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Emotion and the Unreal Self: Depersonalization Disorder and De-Affectualization

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Abstract

Depersonalization disorder (DPD) is a psychiatric condition in which there is a pervasive change in the quality of subjective experience, in the absence of psychosis. The core complaint is a persistent and disturbing feeling that experience of oneself and the world has become empty, lifeless, and not fully real. A greatly reduced emotional responsivity, or "de-affectualization," is frequently described. This article examines the phenomenology and neurobiology of DPD with a particular emphasis on the emotional aspects. It is argued that the study of DPD may provide valuable insights into the relationship between emotion, experience, and identity.

Keywords

depersonalization, emotion regulation, fMRI, subjective experience

The term "depersonalization" was first coined by Dugas in 1898 (see Berrios & Sierra, 1997), and denotes a state in which the sense of self and the quality of subjective first-person experience are oddly altered, such that the person feels somehow alienated or estranged from themselves (depersonalization) and/or their surroundings (derealization). While psychiatric classification and literature distinguish between "depersonalization" (DP) and "derealization" (DR), in practice these two phenomena often co-occur. Some patients with persistent depersonalization symptoms may find that the DP/DR distinction does not ring true for them, as they experience both as part of the same essential alteration of experience (Sierra, 2009). In this article, as in most work on this topic, the term "depersonalization" will be used to denote this general alteration of subjective experience, so it can be taken as including derealization, as well as other experiential aspects explored in what follows.

Brief, self-limiting episodes of mild depersonalization are usually not pathological: indeed they are common among the general population, particularly under conditions of stress and fatigue: the "spaced out," unreal feeling induced by jet lag is an example, while many psychoactive drugs, including alcohol, may produce transient experiences of depersonalization (Medford et al., 2003). However, depersonalization can occur as a persistent, pervasive phenomenon, causing subjective distress and functional impairment. This may be in the context of another neurological or psychiatric disorder, such as major depression or posttraumatic stress disorder, or it may occur as a primary phenomenon, in which case it is classified as a condition in its own right: depersonalization disorder (DPD).

The two major classificatory systems used in contemporary psychiatry are the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV), (American Psychiatric Association, 1994) and the *International Classification of Diseases* (ICD-10), (World Health Organization, 1992). While there are some important differences between them, they are largely in accord regarding diagnostic criteria for DPD (Medford, Sierra, Baker, & David, 2005): For a diagnosis, there should be persistent symptoms of DP/DR, which should not occur as part of another disorder or be directly substance-induced, and the individual should not be suffering from psychosis (which would imply a different diagnosis, such as schizophrenia). The DSM adds the criterion that there should be significant distress and/or functional impairment—this seems appropriate, as without either of these it is hard to argue that the phenomena can usefully be seen as pathological.

Population and clinic surveys suggest that clinically significant depersonalization (either due to primary DPD, or secondary to another condition) affects 1–2% of the population (Hunter, Sierra, & David, 2004), and that the onset is most commonly in adolescence or early adulthood. The condition may go undiagnosed for many years, presumably because the

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topic lacks prominence among psychiatrists and their colleagues in other medical disciplines (Baker et al., 2003). There are reports of successful treatment with a range of psychological and pharmacological interventions, but as yet no strong, large-scale evidence for any specific treatment strategy (see Medford et al., 2005; Sierra, 2009, for reviews).

Phenomenology of DPD: The Syndrome Approach and the Importance of "De-Affectualization"

Attempts to describe and understand the depersonalized state evoke fundamental questions: What do we mean by "sense of self"? How is such a sense generated and maintained? If a person says their surroundings feel "unreal," yet knows that they are in fact real, what might this tell us about the phenomenology of experience? The brief descriptions in the DSM-IV and ICD-10 only hint at this complexity. When the ICD-10 notes that "the sufferer complains that his or her mental activity, body, and/or surroundings are changed in their quality" (World Health Organization, 1992, pp. 171–172), however, it is possible to see that almost any aspect of first-person experience is, in theory, available for inclusion in the definition. Despite this, it is not always appreciated that, in both practice (evidence from symptom surveys) as well as theory (the scope of the formal ICD definition), DPD usually involves symptoms in a number of different domains. Sierra, Baker, Medford, and David (2005, p. 1524) observe that patients with DPD may complain of "numbed emotional experiencing, heightened self-observation; altered body experience, feelings of not being in control of movement; changes in the experiencing of time and space; feelings of mind emptiness, inability to imagine things," as well as perceptual anomalies, for example, the external world seeming oddly flat and two-dimensional, or colors seeming less (or, sometimes, more) vivid than previously.

Various terms have been coined to describe domains of symptoms within DPD. Davidson (1966) suggested the term "de-affectualization" to denote the change in emotional experience commonly reported by patients with DPD, in which there is a persistent diminution or loss of emotional reactivity, and emotions seem to lack spontaneity and subjective validity. There are remarkably consistent first-person accounts of de-affectualization in both older and more recent literature (Baker et al., 2003; Mayer-Gross, 1935; Shorvon, Hill, & Burkitt, 1946; Simeon & Abugel, 2006). Davidson also proposed the term "desomatization" to describe altered body experience in DPD. Typically this involves reduction, loss, or alteration of bodily sensations, and a sense of disembodiment; there may be a raised pain threshold and patients may report a disturbance in the sense of ownership of body parts—for example, a patient may look at his hands and say they do not seem like his hands, even though he knows they are his and that he has control over them. Issues arising from disturbances of bodily feeling are discussed elsewhere in this special section by Colombetti and Ratcliffe.

In addition, the term "de-ideation" has been suggested (Taylor, 1982) for anomalous experiences of thought, concentration, memory, and mental imagery. Difficulty in concentrating is a particularly common complaint in DPD, with patients often describing this in physical terms, for example, "I feel as if my head is full of cotton wool" (Medford et al., 2005).

Empirical evidence supporting a syndrome concept of depersonalization comes from a study of patient responses to the Cambridge Depersonalization Scale (CDS). The CDS (Sierra & Berrios, 2000) is a self-report scale which probes a range of experiences associated with DPD, and factor analysis of patient responses suggests a consistent pattern of symptom clusters within DPD, summarized by the following headings: "Anomalous Body Experience," "Emotional Numbing," "Anomalous Subjective Recall," and "Alienation From Surroundings" (Sierra et al., 2005). These are essentially analogous to the terms desomatization, de-affectualization, de-ideation, and derealization. Using a similar method with a larger sample, a more recent study by another group gave strikingly similar results (Simeon et al., 2008).

It can be seen from the above that the phenomenon of "de-affectualization" or emotional numbing has been consistently described as a core feature of the syndrome. Indeed, this observation probably predates even the coining of the term "depersonalization." Some 60 years before Dugas, Zeller reported five patients who

> . . . complained almost in the same terms of a lack of sensations . . . to them it was a total lack of feelings, as if they were dead . . . they claimed they could think clearly, and properly about everything, but the essential was lacking even in their thoughts . . . (Zeller, 1838, trans. in Berrios & Sierra, 1997)

However, de-affectualization in DPD is not usually accompanied by the objectively blunted affect often seen in chronic schizophrenia (Ackner, 1954; Torch, 1978).

Yet this gives rise to an apparent paradox: if reduced emotional experience is a core feature of DPD, how can this be reconciled with the high levels of subjective distress reported by sufferers? Throughout the literature, first-person reports emphasize the deeply unpleasant and disturbing aspect of the experience (Baker et al., 2003; Mayer-Gross, 1935; Shorvon et al., 1946; Sims, 1995). Sims (1995, p. 204) quotes one patient as saying: "I feel very weird in my head. I have a great deal of torment. My mind will not leave me alone…. I feel as if I'm lost in a fog. I just feel as if I'm not in my head. I feel numb." This quote contains statements exemplifying both aspects of this apparent contradiction: subjective distress ("I have a great deal of torment") and diminished sensitivity to experience ("I feel numb"). It is significant that the distress is described as arising from *the unpleasantness of the depersonalization experience itself*: this is a consistent theme in patient self-reports. At the same time, there is reduced responsivity to the external world, experienced as distant, lifeless, unreal, and lacking in emotional content. A patient seen by the present author made the remark: "I don't have any emotions—it makes me so unhappy." This may seem self-contradictory, but on further questioning, he explained that he experienced considerable inner turmoil related to his experience of being altered and "not himself," but felt little or no emotional response to

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external events or other people. Ackner (1954, p. 852) details similar descriptions by patients, and suggests that in DPD there is an "increased responsiveness for anxiety of internal origin, whereas that of external origin [is] reduced." How can this be? One way of solving this apparent contradiction is through the consideration of attentional processes. Throughout the DPD literature it is noted that sufferers tend to focus attention on inner sensations and concerns, at the expense of attending to the external world (see Hunter, Phillips, Chalder, Sierra, & David, 2003, for a review). If attention is persistently drawn to the strangely altered inner feelings that are the core of the condition, the corresponding lack of attentional focus on the outside world may contribute to the sense that the world has become somehow distant and unreal. This attentional imbalance may explain the combination of a subjective experience of inner turmoil with emotional unresponsivity to external events. The putative connection between attentional style and altered emotional experience in DPD merits further research, particularly around the possibility that exercises aimed at reorienting attention could have a role in treatment (Hunter et al., 2003).

The idea that a pervasive disturbance of subjective feelings was the key to understanding the depersonalization experience enjoyed considerable currency among German writers of the early 20th century, the position exemplified by Osterreich: "we postulate that at the foreground [of depersonalization] there is a more or less generalized inhibition of feelings that leads to a reduction of self-feelings and self-awareness" (Osterreich, 1907, trans. in Berrios & Sierra, 1997). The apparent dampening, or even "shutting down," of emotional responses in DPD is consistent with the notion that depersonalization arises as a defence against anxiety, threat, or negative emotional experience in general. In psychoanalytic theory, depersonalization has long been considered a defense mechanism, though the specific details of this idea vary widely between different schools and theorists (Ambrosino, 1976). Healthy people exposed to life-threatening danger almost always report at least some features of depersonalization (Noyes & Kletti, 1977), supporting the idea that depersonalization may be a normal response to overwhelming threat, and that pathological depersonalization may be understood as a state that arises in susceptible individuals in whom this response is triggered at lower thresholds. This relates to an idea expressed over 70 years ago by Mayer-Gross, who conceived of depersonalization as a "pre-formed response of the brain," that is, a particular psychophysiological state that could be induced by certain circumstances or stimuli, such as situations involving threat (Mayer-Gross, 1935). Clinically, there is often an impression that depersonalization arises in individuals predisposed to anxiety, and because the depersonalization experience is itself strange and unsettling, it generates further anxiety, which in turn serves to reinforce and perpetuate the depersonalization (Medford et al., 2005). Anxiety-related ruminations and behaviors associated with chronic DP/DR may involve obsessional self-checking (frequently checking one's own inner state—"do I feel real now?"—in a way that probably promotes further estrangement from immediate experience; see Torch, 1978) and/or persistent worries that the DP/DR symptoms represent incipient madness, or are caused by some serious condition such as a brain tumor (Simeon & Hollander, 1993). Hunter et al. (2003) outlined a cognitive–behavioral conception of DPD, based on the idea that anxiety-related cognitions and behaviors can exacerbate and perpetuate DP/DR symptoms, are an important factor in the development of chronicity (persistence of symptoms over time) in primary DPD, and can be identified and worked on as part of a psychological treatment approach to DPD. While this approach may have some explanatory power—and therapeutic merits—in cases where such anxieties are prominent, it does not claim to offer an explanation of how DP/DR symptoms initially arise—it primarily addresses cognitions associated with, and consequent upon, DP/DR experiences, rather than the actual experiences themselves. A cognitivist account of DPD may therefore be helpful in identifying patterns of thought and behavior often associated with DP/DR symptoms and which may be usefully addressed in treatment. But such an account will struggle to account for the experiential core of the condition, particularly the emotional and somatic features.

Overall, then, there are both phenomenological and etiological reasons for placing emotion at the heart of any attempt to further understand depersonalization.

Empirical Studies of DPD: Insights for Emotion Research

If this approach to understanding de-affectualization is correct, a testable prediction is that DPD patients should show attenuated responses to experimentally presented emotional stimuli. A number of studies have tested this idea. Sierra et al. (2002) used skin conductance recording to probe autonomic arousal in response to emotionally salient images, and found that DPD patients showed significantly attenuated responses to unpleasant images specifically. A functional magnetic resonance imaging (fMRI) study of memory for emotional and neutral words found that patients with DPD showed similar neural activation patterns regardless of the emotional salience of the presented stimuli, in contrast to a healthy control group who showed extensive emotion-related activations not seen in response to the neutral stimuli (Medford et al., 2006).

Another functional neuroimaging study of interest here is a positron emission tomography (PET) study of eight patients with DPD (Simeon et al., 2000). In this study, patients with DPD were found to differ significantly from controls in activation in regions of temporal and parietal sensory association cortex. The authors suggest that these findings reflect the failure of normal integration of sensation and awareness in DPD—an idea related to comments on insula function mentioned before. Differences in insula activity were not found, however, but the cognitive task used during scanning, a simple verbal learning paradigm, was not designed to probe emotional processing or the generation of feelings, so may not have specifically engaged insula in the controls.

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Phillips et al. (2001) used fMRI to study neural responses to alternating blocks of aversive and neutral scenes. Results from a small (n = 6) DPD group were compared with healthy and clinical control groups. Compared to the other two groups, the DPD group showed significantly reduced neural responses in brain regions associated with emotional processing, particularly anterior insula, when viewing aversive scenes. There was also some evidence that a region of right ventrolateral prefrontal cortex (VLPFC) was involved in inhibiting the neural response to aversive material.

A more recent fMRI study (Medford et al., 2011) used a similar paradigm in a larger group of DPD patients (n = 14). In comparison to healthy controls, patients showed a significant absence of activation in the left anterior insula (LAI) in response to aversive images. Ten of the 14 patients repeated the scanning paradigm after 4–8 months of pharmacotherapy. In patients reporting significant clinical improvement, there was activation of LAI present in response to emotional images at Time 2, and this region was significantly more active in patients whose symptoms had improved than in those whose symptoms had not. A region of right VLPFC, the same area identified in Phillips et al. (2001) as being involved in the suppression of emotional responses, was active in DPD patients at Time 1, but only in nonimproved patients at Time 2.

These findings are of great interest because they link in with key issues in the contemporary neuroscience of emotional experience, emotion regulation, and self-related processes. The anterior insula appears to be underactive during emotional stimulation in DPD, and this area has been identified as a key brain region in the generation of subjective feeling states. The influential work of Damasio and colleagues has centered around the idea that feeling states are produced by the integration of bodily sensations into conscious awareness (e.g., Damasio, 2003), so that subjective feelings arise out of signals from afferent somatosensory systems, which in combination produce "interoception," definable as a sense of the internal milieu (Craig, 2002). A wealth of converging evidence suggests that the anterior insula is a crucial "hub" for this process (see Craig, 2002, 2009; Medford & Critchley, 2010, for reviews). Across functional neuroimaging studies, anterior insula activation has been reported in a wide range of experimental contexts involving physical sensation or the induction of feeling states, including tactile stimulation, sexual arousal, visceral distension, happiness, anger, fear, sadness, and feelings of romantic love. Studies that have probed interoceptive awareness directly (e.g., by asking participants to make judgments about the timing of their own heartbeats, as in Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004) have identified anterior insula activity as directly correlated with such awareness, and statistically significant correlations between interoceptive awareness and self-reported emotional experience have also been demonstrated (Feldman Barrett, Quigley, Bliss-Moreau, & Aronson, 2004).

In DPD, then, it is possible to see anterior insula underactivity as the key neural correlate of the self-reported experience of de-affectualization. The involvement of anterior insula in representations of bodily state further suggests that reduced activity of this region may also be the biological substrate of the "desomatization" aspect of DPD symptomatology. It is not being conceptually greedy to link both these symptom domains with the anterior insula: the evidence that bodily sensations and feeling states are integrated in anterior insula is now sufficiently strong (Craig, 2009) to make this a logical suggestion. Indeed, one can go further and suggest that this alteration in the quality of bodily and emotional experience is the essential core of DPD: if subjective feelings are derived from higher order representations of bodily states in anterior insula, then abnormalities of this process can, in theory, have consequences for the whole spectrum of first-person experience, as is the case in DPD (see also Colombetti & Ratcliffe, 2012). If this is correct, then one might predict that DP/DR symptoms may occur when there is disturbance of sensory systems. There is some evidence that this is true. Symptoms of depersonalization are significantly more common in patients with vestibular disease than would be expected by chance (Sang, Jauregui-Renaud, Green, Bronstein, & Gresty, 2006), giving rise to the idea that depersonalization may arise when sensory deficits give rise to disorientation: if perceptions of the spatial relationship of the body to external reality are unreliable, this may predispose to a more general experience of strangeness and unreality, as occurs in DPD (Jauregui-Renaud, Sang, Gresty, Green, & Bronstein, 2008).

As detailed above, fMRI data suggest a role for right VLPFC (Brodmann Area 47) in the suppression of emotional responses in DPD. Studies of healthy participants have identified this area as being involved in the control of emotion (Ochsner & Gross, 2005; Ohira et al., 2006). A recent study of the regulation of both positive and negative emotion found this area to be involved in emotion regulation in general, but most particularly when participants were attempting to decrease the experience of negative emotional responses (Kim & Hamann, 2007). Perhaps most significantly, a study of cognitive reappraisal (a mental strategy of deliberate conscious reframing or reinterpretation of emotional material in such a way as to reduce its affective impact) found that right VLPFC activity was closely associated with successful (i.e., emotion-reducing) reappraisal, and identified a pathway from the right VLPFC through nucleus accumbens and ventral striatum, which appears to be specialized for the inhibition of responses to aversive stimuli (Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008). This is of potential relevance to DPD, firstly because the area of right VLPFC identified by these authors is anatomically very close to that repeatedly identified in DPD, but also because in DPD there is a clinical impression that patients unwittingly tend towards a ruminative intellectualization of emotional issues and situations (Hunter et al., 2003; Medford et al., 2005; Torch, 1978), which may be analogous to cognitive reappraisal. All these studies examine the *voluntary* suppression of emotional responses: in DPD such suppression is apparently involuntary (and largely resistant to volitional control), but it is reasonable to suppose that this will nevertheless engage similar inhibitory networks.

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Future Directions

While this article has focused on primary DPD, symptoms of depersonalization are common outside of this clinical context, occurring in a range of neuropsychiatric conditions. With regard to schizophrenia in particular, there is a long-standing current of thought that regards DP/DR as important early symptoms in the development of schizophreniform psychosis (e.g., Huber's notion of "basic symptoms" in schizophrenia; see Gross, 1997). Yet there has been very little empirical research examining either the prevalence and character of DP/DR symptoms in this wider clinical arena, or how such symptoms relate to other aspects of specific conditions. More research along these lines has the potential to substantially improve our understanding of the experiences of patients, and to suggest new psychotherapeutic and pharmacological treatment strategies.

Closing Remarks

Depersonalization, both as a primary disorder (DPD) and as a phenomenon in general, provides an unusual and valuable real-life sounding board for important ideas about the nature of first-person experience, the processes through which feelings are generated, and the ways in which subjective experiences shape an individual's sense of themselves. Empirical studies of DPD can shed light on the psychological and biological processes that underpin these sometimes abstract concepts. It is to be hoped that this hitherto little-studied condition will become a focus of interest for neuroscientists and philosophers working on these issues, and yield insights of relevance not only to psychiatry but also to the wider understanding of the human condition.

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