
From the Instructor

A key hypothesis tested in this course is that all psychological behaviors are entailed by neurological processes. In the case of Dissociative Identity Disorder (DID), in which the host self breaks into one or more distinct alters, is it the trauma of early abuse and resultant stress that ultimately modifies the brain's structure, or are there also psychological processes at work? Why don't all traumatized children develop DID? These are the kinds of questions Sean's detailed and complex hypothesis aims to investigate.

It is clear from the first pages of his paper that Sean is most persuaded by the neurobiological evidence. He has read the journal articles closely, so his summaries are focused, logical, and detailed. He can draw his own convincing conclusions from the research in support of the analysis he is conducting. I especially like the clarity of his scientific prose. He defines key terms quickly and exactly and describes essential processes in immediately accessible language so that he never loses his reader.

Early versions of this paper developed the possible role of the Orbital Frontal Cortex in the expression of typical DID behavior, but those drafts lacked a trigger to show how and why the dissociation occurred. It was Sean's discovery of the more psychological attachment theory that made all the pieces fall together. Sean's logical organization of the paper creates a sense of continuous discovery, of having many of the questions posed by the biology very closely answered by the psychology. This combination of deep research and original speculation make the paper a real pleasure to read again half a year later.

— Stephen Scheurman

SEAN MANTON

Prize Essay Winner

DISORGANIZED ATTACHMENT AND THE ORBITOFRONTAL CORTEX AS THE BASIS FOR THE DEVELOPMENT OF DISSOCIATIVE IDENTITY DISORDER

Dissociative identity disorder (DID), formerly known as multiple personality disorder, is perhaps one of the most well-known and extreme psychological disorders. While DID has been extensively studied, a clear cause is still undetermined. Childhood trauma has been identified as an important factor; however, it is not sufficient on its own to explain the roots of DID. One particularly promising theory posits that, in addition to traumagenic origins, infant disorganized attachment may be a significant contributor to the development of DID. Neuroimaging studies have identified areas of the brain, the orbitofrontal cortex in particular, that function differently in DID patients, thus providing a neurobiological basis for the disorder. By examining the effects of trauma on neurodevelopment, some of the differences between the normal and the DID brain can be accounted for. Attachment theory allows the cause of DID to be traced even further back to neurodevelopment that occurs during infancy. The combination of disorganized attachment with later childhood trauma provides a strong basis for the development of DID.

Neuroimaging, in its many forms, can provide structural and functional information about the brain and is a powerful tool in understanding neurobiology. Thus, neuroimaging studies pave the way in the search for a neurobiological understanding of DID. Several promising studies have been performed that imaged the DID brain. Vermetten, et al. used MRI to compare the brain structure of female patients with DID to healthy subjects and found that the hippocampus and amygdala of the DID patients were significantly smaller (19.2% and 31.6%, respectively) (630). Such a significant difference in brain structure would imply that the hippocampus

and amygdala are key in understanding DID, which seems to make good sense given the hippocampus's role in forming long-term memories and the amygdala's in regulating emotion. Irregularities in these brain areas would, thus, help account for the variance of memory and emotions among the different alters present in DID.

Unfortunately, the results of Vermetten's study were largely discredited by another study, which used MRI to compare subjects with post-traumatic stress disorder (PTSD) to healthy subjects, as well as subjects with dissociative amnesia (DA) or DID but not PTSD. This study found similar results in terms of reduced hippocampal and amygdalar volume in PTSD patients, but found that there was no significant difference between the DA/DID patients and normal subjects (Weniger, et al. 281). Vermetten recognized the possibility of such a result, stating that "a potential limitation of this [Vermetten's own] study is that all of the patients with dissociative identity disorder also met the criteria for PTSD, which makes it impossible to establish that the findings are not related to the comorbid PTSD diagnosis" (634). However, he also points out that "patients with true dissociative identity disorder without PTSD essentially do not exist" (635). Seeing as Weniger's study managed to find patients with DID but not PTSD, Vermetten's statement would appear to be an exaggeration. Nonetheless, it is true that DID is often, if not in the majority of cases, accompanied with comorbid PTSD. Thus, although Vermetten's findings cannot shed much light on the specific neural basis of DID, his study does have potential to provide insight into some of the symptoms of many DID patients.

While there may not be such a clearly identifiable structural difference in DID patients, functional imaging studies (those that measure the amount of brain activity) have produced some valuable results. Two studies of interest measured regional cerebral blood flow (rCBF) as a way of inferring relative activity in different areas of the brain. One study compared rCBF of DID patients while they were in their host personality with normal controls and observed lower rCBF in the orbitofrontal cortex (OFC) of the DID subjects (Sar, et al. 219–20). The orbitofrontal cortex is thought to be involved in decision-making. Thus, Sar hypothesizes that the decreased functioning of the OFC results in impulsivity and that the

switch to an alter personality may represent a drastic expression of impulsive behavior caused by cognitive and emotional conflicts (222).

The second study, conducted by Reinders, et al., measured rCBF of DID subjects in a neutral personality state (NPS) compared with a traumatic personality state (TPS) while they listened to a memory script. The procedure is described: “Subjects listened to two autobiographical audiotaped memory scripts involving a neutral and a trauma-related experience. The neutral memory script was regarded as a personal experience by both personality states. However, only the TPS experienced the trauma-related script as personally relevant” (2120). The study discovered no difference between the NPS and TPS when listening to the neutral script, as well as no difference between the neutral and traumatic scripts for the NPS. Comparing the NPS and TPS rCBF when listening to the traumatic script, though, the study found a deactivation pattern of brain areas in the NPS. This pattern matches the deactivation pattern found in studies of normal subjects when recalling non-autobiographical memories as opposed to autobiographical memories. Thus, one conclusion of the study is that, on a neurobiological level, the alters in DID do in fact have different autobiographical selves. Furthermore, among the brain areas that were deactivated in the NPS versus the TPS is the medial prefrontal cortex (2122–3). This is a significant finding, as the prefrontal cortex is involved in personality expression and also contains the orbitofrontal cortex. These two studies both implicate the orbitofrontal cortex as a key to understanding DID.

The function of the orbitofrontal cortex can be summarized very simply as decision-making. However, to leave it at that would be a gross understatement of the functions it actually performs. A more adequate description, provided by Rhawn Joseph, is that the OFC is the “senior executive of the emotional brain” (qtd. in Schore 29). An examination of the complex workings of the OFC will illuminate the degree to which it can be seen as the crucial element in DID. Schore summarizes the results of several studies into the functions of the OFC:

Indeed, this prefrontal system [the OFC] appraises visual facial information (Scalaidhe, Wilson, & Goldman-Rakic, 1997), and processes responses to pleasant touch, taste, smell (Francis, D., *et al.*, 1999) and music

(Blood, Zatorre, Bermudez, & Evans, 1999) as well as to unpleasant images of angry and sad faces (Blair, Morris, Frith, Perrett, & Dolan, 1999). But this system is also involved in the regulation of the body state and reflects changes taking place in that state (Luria, 1980). (30)

Antonio Damasio posits in his model of consciousness that the development of a notion of self arises from the brain's second order mapping of the relation between "objects" and the organism (169–70). Within this model of consciousness, the OFC, with its functions in both emotional processing of sensory information as well as homeostasis and the mapping of the body, would seem to be a critical component in the generation of a self. Thus, it is quite plausible that an abnormally functioning OFC could lead to the generation of multiple selves. Furthermore, Schore states:

The functioning of the orbitofrontal control system in the regulation of emotion (Baker, Frith & Dolan, 1997) and in "acquiring very specific forms of knowledge for regulating interpersonal and social behavior" (Dolan, 1999, p. 928) is central to self-regulation, the ability to flexibly regulate emotional states through interactions with other humans—that is, interactive regulation in interconnected contexts—and without other humans—that is, autoregulation in autonomous contexts. (33)

What Schore terms here as "self-regulation" seems to describe the concept of a unified personality. Hence, it seems that abnormal functioning of the OFC serves as a very solid neurobiological basis for the development of DID. With this basis in mind, it is easier to identify a neurodevelopmental cause of DID. Complementarily, by examining the neurodevelopmental aspects of DID and the OFC, the role of the OFC in DID will be further solidified.

Generally, childhood trauma has been implicated as the causative factor in DID. It is well documented that traumatic experiences are disruptive to normal development in children. Heide reviews the findings of several studies to identify a broad spectrum of negative effects caused by trauma on childhood development. These include that childhood trauma can lead to long-term changes in the brain through negative effects on

areas of the brain such as the limbic system (which contains the amygdala and hippocampus) as well as on neurotransmitters, and that traumatic experiences can cause dissociation of mind and body (224–8). Childhood trauma certainly has enough strong neurodevelopmental consequences that it would seem the likely candidate in the explanation of the origins of DID. A study by Briere of 618 subjects who responded to the Multiscale Dissociative Inventory (MDI) and the Detailed Assessment of Posttraumatic Stress found that 90% of subjects with clinically elevated scores on MDI scales also reported a trauma history. However, the subjects with clinically elevated scores represented only 8% of the subjects who reported a trauma history (80). While Briere's study confirms that trauma is a unifying factor in dissociative disorders, the fact that only a small group of subjects with trauma develop DID or any sort of dissociative disorder means that there must be additional factors that play into the development of DID.

Attachment theory provides another perspective and traces the origins of DID further back to attachment behaviors in infancy through a psychological model hypothesized by Giovanni Liotti. Attachment theory posits that an infant's development of attachment to its caregiver, usually its parent(s), plays a large role in the development of its personality and later social behaviors. Parenting style is the main factor that leads to different patterns of attachment behavior. These patterns are classified based upon the infant's behavior around its parent, especially when the parent leaves or returns. Most infants exhibit secure attachment. They will cry when their parent leaves them and are comforted when their parent returns. Secure attachment forms as a result of the parent consistently responding to the infant's needs. On the other hand, neglectful parenting tends to lead to insecure attachment, in which the infant either behaves indifferently to the parent or is upset when the parent leaves but continues to be upset at the parent even on his or her return. Lastly, of interest to DID is disorganized attachment, in which the infant displays conflicting or disoriented behaviors around the caregiver. Main and Hesse found that a combination of frightening and frightened behavior on the part of the caregiver may result in disorganized attachment in an infant. If the caregiver displays frightening or abusive behavior, the infant is faced with the paradox of the caregiver as a source both of safety and of danger. If

the caregiver appears frightened, the infant may be led to believe there is a threat nearby, or that the caregiver is frightened of the infant. Main and Hesse hypothesize that disorganized attachment is, thus, accompanied by the development of conflicting models of self within the infant. Frightening behavior by the caregiver will lead the infant to view itself as helpless and vulnerable. Frightened behavior will lead the infant to view itself as threatening. In some situations, the infant may also develop a model of self where it sees itself as a caregiver (qtd. by Liotti 198–199).

Liotti builds upon the work of Main and Hesse by hypothesizing that the conflicting models of self that are developed within an infant with disorganized attachment create the risk for the later development of DID. He proposes different possible outcomes for such an infant. If later experiences favor one model of the self, it may eventually be selected over the others. On the other hand, multiple models of the self may continue to develop, disposing the child towards dissociation as a way of handling stress. If the child then experiences trauma, she will dissociate as a defensive mechanism and may begin to use one of the models of self to develop an alter (198–201). Thus, Liotti's model provides a strong possibility that the development of DID will be linked to disorganized attachment during infancy.

Liotti's model is supported by the findings of a study by Ogawa, et al., which measured the dissociative symptomology of 168 18- to 19-year-old subjects four times across 19 years and found that disorganized attachment during infancy was a strong predictor of later dissociative symptoms (860, 874). However, even more convincing is Schore's statement that "attachment experiences, face-to-face transactions of affect synchrony between caregiver and infant, directly influence the imprinting, the circuit wiring of the orbital prefrontal cortex" (30). Thus, a neurodevelopmental link exists between attachment and the OFC, solidifying the case for both as bases for DID. The conflicting attachment experiences endured by an infant with disorganized attachment would lead to irregular development of the OFC, which would mirror the development of the conflicting models of self. Later childhood trauma would further affect the brain's development and, thus, allow for the dramatically different neurological functioning of patients with DID. Liotti's attachment theory model of DID provides an intuitive understanding for how DID arises from a psy-

chological perspective, which, combined with the neurobiological perspective provided by neuroimaging studies, creates a very complete foundation for understanding the causes of DID.

The attachment-OFC model has some important implications. Lakatos states that disorganized attachment is displayed by 80% of infants who have been maltreated, but also in 15% of infants in low-social-risk populations (633). This indicates that while poor parenting or maltreatment is the primary cause of disorganized attachment, other factors also play a role. Lakatos performed a study in which DNA samples were taken from one-year-old infants who were classified as disorganized or non-disorganized. The study found that infants with a 7-repeat allele in the dopamine D4 receptor (DRD4) gene were four times as likely to display disorganized attachment (633–4). Thus, the attachment model reveals a genetic risk factor for developing DID.

The attachment model also serves to resolve a long-term debate about DID. Due to its almost unbelievable symptoms, DID has always been a controversial diagnosis. Many skeptics argue that DID is iatrogenic rather than traumagenic in origin. That is, they believe that DID is the result of the suggestive influence of a therapist rather than a legitimate disorder. Proponents of this iatrogenic model of DID believe that patients' alters are not truly present before therapy, but appear during therapy as a result of a therapist suggesting to the patient that he has an alter. Furthermore, they question the link between childhood trauma and DID, suggesting that the therapist implants false memories of trauma. These concerns are not unreasonable; the DSM-IV states that many DID patients also score highly on hypnotizability scales (American Psychiatric Association). Thus, it seems possible that the suggestive power of a therapist might lead an easily hypnotizable patient to believe he has DID and memories of childhood trauma and, thus, display the symptoms. However, while some cases of DID might have iatrogenic origins, the attachment-OFC model provides an undeniable neurobiological basis for traumagenic origins of DID, thus refuting the possibility of a purely iatrogenic model of DID. Those who continue to support the iatrogenic model would simply seem to be in denial of the amazing neurodevelopmental capabilities of the brain.

Much remains to be understood about this complex and fascinating disorder. However, the attachment-OFC model of DID offers a very

promising groundwork to unravel these mysteries as well as improve the treatment strategies and provide insight into the nature of human consciousness in general. Understanding DID from an attachment perspective provides a different perspective from which to approach psychotherapy treatments for DID patients. In addition to traditional methods of treatment, such as encouraging the patient to consolidate his personalities, therapies that address the patient's attachment patterns may prove useful. The fact that the extraordinary symptoms of DID can be traced to clear neurobiological and neurodevelopmental origins is an undeniable example of the amazing potential of the brain's neuroplasticity. While the expression of this potential is often quite tragic for those who suffer from the negative symptoms of DID, it is also this potential that makes all the wonders of human consciousness possible. Additionally, DID sheds light on the importance of the integration of the entire brain in the production of consciousness. The irregular functioning of the OFC leading to DID is an extreme case but it is illustrative of how differences in even one brain area can lead to dramatic changes in the manifestation of consciousness as a whole. For those who suffer from DID, it is a very unfortunate occurrence; however, the ability to study these patients is a great gift to the furthering of cognitive neuroscience. Continuing to build upon the attachment-OFC model of DID will lead towards a better future for the treatment and prevention of DID and the cracking of one of the greatest mysteries known to man—that of his own mind.

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WORK CONSULTED

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