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Existence in a Shambles:
Examining the Curious Case of Depersonalization Disorder

One night during my adolescence will be perpetually engrained in my memory. The evening had passed and I found myself lying in bed, wide-eyed and full of energy, even as the clock crept steadily toward the wee hours of the night. As I basked in the silent darkness, my mind wandered from the petty topics of the day to the deeper existential questions of life. "Who am I?" I asked myself. "Why am I here? Is any of this real?" Suddenly, as my gaze drifted around the room, the objects in my field of vision began to blur. The desk to my left slowly grew in size and the chair to my right seemed to shift its shape. I was completely perplexed. In a rush of panic, I quickly sat up, closed my eyes, and forced myself back to reality. As I opened my eyes, the world was as it was just minutes before. Could this experience have been merely a dream? It is impossible to say. Whatever the nature of the experience had been, it had given rise to very real feelings of depersonalization and derealization, two all too common but seldom recognized psychological phenomena that form the basis of the psychopathology known as depersonalization disorder. In the following analysis, I intend to examine this disorder in terms of clinical criteria for diagnosis, the conceptual understanding of depersonalization and derealization, the prevalence of these phenomena and their pathological form, and the grounding of these phenomena in various traumatic and biological factors.

The DSM-IV-TR (American Psychiatric Association, 2000) outlines several criteria for the diagnosis of depersonalization disorder. First, for the diagnosis to be made, an individual must be suffering from persistent and recurring depersonalization and/or derealization symptoms. Furthermore, these symptoms must cause marked distress and/or impairment in normal functioning. Also, the symptoms previously described must not occur exclusively in the context of another general medical or psychiatric condition. Finally, and most importantly, the individual must retain intact reality testing. In other words, the individual does not believe that he, she, or the external world is *literally* unreal. Rather, the individual feels *as if* he, she, or the external world is unreal. Therefore, it cannot be said that a genuine sufferer of depersonalization disorder is psychotic. However, as previously mentioned, it is common that those experiencing depersonalization and/or derealization symptoms feel like they are becoming psychotic. Upon viewing these criteria for diagnosis, a singular question comes to mind: How can depersonalization and derealization symptoms be characterized?

Depersonalization and derealization, in the context of depersonalization disorder, can be seen as essentially manifestations of psychological dissociation in which an individual experiences a subjective feeling of estrangement, detachment, and/or disconnection from the self (Simeon & Abugel, 2006). Depersonalization

and derealization symptoms reflect a sense of unreality with either the self or the outside world. The term "unreal" can be quite troublesome unless adequately defined. Radovic and Radovic (2002) formulated three distinct definitions of unreality. First, one might imagine that, in the case of imaginary friends for example, certain things are unreal in the sense of not existing at all. However, for something to be unreal it does not necessarily have to lack existence entirely. Unreality can take the form of artificiality, as in the case of children's toys. For example, a child's stuffed elephant can be considered unreal in the sense that it is merely a substitute or representative of the real thing. Finally, something may be considered unreal in the sense that it is atypical or untrue. This final definition seems to represent the quintessence of unreality as experienced in depersonalization and derealization. Aside from this core experience of unreality, various other symptoms are commonly seen in depersonalization and derealization as well as the more pathological depersonalization disorder.

First and foremost, emotional numbing is by far the most common symptom of depersonalization disorder (Simeon & Abugel, 2006). As will be subsequently examined, numerous physiological antecedents to this hypoemotionality have been discovered. Additionally, the broad spectrum of depersonalization symptomatology includes the following: the sensation of being an outside observer of oneself, feeling like an automaton or a machine (in other words, feeling as if one is not in control of one's actions), feeling as if life is being experienced merely as a dream or a movie, changes in the subjective experiencing of time and/or space, a heightened sense of self-observation (leading to obsessions and ruminations), the inability to focus and/or sustain attention, and finally an overall sense of mind emptiness (Simeon & Abugel, 2006). As seen earlier, depersonalization and derealization often go hand in hand. But what exactly is derealization? Derealization, unlike depersonalization, involves the observable world outside of the self and can be described as the sense that the world and objects (oftentimes including other people) within it seem strange or unreal. Harkening back to my personal experience of derealization, objects can inexplicably appear to change in size and shape. Macropsia – objects appearing to enlarge – and micropsia – objects appearing to shrink – are distortions in the subjective experiencing of reality that are often so convincing that an individual may begin to panic and feel as if he or she is losing touch with reality entirely (Simeon & Abugel, 2006). In the context of these many varied symptoms and the aforementioned criteria for diagnosis, one might wish to elucidate the prevalence of depersonalization disorder.

Surprisingly, depersonalization disorder is not as uncommon as one might imagine. In population-based community surveys as well as surveys of clinical (in-patient) and non-clinical samples, researchers have estimated that of the general population, from approximately one to two percent of individuals

currently fit the diagnostic criteria for depersonalization disorder (Hunter, Sierra, & David, 2004). Taken in context, this statistic is astonishing, seeing that roughly the same percentage of individuals from the general population currently fit the criteria for a diagnosis of obsessive-compulsive disorder, a much more widely known and studied pathology. The percentages of depersonalization disorder are found to increase to range from one to sixteen percent in psychiatric in-patient samples. Furthermore, over the course of the average individual's lifetime, he or she has approximately a twenty-six to seventy-four percent chance of experiencing at least one episode of transient depersonalization and/or derealization. Finally, the experiencing of transient depersonalization and/or derealization during or after a traumatic event ranges in prevalence from thirty-one to sixty-six percent (Hunter, Sierra, & David, 2004). What distinguishes these transient episodes of depersonalization and/or derealization from depersonalization disorder rests with the clinical criteria for diagnosis. Most notably, these episodes must cause marked *distress* and/or *functional impairment* in everyday life (American Psychiatric Association, 2000).

Continuing with the analysis, it is imperative to examine the high percentage of experiences of transient depersonalization and/or derealization during or after a traumatic event. Though the specific relationship between trauma and depersonalization has been largely unexamined, a study by one of the foremost researchers in the field has shown that childhood interpersonal trauma is highly predictive of depersonalization disorder. Simeon and colleagues (Simeon, Guralnik, Schmeidler, Sirof, & Knutelska, 2001) found that this childhood interpersonal trauma primarily takes the form of emotional abuse (rather than physical or sexual abuse) in the development of depersonalization disorder. These researchers define emotional abuse by breaking down the concept into five distinct categories: rejection/degradation, terror, exploitation/corruption, denial of emotional responsiveness, and isolation. Though interpersonal trauma can be seen as an antecedent to the development of pathological depersonalization and derealization symptoms, it is important to distinguish trauma as it relates to depersonalization disorder and trauma as it relates to the more commonly known post-traumatic stress disorder (PTSD).

To be clear, a diagnosis of depersonalization disorder according to the DSM-IV-TR, unlike PTSD, does not *require* that the individual experienced a traumatic incident that has brought on the symptom profile in question. Furthermore, PTSD involves the persistent re-experiencing of the traumatic event in the form of flashback memories or nightmares, for example. Additionally, unlike depersonalization disorder, those suffering with PTSD typically experience a great psychological need to avoid places, people, or things that remind them of the traumatic event. PTSD also includes symptoms of hyper-arousal (increased startle response, hyper-vigilance, etc.) not characteristic of depersonalization

disorder. Finally, depersonalization disorder, even when directly associated with traumatic experience, does not usually result in the inability to remember all or part of the traumatic experience itself, a phenomenon notably present in PTSD (American Psychiatric Association, 2000). However, the two disorders can be seen as similar with respect to dissociative symptomatology. Depersonalization disorder hinges upon characteristic dissociative symptoms such as emotional numbing, subjective detachment from oneself and the world, and overall feelings of unreality. Emotional numbing in particular can also be seen in those suffering from PTSD (American Psychiatric Association, 2000). In addition to traumatic antecedents, research has also begun to uncover the biological nature and origins of depersonalization disorder.

The research concerning the biology of depersonalization disorder is still in its infancy, much of it being pioneered by the American psychiatrist Daphne Simeon. Though the current research is slim, this in itself does not invalidate or in any way lessen the impact of the many discoveries that are continually advancing our understanding of depersonalization disorder and mental illness as a whole. To begin to understand the role of biology in depersonalization disorder, it has been seen that numerous brain regions are involved in the development of the disorder. One such region is the sensory cortex. The sensory cortex is responsible for making hierarchical perceptual associations in the brain. As incoming sensory information is initially received, the primary cortical areas gather and relay this information to secondary unimodal association areas. These areas serve to synthesize sensory information and relay it to cross-modal (or polymodal) association areas, the last stop in the perceptual association hierarchy. Cross-modal association areas gather the synthesized sensory information to form what we might call "experience" – that is, subjective reality (Simeon & Abugel, 2006). Two areas of the brain that are essential in understanding the exceptionally common symptom of hypoemotionality in depersonalization disorder are the amygdala and the medial prefrontal cortex. The amygdala, a core component of the limbic system that serves as the basis for emotional responses in the brain, has been found to be underactive in those afflicted with depersonalization disorder (Simeon & Abugel, 2006). Due to this underactivation, numbness and lack of emotion inevitably result. Furthermore, the medial prefrontal cortex, responsible for dampening emotional responsiveness, is seen to be overactive in depersonalization disorder. With the underactivity of the amygdala and the overactivity of the medial prefrontal cortex, the limbic system and associated emotional responsiveness are severely inhibited (Simeon & Abugel, 2006). In addition to the brain regions previously mentioned, the insula, which serves to register internal bodily sensations, is underactive in depersonalization disorder, thus leading to an overall feeling of sensory dullness (Simeon & Abugel, 2006). Finally, a brain region important to the understanding of depersonalization

disorder is the hippocampus. The hippocampus, responsible for narrative (or concrete) memory encoding, fails to initially encode information. Though depersonalization does not inherently involve amnesia for traumatic events, abnormal encoding of memory (in the form of images versus a cohesive narrative, for example) still plays a role in the development of depersonalization disorder (Simeon & Abugel, 2006). Now that an essential understanding of the various brain regions involved in depersonalization disorder has been established, we can now turn to the question of the theoretical models of the biology of depersonalization.

One such theoretical model is the temporal lobe model of depersonalization. Researchers have found that after stimulating the temporal cortex, individuals have reported experiencing sensations of floating away and out-of-body experiences. It is hypothesized that these experiences as well as other symptoms of depersonalization disorder are the result of the abnormal "tagging" of incoming sensory information as unknown against existing memory templates. The resulting subjective experience is often a sense of strangeness, unfamiliarity, or overt unreality with reference to the self and/or the external world (Simeon & Abugel, 2006).

Another theoretical model of depersonalization is the cortico-limbic disconnection model. The essential component of this model is the lack of synchronicity between various regions of the brain. This model reflects the notion of the overactivity of the medial prefrontal cortex and the underactivity of the amygdala previously described. Depersonalization disorder is said to result in the event of the lack of synchronicity between the cortex – which is responsible for cognition and the dampening of emotions – and the limbic system – which is the emotional center of the brain (Simeon & Abugel, 2006).

One final theoretical model of depersonalization is the cortico-cortical disconnection model – the most wide-ranging and all-encompassing model of the three. The cortico-cortical disconnection model not only entails overall disconnections between regions of the cortex, but also involves cortico-limbic disconnection as well as what is known as thalamo-cortical disconnection (that is, abnormalities in the gating, relaying, and modulating of sensory information). It has been shown that disconnections between various areas of the cortex result from the blocking of the NMDA receptor, the receptor for the excitatory neurotransmitter glutamate which is usually responsible for the proper regulation of connections in the cortex (Simeon & Abugel, 2006).

Various neurological studies have shown an abnormal functioning of the sensory association cortex, which is responsible for processing sensory information and making necessary associations (Sierra & Berrios, 1998). Furthermore, brain-imaging studies including PET scans and fMRIs have been consistent with the theoretical models previously described. The overactivation of

the prefrontal cortex and the underactivation of the limbic system have been demonstrated in these brain-imaging studies as components of depersonalization disorder. In this sense, individuals are hypercognitive and hypoemotional – that is, they are cognitively aware that they should experience emotional responsiveness but they lack the ability to actually feel (Phillips et al., 2001; Simeon et al., 2000). Additionally, neurological studies examining the neurochemistry behind depersonalization have shown that various neurotransmitters could be involved in depersonalization.

Firstly, a possible serotonin component of depersonalization has been demonstrated. The hallucinogen and serotonin receptor agonist lysergic acid diethylamide (LSD) has been shown to trigger depersonalization symptoms in healthy individuals (Simeon & Abugel, 2006). Furthermore, meta-chlorophenylpiperazine (mCPP), another serotonin receptor agonist, has induced episodes of transient depersonalization (Simeon et al., 1995). However, in the treatment of depersonalization disorder, it still remains to be established whether selective serotonin reuptake inhibitors (SSRIs) are effective treatments. In addition to a possible serotonin involvement, it has been demonstrated that NMDA receptor antagonists – which serve to block glutamate at these receptors – such as ketamine (the "dissociative anesthetic") as well as cannabinoids, hallucinogens, and opioids often induce states of depersonalization and/or derealization (Simeon & Abugel, 2006). In addition to the neurochemistry of depersonalization, other studies have also validated the three theoretical models previously described.

Such studies have involved measurements of skin conductance – that is, the electrical properties of the skin – in response to various stimuli ranging from emotionally pleasant to neutral to unpleasant. Researchers have found a severely reduced skin conductance response to emotionally unpleasant stimuli in individuals suffering from depersonalization disorder. This goes to show the profound hypoemotionality inherent in the disorder. Also, researchers have found that these same individuals demonstrated an increased response to unexpected stimuli such as claps and other loud noises. It has accordingly been postulated that depersonalization serves the evolutionary function of increasing survival in situations of danger by simultaneously suppressing fear and increasing alertness (Sierra et al., 2002).

In conclusion, it is evident that depersonalization and derealization are relatively common dissociative phenomena whose etiological roots can be seen with reference to various biological and traumatic dissociative processes. It is imperative that an increased awareness of the existence, prevalence, and causes of depersonalization disorder should be established in order to foster in those suffering from the disorder a sense of community and societal acceptance. Future research should aim to build upon the current framework of knowledge relating to

depersonalization disorder and to endeavor to enhance effective treatment options for those suffering with the disorder.

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