traumatic Stress Disorder (PTSD) except that in a patient with DID the trauma happens in early childhood and in PTSD it occurs in adulthood (Gillig, 2009). In addition, 80-100% of patients diagnosed with DID are diagnosed with PTSD as well (Vermetten et.al., 2006).

There are cases in which one alter ego can be deathly allergic to bee stings, or may need glasses to see, while another alter ego in the same body may not have any reaction to bee stings or may not need glasses to see. There is a case study involving a 33-year-old female diagnosed with DID who suffered from blindness after a car accident. She was confirmed to be visually impaired after going through tests such as ocular fundus inspection, Humphrey refractometry, and intraocular tension measurement, which are all ways to test for vision or lack thereof. She even failed to show signs of involuntary reflexes such as eye watering and winking. After years of treating the patient with therapy for DID, one of the identities became visually active and soon many of the identities followed and retrieved vision. Because only some identities were blind, just like any identities can fluctuate, the patient was fluctuating between sight and blindness within seconds. Proof of complete blindness in one identity and complete sight in another was seen by visual evoked potential (VEP) recording system. In a blind identity the pattern evoked potentials showed a disorganized blind pattern. When the VEP was run on a sighted identity, there were organized patterns cohesive to a visually active person. The VEP results for the blind identity shows lack of vision, yet insufficient for complete blindness. The blindness that was relayed by the tests can be explained as psychogenic blindness that was activated by a temporary loss of vision caused by the accident. The psychogenic blindness is proof that in DID psychology takes a toll on the biology of a person (Strasburger, Waldvogel, (2015).

Dissociative identity disorder creates questions within the realms of sense of self; how can it be that two completely different identities are being controlled by one brain? Experiments using Positron Emission Tomography (PET) were performed to see how the brain creates these two different senses of self. To help explain this phenomena Damasio and coworkers created a theory that there is a core self, constantly synchronized

Correlation Between Stress and Development of Dissociative Identity Disorder

with the body and controlled by the body's biology, and the autobiographical self that are memories and is influenced by environmental factors. According to this theory only the autobiographical self is capable of change, yet the core self remains intact because it comes from the same biological makeup. Since the autobiographical manner of processing memory can be disrupted, the neutral personality state processes trauma in a non-autobiographical way, causing different senses of self in the brain. A study consisting of 11 females with DID in four different conditions was done. These four conditions included a neutral memory exposed to both the neutral and traumatic personality state, as well as a traumatic memory exposed to both a neutral and traumatic personality state. There were decreased amounts of regional cerebral blood flow (rCBF) perfusions when a traumatic memory was exposed to the traumatic personality state. The rCBF perfusions were unchanged when a traumatic memory was exposed to the neutral personality state, and when a neutral memory was exposed to both traumatic and neutral personality state. The areas that showed a decrease in rCBF perfusion correlated with the areas associated with the autobiographical senses-of-self. Decreased perfusion in the visual association area and the middle occipital gyrus causes the lack of integration of information and blocks emotional processing; this is the defense system manifested by patients with DID. Another symptom of DID is depersonalization, which is caused by decreased perfusion in parietal and occipital areas of the brain. Additionally, there is decreased perfusion in the medial prefrontal cortex that plays a big part in a person's sense of self. The only part of the brain that shows increased perfusion of rCBF is the parietal operculum regulating emotions in response to pain. This comes forth as emotional dissociation in the traumatic personality state of a DID patient (Reinders et.al., 2003).

Testing the autonomic nervous system through heart rate and skin conductance is an additional test that proves the authenticity of the various alter egos. Nine patients diagnosed with DID were brought in each day for testing. Each patient had three identities involved in the study: the host personality and two other identities. As the researchers hypothesized, each identity showed differing results of heart rate and skin conductance when exposed to specific stimuli. Since the autonomic nervous system is completely voluntary it is safe to assume that the identities are genuinely distinct (Putman et.al., 1990).

Amnesia and Memory Transfer

According to the DMV, DID should be a possible diagnosis when there is an "inability to recall important personal information that is too extensive to be explained by ordinary forgetfulness." Amnesia is the most dominant symptom in a patient with DID and according to the *Journal of Abnormal Psychology* is said to be a psychological disorder as a response to stress or trauma. Furthermore, it's questionable whether inter-identity memory transfers exist. A

study was conducted proving that DID patients only show memory transfer of implicit memories/words, but when there was an emotional or explicit word the patient regressed into an amnesic state. When these memories are restored it's possible to develop dissociative memories in which at times the trauma is remembered and at times the patient has amnesia.

The amnesic periods are only found between different identities and not within one identity. Therefore, in order to get to that amnesic state the identity fluctuates. Accordingly, the psychological perspective to DID is believed to be that DID patients are capable of switching identities in order to forget, and to avoid re-experiencing the trauma (Elzinga et.al., 2003).

Trauma During Childhood

Vermetten et. al. (2006) describe DID as a childhood-onset posttraumatic developmental disorder. The post traumatic model of DID states that when a child goes through the traumatic experience of physical or sexual abuse it is natural that the child will dissociate, mentally compartmentalize, and have amnesic periods as a response. This coping mechanism removes unbearable memories that the child cannot deal with. As the child grows up, each painful experience gets compartmentalized and is expressed as a different identity (Piper, Mersky, 2004). Studies show that childhood trauma is imperative to the development of DID. Traumatic is a relative word and is not referring to a one-time beating. The trauma endured is unusually sadistic abuse and it happens about twice a week for 50 weeks out of the year for 10 years. Statistically the type of abuse that will cause DID is 60-75% physical abuse, and 68-83% sexual abuse (Piper et.al., 2004).

DID specifically only develops when the trauma occurs in childhood because children are not yet equipped with the resources to react. Furthermore, children are more prone to peritraumatic dissociation, and do not personalize the trauma that they are experiencing. DID can be classified as a developmental disorder due to the child not being able to handle dealing with the trauma and it takes a toll in later years. When a normal person deals with stress their mental health is improving. However, when the stress is overbearing it reduces the mental health of a person (Goleman, 1985). Stress in early development biologically embeds itself in a person and has long lasting effects. When a child experiences abuse versus being cared for it will affect his social experiences in later life (Mcewen et.al., 2015).

Hippocampus and Amygdala

Stress related trauma plays a role psychologically and biologically in patients with DID.

Changes in the limbic system have been linked to stress factors (Gulyaeva, 2014). Parts of the limbic system such as the hippocampus and amygdala are two major parts of the brain that are involved in the formation of DID. Patients with DID

have a significantly reduced sized hippocampus and amygdala. The hippocampus and amygdala are located in the medial temporal lobe (Figure 1) and although they have different functions they ultimately influence each other (Phelps, 2004).

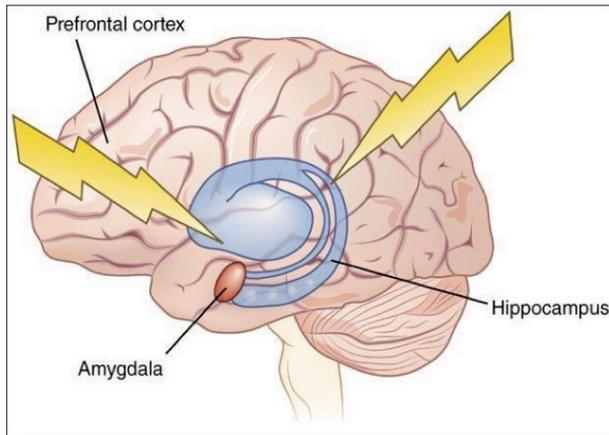


Figure 1: illustration of the hippocampus and amygdala. Notice how the hippocampus and amygdala are neighboring each other. Source: Mechanism of stress in the brain. McEwen, et. al.. (2015)

The hippocampus is the part of the brain that is involved in episodic memory and the primary memory system of the brain. Learning and stress regulation are also part of hippocampal function. The amygdala controls fear conditioning and processes emotion. Although the hippocampus and amygdala reside adjacent to each other, studies introducing a blue box that gives a shock proved that the hippocampus and amygdala have independent functions. The study was performed by introducing a blue box that gives a shock. People with focal lesions on the amygdala understood that the blue box causes a shock but failed to show any sign of fear. Focal lesions to the hippocampus caused one to fear the shock but fail to register that the shock comes from the blue box (Phelps, 2004).

The hippocampus and amygdala are dependent on each other when it comes to emotional memories. The amygdaloid complex consists of the subunits of the amygdala that are difficult to distinguish in MRIs. The amygdaloid complex is divided by function, receives, and relays information involved in fear conditioning and response. The forebrain receives input from the amygdala to stimulate attention, and the brainstem stimulates fear. The hypothalamus receives input from the amygdala for hormone release during stress that eventually affects the hippocampus. Through animal studies researchers were able to see that stress hormones were attacking androgenic receptors on the basolateral amygdala that were affecting hormones in the hippocampus. Further research revealed through fMRI scans that activity of the hippocampus causes less activity of the amygdala and vice versa (Phelps, 2004). When studies were done removing the temporal lobe (which includes both hippocampus and

amygdala), symptoms normally seen in patients with DID, such as amnesia and hypermetamorphosis were present. (Cristinzio et.al., 2007).

Amygdala and Hippocampal Size Difference

The difference in hippocampal volume between a patient with DID and a healthy person is significant. The hippocampus in a patient with DID is 19.2% smaller and the amygdala is 31.6% smaller than that of an average person. In addition to stress causing smaller hippocampal volumes in patients with DID, other disorders such as PTSD that are associated with abuse in childhood show smaller hippocampal volumes, which can be seen on MRI readings. On the contrary, people who experienced childhood abuse and did not develop PTSD or DID, showed a normal sized hippocampus. There is no proven decrease in the size and volume of the amygdala in PTSD patients with no childhood trauma. In contrast, a PTSD patient with record of childhood abuse revealed a 15% decrease in amygdala size (Vermetten et.al., 2006).

Methods

To research the different mechanisms and causes that stress has on DID, search engines such as Touro College Library, Proquest, and Google Scholar were used. Keywords such as “dissociative identity disorder”, “glucocorticoids”, and “hippocampus and amygdala” were used to retrieve appropriate information. Multiple articles and medical journals were found presenting the different perspectives to answer the question above.

Discussion

Not much is known regarding the neurobiology of DID, but the way that stress affects parts of the brain associated with DID is discussed. The parts of the brain affected may be the underlying cause of the symptoms of DID.

The homeostasis of the body is disrupted as a result of stress (Krugers et.al., 2010). Allostasis is the body adapting to stressors through mediators. Mediators can be steroid hormones such as glucocorticoids. Through glucocorticoids the body is able to adjust and adapt to the stress that it is handling. These effects of glucocorticoids can either be helpful for the person’s survival or detrimental to the brain. The brain can be altered through the expansion or retraction of the dendrites or change in synaptic density. Receptors in the brain are important factors in brain alterations (McEwen et.al., 2015),

Glucocorticoids

Glucocorticoids are adrenal steroid hormones that are produced in the zona fasciculata of the adrenal cortex in response to stress. One major class of glucocorticoids is cortisol. Glucocorticoid or cortisol secretion from acute stress is crucial for the survival of a person. Stored energy is retrieved to

Correlation Between Stress and Development of Dissociative Identity Disorder

enable the body to react appropriately to stressors. The harmful effect of glucocorticoids appear in the hippocampus and can take a toll on a person's memory, specifically explicit memory (Sapolsky, 2003).

HPA Axis

A major gland involved in stress regulation is the adrenal gland. Located superior to the kidney, the adrenal gland either reacts to stress in a fight or flight response or supplies energy for the body's response via the HPA axis. The adrenal gland is made up of the adrenal cortex and the adrenal medulla. The stimulation of stress causes the adrenal medulla to secrete epinephrine (adrenaline) and norepinephrine (noradrenaline) so the body can respond in a fight or flight manner. When the stress is continuous the adrenal cortex secretes glucocorticoids or cortisol which mostly bind to proteins. The cortisol that does not bind to proteins exists as free cortisol and supplies the body with the energy it needs to react to the stress. Cortisol acts in different reactions to produce glucose through gluconeogenesis, fatty acids from triglycerides, and amino acids from proteins to create new energy or to repair cells that were damaged by the stress (Hannibal, Bishop 2014).

Hypothalamic pituitary adrenal axis (HPA axis) is a negative feedback mechanism that helps regulate glucocorticoid production in times of stress (figure 2). The reaction begins in the hypothalamus, the link between the nervous and endocrine system. The paraventricular nucleus in the hypothalamus responds to a relative amount of stress and causes the release of corticotropin releasing hormone (CRH). The CRH thus stimulates the anterior pituitary to release adrenocorticotropic hormone (ACTH) that travels through the blood to the adrenal cortex. Glucocorticoids are released and supply energy to the body. When there is an excess of glucocorticoids the hypothalamus and anterior pituitary receive signals to stop the production of CRH and ACTH in order to inhibit the overproduction of glucocorticoids (Zhu et.al., 2014). However, this negative feedback mechanism may be disrupted. Only a small amount of stress is required to activate the HPA axis, but when there is too much stress it is possible for the HPA axis to become hyperactive.

HPA Axis Hyperactivity

Hyperactivity of the HPA axis can be caused by many factors, including excess glucocorticoids. Excess glucocorticoids in the brain can be attributed to receptor changes. Two different glucocorticoid receptors in the brain are: glucocorticoid receptors (GR) and mineralocorticoid receptors (MR). GR and MR are found on both the hippocampus and the hypothalamus. MR are more likely to attract glucocorticoids even when glucocorticoid levels are low. GR only receive glucocorticoids when there is an excess amount of glucocorticoids from stress (Krugers et.al., 2010). If there is a decrease in GR in the hippocampus it can

help cause the hyperactivity of the HPA axis (Zhu et.al., 2014).

A study using metyrapone was conducted on rats to determine the correlation between glucocorticoids and HPA axis hyperactivity that causes depressive behaviors. Enzyme 11- β -hydroxylase, found in the adrenal cortex, is responsible to convert

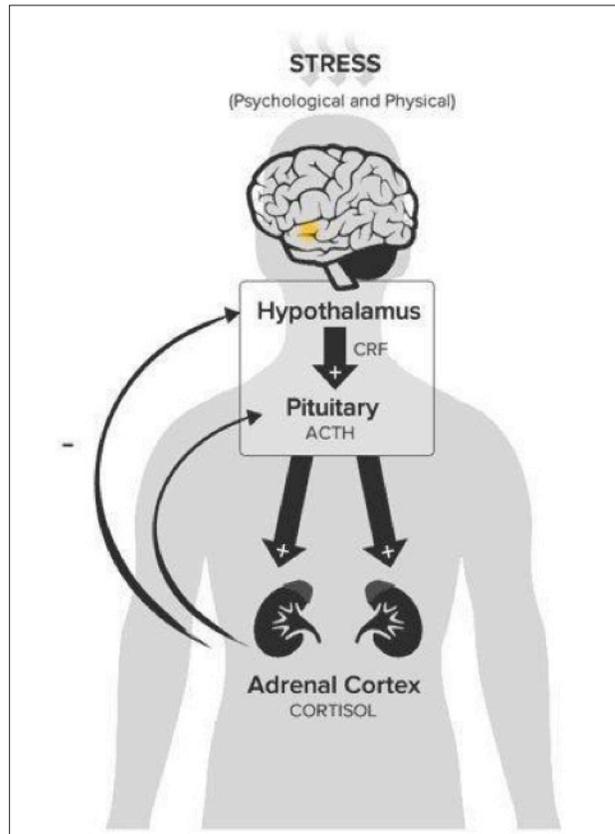


Figure 2: A clear chart of the HPA axis. The arrows with a positive depicts stimulation of hormone release. The arrows with a negative sign depicts the inhibition of hormone release. Source: <https://www.integrativepro.com/Resources/Integrative-Blog/2016/The-HPA-Axis>

deoxycorticosterone to glucocorticoids. Metyrapone, is used to block the function of 11- β -hydroxylase therefore terminating the process which would result in hyperactivity and depressive behaviors (Zhu et.al., 2014).

MR-NNOS Pathway-Hyperactivity of HPA Axis

An additional pathway that decreases the activity of GR in the hippocampus is the MR-nNOS pathway. Glucocorticoids react with neuronal nitric oxide synthesis enzyme (nNos), causing the overproduction of nitric oxide (NO) in the hippocampus. The GR in the hippocampus, which is crucial for the HPA axis to maintain homeostasis, interacts with the NO causing the negative feedback mechanism to be disrupted. The relatively decreased amount of GR in the hippocampus explains why excess glucocorticoid exposure would generate hyperactivity of the HPA axis.

The reason that the hypothalamus is not involved with the hyperactivity of the HPA axis is because of the low amount of MR in the hypothalamus. Thus, the MR-nNOS-NO pathway is non-existent in the hypothalamus and the hypothalamus is only capable of activating the negative feedback of the HPA axis (Zhu et.al., 2014).

Different Pathways of Glucocorticoids

There are two effects that glucocorticoids can have: genomic or nongenomic. A non-genomic effect is a transcription independent mechanism that can interact with a cell membrane specifically or nonspecifically. The genomic effect works through interaction with Glucocorticoid Response Element directly or indirectly. Direct interaction is through receptors themselves and indirect interaction is through other transcription factors. Direct interaction of glucocorticoids is responsible for glutamate secretion. Indirect interaction of glucocorticoids uses endocannabinoids to control glutamate and GABA (Mcewen et.al., 2015).

NMDA and AMPA Receptors

One type of glutamate receptor is N-methyl-d-aspartic acid (NMDA) receptor found on the hippocampus. NMDAR is activated by glutamate with glycine and when activated permits charged ions to travel through the cell membrane (Mcewen et.al., 2015). Studies show that ketamine, an NMDA antagonist, causes the same symptoms seen in DID: amnesia, out of body experiences, and time standing still (Vermetten et.al., 2006). Alpha-amino-3-hydroxyl-5-methyl-4-isoxazole-propionate receptor (AMPA) or quisqualate receptor is a transmembrane receptor. AMPAR only requires glutamate to activate the ion channel. Recent research show that activated glucocorticoids regulate AMPAR and effect learning and memory in the hippocampus. Learning and memory are disrupted by the synaptic connectivity in the hippocampus. Plasticity of the brain is in the control of the pre- and postsynaptic sites, and can be controlled by either limiting the neurotransmitters or the neurotransmitter receptors at the pre and postsynaptic sites, respectively. Further research proves that AMPAR also causes changes in synaptic function and plasticity of the brain. Miniature excitatory postsynaptic currents (mEPSC) are used to measure the strength of a synapse. When the nongenomic effect (fast) on AMPARs was measured the increase in the mEPSC of the hippocampus and amygdala were similar to those experienced upon glucocorticoid exposure. The genomic effect (slower) of AMPARs is detrimental to the function of neurons (Krugers et.al., 2010).

Brain Plasticity

Brain plasticity is caused by the conjunction of glucocorticoids and amino acids.

Dendrites are altered in the way of expansion or retraction, and increase or decrease of dendritic synapses. For example,

CRH, released in the hippocampus, down regulates thin spines. Parts of brain affected by hormones and intra and extracellular mediators are the hippocampus, amygdala and prefrontal cortex (Mcewen et.al., 2015).

Studies show that chronic stress leads to permanent alterations to the brain. Regardless if the chronic stress subsides, the brain creates a new way to handle stress and hippocampal changes remain. Additionally, experiments with rats prove gene alterations due to stress, including delayed alterations. The delay in gene response comes from epigenetic regulators. Even though the dendrites can regrow they are different in that the dendrites grow more proximal to the cell body (Mcewen et.al., 2016).

The neuroendocrine model was postulated regarding the effects that stress has on the hippocampus and the role of the amygdala in hippocampal plasticity. It was proven that hippocampal plasticity can be achieved through limiting input from the amygdala.

A part of the hippocampus, the dentate gyrus, is known for its constant rearrangement and its reproduction of its cells to replace lost cells. Reproduction of the new cells is inhibited by the amount of adrenal hormones in the brain through a mechanism involving NMDA receptors. Stress exposure results in glucocorticoid production and the release of glutamate from the hippocampus which blocks the reproduction of neurons in the dentate gyrus. Only chronic stress has effect on proliferation in the dentate gyrus, because small amounts of stress were seen to have no effect on neurogenesis in the dentate gyrus.

In acute stress the new cells that are produced take part in stress response. When there is an excess amount of glucocorticoids caused by chronic stress the dendrites in the hippocampus retract and do not partake in stress response (Gulyaeva, 2014).

Hippocampal and Amygdalar Role in Memory

Both the hippocampus and the amygdala contain GR and MR. As a result of stress, the role of the amygdala in fear conditioning is altered. According to Sapolsky (2016) when the hippocampus is affected by stress the amygdala will start to process the memories. For example, amygdala long term potentiation will substitute the hippocampal long-term processing. Studies show that when hippocampal dendritic processes go through atrophy it causes the extension of the amygdalar processes'. The mechanism as to why this happens is currently unknown. Despite the fact that the hippocampus is irrelevant in memory processing, the hippocampus is said to process explicit memories. The reason is that the hippocampus and the amygdala work together to process the memories. Since the hippocampus cannot process the memories normally and neutral it acts like the amygdala and processes the memories inaccurately, causing flashback-like memories. The mechanism is controlled by the sympathetic nervous system and is activated by stress. Stress causes catecholamines to be released by the adrenal medulla and stimulates

Correlation Between Stress and Development of Dissociative Identity Disorder

the vagus nerve. Thus, the vagus nerve stimulates the nucleus of the tractus solitarius (NTS) to have an effect on the amygdala. The amygdala begins to play a part in memory processing from the glucocorticoids in the hippocampus, amygdala and the NTS (Phelps, 2004).

Animal studies show that by exposing glucocorticoids to the hippocampus it causes a decrease of pyramidal neurons and dendritic branching that are used to integrate memories (Nijenhuis, Steele, 2010).

Genetic Factors

Genetics are very influential in the development of any disorder. People that are born with a smaller hippocampal or amygdalar volumes have been seen to be more prone to developing DID (Vermetten et.al., 2006). When one is born with an overabundance of glucocorticoid receptors, there is an increased probability to develop mood disorders and are more responsive to antidepressant drugs. The opposite effect is on those who are born with less GR than an average person (McEwen et.al., 2016).

It is more common for a male to develop DID than a female. The hippocampus in a male naturally becomes reduced 1-1.5% per year while the amygdala is unchanged. Female resistance to this reduction is attributed to the production of estrogen which somehow acts as a prevention (Vermetten et.al., 2006).

Conclusion

It is clear how much damage stress can do in terms of brain plasticity. Through mechanisms of how stress affects the brain it can be understood how DID can possibly develop. Treatment regarding DID is not proven to be 100% efficient or effective, but it may improve and make the disorder livable. Therapists dealing with DID patients will try to bring forth all their identities and help the patient deal with the trauma they experienced that sparked this disorder. As seen in the Three Faces of Eve, therapists may attempt for years to merge all the personalities together in order to diminish symptoms of dissociation. Cognitive behavior therapists introduce the patient to various coping mechanisms as an alternative to switching identities. One form of treatment that is not effective is to diminish personality states that are not positive or that may be detrimental to the environment. Since those identities exist to protect the patient from abuse it should be incorporated appropriately into their comprehensive personality (Gillig, 2009). Antidepressants may be another form of treatment for DID because in rodents neurogenesis of hippocampus was induced by them (Vermetten et.al., 2006). Recent studies show that the drug paroxetine may be useful in treating DID. Paroxetine has proven to regenerate hippocampal growth by 4.6% and to limit the symptoms of DID and PTSD. The correlation between DID and PTSD has shown to be very similar in neurobiology and causation of the

disorders (Nijenhuis et.al., 2010). So many factors are involved in the development of DID, thus making the disorder very rare. The treatments noted are just a step in the direction to help understand the disorder and to further the subjects of research on DID. Even though it is understood how stress causes plasticity to the hippocampus and amygdala creating symptoms involved in DID, there is still so much about DID that is unknown to the world and is a mystery till today.

References

- Cristinzio Perrin, C., & Vuilleumier, P. (2007). The role of amygdala in emotional and social functions: Implications for temporal lobe epilepsy. *Epileptologie*, 24, 78-89. Retrieved from <https://archive-ouverte.unige.ch/unige:44299>
- Elzinga, B. M., Phaf, R. H., & Ardon, A. M. (2003). Directed Forgetting Between, But Not Within, Dissociative Personality States. *Journal of Abnormal Psychology*, 112(2), 237-243.
- Gillig PM. Dissociative Identity Disorder: A Controversial Diagnosis. Gillig PM, ed. *Psychiatry (Edgmont)*. 2009;6(3):24-29
- Goleman, D. New focus on multiple personality. *New York Times* (1923-Current file). May 21 1985:2. Available from: <https://search.proquest.com/docview/11176836?accountid=14375>
- Gulyaeva NV. Effects of stress factors on the functioning of the adult hippocampus: Molecular-cellular mechanisms and the dorsoventral gradient. *Neurosci Behav Physiol*. 2014;44(9):973-981. <https://search.proquest.com/docview/1711187208?accountid=14375>. doi: <http://dx.doi.org/10.1007/s11055-014-0012-5>.
- Hannibal KE, Bishop MD. Chronic stress, cortisol dysfunction, and pain: A psychoneuroendocrine rationale for stress management in pain rehabilitation. *Phys Ther*. 2014;94(12):1816-25. <https://search.proquest.com/docview/1635218867?accountid=14375>.
- Krugers HJ, Hoogenraad CC, Groc L. Stress hormones and AMPA receptor trafficking in synaptic plasticity and memory. *Nature Reviews Neuroscience*. 2010;11(10):675-81. <https://search.proquest.com/docview/751840867?accountid=14375>. doi: <http://dx.doi.org/10.1038/nrn2913>.
- Lehman KJ. Woman, divided: Gender, family, and multiple personalities in media. *The Journal of American Culture*. 2014;37(1):64-73. <https://search.proquest.com/docview/1561135304?accountid=14375>.
- Leiber, A. Multiple personality disorder (dissociative identity disorder). *Psychom*. 2016. <https://www.psychom.net/mchugh.html>
- McEwen BS, Bowles NP, Gray JD, et al. Mechanisms of stress in the brain. *Nature neuroscience*. 2015;18(10):1353-1363. doi: 10.1038/nn.4086. <http://dx.doi.org/10.1371/journal.pone.0097689>.
- Nijenhuis E, van dH, Steele K. TRAUMA-RELATED

STRUCTURAL DISSOCIATION OF THE PERSONALITY. *Act Nerv Super.* 2010;52(1):1-23.

<https://search.proquest.com/docview/603596641?accountid=14375>.

Phelps, E.A. (2004). Human emotion and memory: interactions of the amygdala and hippocampal complex. *Current opinion in neurobiology*, 14(2), 198-202.

Piper A, Merskey H. The persistence of folly: A critical examination of dissociative identity disorder. part I. the excesses of an improbable concept. *Canadian Journal of Psychiatry*. 2004;49(9):592-600. <https://search.proquest.com/docview/222846787?accountid=14375>.

Putnam FW, Zahn TP, Post RM. Differential autonomic nervous system activity in multiple personality disorder. *Psychiatry Res.* 1990;31(3):251-260.

<https://search.proquest.com/docview/85266816?accountid=14375>.

Reinders A, Nijenhuis E, Paans A, Korf J, Willemsen A, den Boer J. One brain, two selves.

Neuroimage. 2003;20(4):2119-2125.

<https://search.proquest.com/docview/1506603948?accountid=14375>. doi:

<http://dx.doi.org/10.1016/j.neuroimage.2003.08.021>.

Sapolsky RM. Stress and plasticity in the limbic system*. *Neurochem Res.*

2003;28(11):1735-42. <https://search.proquest.com/docview/221777581?accountid=14375>.

Seligman R, Kirmayer LJ. Dissociative experience and cultural neuroscience: Narrative, metaphor and mechanism. *Cult Med Psychiatry*. 2008;32(1):31-64. <https://search.proquest.com/docview/229984055?accountid=14375>. doi: <http://dx.doi.org/10.1007/s11013-007-9077-8>.

Strasburger H, Waldvogel B. Sight and blindness in the same person: Gating in the visual system. *PsyCh Journal*. 2015;4(4):178-185. <https://search.proquest.com/docview/1748051616?accountid=14375>. doi:

<http://dx.doi.org/10.1002/pchj.109>.

Van der Kruijs S.J.M., Bodde NMG, Carrette E, et al. Neurophysiological correlates of dissociative symptoms. *Journal of Neurology, Neurosurgery and Psychiatry*. 2014;85(2):174. <https://search.proquest.com/docview/1781249454?accountid=14375>. doi: <http://dx.doi.org/10.1136/jnnp-2012-302905>.

Vermetten E, Schmahl C, Lindner S, Loewenstein RJ, Bremner JD. Hippocampal and amygdalar volumes in dissociative identity disorder. *Am J Psychiatry*. 2006;163(4):630-6. <https://search.proquest.com/docview/220509428?accountid=14375>.

Zhu L, Liu M, Li H, et al. The different roles of glucocorticoids in the hippocampus and hypothalamus in chronic stress-induced HPA axis hyperactivity. *PLoS One*. 2014;9(5). <https://search.proquest.com/docview/1524876202?accountid=14375>. doi: