

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



Journal:	<i>Emotion Review</i>
Manuscript ID:	EMR-11-302.R1
Manuscript Type:	Original Manuscript
Date Submitted by the Author:	n/a
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Area/Discipline:	neuroscience, clinical < psychology
Keywords:	depersonalization, emotion regulation, subjective experience, functional MRI
Abstract:	Depersonalization disorder 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 responsiveness, 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.

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8 Emotion and the Unreal Self: Depersonalization Disorder and De-Affectualization
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For Peer Review

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6 Abstract
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10 Depersonalization disorder is a psychiatric condition in which there is a pervasive change in
11 the quality of subjective experience, in the absence of psychosis. The core complaint is a
12 persistent and disturbing feeling that experience of oneself and the world has become empty,
13 lifeless, and not fully real. A greatly reduced emotional responsivity, or 'de-affectualization',
14 is frequently described. This article examines the phenomenology and neurobiology of DPD
15 with a particular emphasis on the emotional aspects. It is argued that the study of DPD may
16 provide valuable insights into the relationship between emotion, experience, and identity.
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34 Keywords: depersonalization, emotion regulation, subjective experience, functional MRI.
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Introduction and general overview of DPD

The term 'depersonalization' was first coined by Dugas in 1898 (see Berrios and 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 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 can be taken as including derealization, as well as other experiential aspects explored below.

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 post-traumatic 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).

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3 The two major classificatory systems used in contemporary psychiatry are the DSM-IV
4 (Diagnostic and Statistical Manual, American Psychiatric Association) and the ICD-10
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6 (International Classification of Diseases, World Health Organisation). While there are some
7
8 important differences between them, they are largely in accord regarding diagnostic criteria
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10 for DPD (Medford et al 2005): for a diagnosis, there should be persistent symptoms of
11
12 DP/DR, which should not occur as part of another disorder or be directly substance-induced,
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14 and the individual should not be suffering from psychosis (which would imply a different
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16 diagnosis, such as schizophrenia). DSM adds the criterion that there should be significant
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18 distress and/or functional impairment- this seems appropriate, as without either of these it is
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20 hard to argue that the phenomena can usefully be seen as pathological.
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31 Population and clinic surveys suggest that clinically significant depersonalization (due to
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33 either primary DPD, or secondary to another condition) affects 1-2% of the population
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35 (Hunter et al, 2004), and that the onset is most commonly in adolescence or early adulthood.
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37 The condition may go undiagnosed for many years, presumably because the topic lacks
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39 prominence among psychiatrists and their colleagues in other medical disciplines (Baker et al
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41 2003). There are reports of successful treatment with a range of psychological and
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43 pharmacological interventions, but as yet no strong, large-scale evidence for any specific
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45 treatment strategy (see Medford et al 2005, Sierra 2009, for reviews).
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3 Phenomenology of DPD: the syndrome approach and the importance of ‘de-affectualization’.
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10 Attempts to describe and understand the depersonalized state evoke fundamental questions:
11 what do we mean by “sense of self”? How is such a sense generated and maintained? If a
12 person says their surroundings feel “unreal”, yet knows that they are in fact real, what might
13 this tell us about the phenomenology of experience? The brief descriptions in DSM-IV and
14 ICD-10 only hint at this complexity. When ICD-10 notes that “the sufferer complains that his
15 or her mental activity, body, and/or surroundings are changed in their quality”, however, it is
16 possible to see that almost any aspect of first-person experience is, in theory, available for
17 inclusion in the definition. Despite this, it is not always appreciated that, in both practice
18 (evidence from symptom surveys) as well as theory (the scope of the formal ICD definition),
19 DPD usually involves symptoms in a number of different domains. Sierra *et al* (2005)
20 observe that patients with DPD may complain of “*numbed emotional experiencing,*
21 *heightened self-observation; altered body experience, feelings of not being in control of*
22 *movement; changes in the experiencing of time and space; feelings of mind emptiness,*
23 *inability to imagine things*”, as well as perceptual anomalies e.g the external world seeming
24 oddly flat and two-dimensional, or colours seeming less (or, sometimes, more) vivid than
25 previously.
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51 Various terms have been coined to describe domains of symptoms within DPD. Davidson
52 (1966) suggested the term “de-affectualization” to denote the change in emotional experience
53 commonly reported by patients with DPD, in which there is a persistent diminution or loss of
54 emotional reactivity, and emotions seem to lack spontaneity and subjective validity. There are
55 remarkably consistent first-person accounts of de-affectualization in both older and more
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3 recent literature (Mayer-Gross, 1935, Shorvon et al 1946, Baker *et al*, 2003, Simeon and
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5 Abugel 2006). Davidson also proposed the term “desomatization” to describe altered body
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7 experience in DPD- typically this involves reduction, loss or alteration of bodily sensations,
8
9 and a sense of disembodiment; there may be a raised pain threshold and patients may report a
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11 disturbance in the sense of ownership of body parts e.g. a patient may look at his hands and
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13 say they do not seem like his hands, even though he knows they are his and that he has
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15 control over them. Issues arising from disturbances of bodily feeling are discussed elsewhere
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17 in this special issue by Colombetti and Ratcliffe.
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26 In addition, the term “de-ideation” has been suggested (Taylor, 1982) for anomalous
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28 experiences of thought, concentration, memory and mental imagery. Difficulty in
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30 concentrating is a particularly common complaint in DPD, with patients often describing this
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32 in physical terms e.g. “*I feel as if my head is full of cotton wool*”. (Medford *et al*, 2005).
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41 Empirical evidence supporting a syndrome concept of depersonalization comes from a study
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43 of patient responses to the Cambridge Depersonalization Scale (CDS). The CDS (Sierra and
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45 Berrios, 2000) is a self-report scale which probes a range of experiences associated with
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47 DPD, and factor analysis of patient responses suggests a consistent pattern of symptom
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49 clusters within DPD, summarized by the following headings: ‘Anomalous Body Experience’,
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51 ‘Emotional Numbing’, ‘Anomalous Subjective Recall’, and ‘Alienation From Surroundings’
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53 (Sierra et al, 2005). These are essentially analogous to the terms desomatization, de-
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55 affectualization, de-ideation, and derealization. Using a similar method with a larger sample,
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57 a more recent study by another group gave strikingly similar results (Simeon *et al*, 2008).
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7 It can be seen from the above that the phenomenon of ‘de-affectualization’ or emotional
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9 numbing has been consistently described as a core feature of the syndrome. Indeed this
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11 observation probably predates even the coining of the term “depersonalization”. Some sixty
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13 years before Dugas, Zeller reported five patients who “complained almost in the same terms
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15 of a lack of sensations ... to them it was a total lack of feelings, as if they were dead ... they
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17 claimed they could think clearly, and properly about everything, but the essential was lacking
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19 even in their thoughts ...” (Zeller, 1838, trans. in Berrios and Sierra, 1997). However, de-
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21 affectualization in DPD is not usually accompanied by the objectively blunted affect often
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23 seen in chronic schizophrenia (Ackner 1954, Torch 1978).
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32 Yet this gives rise to an apparent paradox: if reduced emotional experience is a core feature
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34 of DPD, how can this be reconciled with the high levels of subjective distress reported by
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36 sufferers? Throughout the literature, first-person reports emphasise the deeply unpleasant and
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38 disturbing aspect of the experience (Mayer-Gross 1935, Shorvon 1946, Sims 1995, Baker *et*
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40 *al* 2003). Sims (1995) quotes one patient as saying: “*I feel very weird in my head. I have a*
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42 *great deal of torment. My mind will not leave me alone.... I feel as if I’m lost in a fog. I just*
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44 *feel as if I’m not in my head. I feel numb.*” This quote contains statements exemplifying both
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46 aspects of this apparent contradiction: subjective distress (“I have a great deal of torment”)
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48 and diminished sensitivity to experience (“I feel numb”). It is significant that the distress is
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50 described as arising from *the unpleasantness of the depersonalization experience itself*: this is
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52 a consistent theme in patient self-reports. At the same time, there is reduced responsivity to
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54 the external world, experienced as distant, lifeless, unreal, and lacking in emotional content.
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60 A patient seen by the present author made the remark: “*I don’t have any emotions- it makes*

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3 *me so unhappy*". This may seem self-contradictory, but on further questioning, he explained
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5 that he experienced considerable inner turmoil, related to his experience of being altered and
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7 'not himself', but felt little or no emotional response to external events or other people.
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10 Ackner (1954) details similar descriptions by patients, and suggests that in DPD there is an
11
12 "*increased responsiveness for anxiety of internal origin, whereas that of external origin [is]*
13
14 *reduced*". How can this be? One way of solving this apparent contradiction is through the
15
16 consideration of attentional processes. Throughout the DPD literature it is noted that
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18 sufferers tend to focus attention on inner sensations and concerns, at the expense of attending
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20 to the external world (see Hunter et al, 2003, for a review). If attention is persistently drawn
21
22 to the strangely altered inner feelings that are the core of the condition, the corresponding
23
24 lack of attentional focus on the outside world may contribute to the sense that the world has
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26 become somehow distant and unreal. This attentional imbalance may explain the combination
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28 of a subjective experience of inner turmoil with emotional unresponsivity to external events.
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30 The putative connection between attentional style and altered emotional experience in DPD
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32 merits further research, particularly around the possibility that exercises aimed at re-orienting
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34 attention could have a role in treatment (Hunter et al, 2003).
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45 The idea that a pervasive disturbance of subjective feelings was the key to understanding the
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47 depersonalization experience enjoyed considerable currency among German writers of the
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49 early 20th century, the position exemplified by Osterreich: "we postulate that at the
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51 foreground [of depersonalization] there is a more or less generalized inhibition of feelings
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53 that leads to a reduction of self-feelings and self-awareness" (Osterreich, 1907, trans. Berrios
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55 and Sierra, 1997). The apparent dampening, or even 'shutting down', of emotional responses
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57 in DPD is consistent with the notion that depersonalization arises as a defence against
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59 anxiety, threat, or negative emotional experience in general. In psychoanalytic theory,
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3 depersonalization has long been considered a defence mechanism, though the specific details
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5 of this idea vary widely between different schools and theorists (Ambrosino, 1976). Healthy
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7 people exposed to life-threatening danger almost always report at least some features of
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9 depersonalization (Noyes, Jr. and Kletti, 1977), supporting the idea that depersonalization
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11 may be a normal response to overwhelming threat, and that pathological depersonalization
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13 may be understood as a state that arises in susceptible individuals in whom this response is
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15 triggered at lower thresholds. This relates to an idea expressed over 70 years ago by Mayer-
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17 Gross, who conceived of depersonalization as a “pre-formed response of the brain” i.e. a
18
19 particular psychophysiological state that could be induced by certain circumstances or
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21 stimuli, such as situations involving threat (Mayer-Gross, 1935). Clinically, there is often an
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23 impression that depersonalization arises in individuals predisposed to anxiety, and because
24
25 the depersonalization experience is itself strange and unsettling, it generates further anxiety,
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27 which in turn serves to reinforce and perpetuate the depersonalization (Medford *et al*, 2005).
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29 Anxiety-related ruminations and behaviours associated with chronic DP/DR may involve
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31 obsessional self-checking (frequently checking one’s own inner state- “do I feel real now?”-
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33 in a way that probably promotes further estrangement from immediate experience, see Torch
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35 1978) and/or persistent worries that the DP/DR symptoms represent incipient madness, or are
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37 caused by some serious condition such as a brain tumour (Simeon and Hollander 1993).
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39 Hunter et al (2003) outlined a cognitive-behavioural conception of DPD, based on the idea
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41 that anxiety-related cognitions and behaviours can exacerbate and perpetuate DP/DR
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43 symptoms, are an important factor in the development of chronicity (persistence of symptoms
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45 over time) in primary DPD, and can be identified and worked on as part of a psychological
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47 treatment approach to DPD. While this approach may have some explanatory power- and
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49 therapeutic merits- in cases where such anxieties are prominent, it does not claim to offer an
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51 explanation of how DP/DR symptoms initially arise- it primarily addresses cognitions
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3 associated with, and consequent upon, DP/DR experiences, rather than the actual experiences
4 themselves. A cognitivist account of DPD may therefore be helpful in identifying patterns of
5 thought and behaviour often associated with DP/DR symptoms and which may be usefully
6 addressed in treatment. But such an account will struggle to account for the experiential core
7 of the condition, particularly the emotional and somatic features.
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19 Overall, then, there are both phenomenological and aetiological reasons for placing emotion
20 at the heart of any attempt to further understand depersonalization.
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31 Empirical studies of DPD: insights for emotion research

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38 If this approach to understanding de-affectualization is correct, a testable prediction is that
39 DPD patients should show attenuated responses to experimentally presented emotional
40 stimuli. A number of studies have tested this idea. Sierra et al (2002) used skin conductance
41 recording to probe autonomic arousal in response to emotionally salient images, and found
42 that DPD patients showed significantly attenuated responses to unpleasant images
43 specifically. A functional MRI study of memory for emotional and neutral words found that
44 patients with DPD showed similar neural activation patterns regardless of the emotional
45 salience of the presented stimuli, in contrast to a healthy control group who showed extensive
46 emotion-related activations not seen in response to the neutral stimuli (Medford et al 2006).
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3 Another functional neuroimaging study of interest here is a PET study of 8 patients with DPD
4 (Simeon et al 2000). In this study, patients with DPD were found to differ significantly from
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6 controls in activation in regions of temporal and parietal sensory association cortex. The
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8 authors suggest that these findings reflect the failure of normal integration of sensation and
9
10 awareness in DPD- an idea related to comments on insula function above. Differences in
11
12 insula activity were not found, however, but the cognitive task used during scanning, a simple
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14 verbal learning paradigm, was not designed to probe emotional processing or the generation
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16 of feelings, so may not have specifically engaged insula in the controls.
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26 Phillips et al (2001) used functional MRI to study neural responses to alternating blocks of
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28 aversive and neutral scenes. Results from a small (n=6) DPD group were compared with
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30 healthy and clinical control groups. Compared to the other two groups, the DPD group
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32 showed significantly reduced neural responses in brain regions associated with emotional
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34 processing, particularly anterior insula, when viewing aversive scenes. There was also some
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36 evidence that a region of right ventrolateral prefrontal cortex (VLPFC) was involved in
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38 inhibiting the neural response to aversive material.
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47 A more recent fMRI study (Medford et al, manuscript in preparation) used a similar paradigm
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49 in a larger group of DPD patients (n=14). In comparison to healthy controls, patients showed
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51 a significant absence of activation in the left anterior insula (LAI) in response to aversive
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53 images. 10 of the 14 patients repeated the scanning paradigm after 4-8 months of
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55 pharmacotherapy. In patients reporting significant clinical improvement, there was activation
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57 of LAI present in response to emotional images at time 2, and this region was significantly
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59 more active in patients whose symptoms had improved than in those whose symptoms had
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3 not. A region of right VLPFC, the same area identified in Phillips et al (2001, see above) as
4 being involved in the suppression of emotional responses, was active in DPD patients at time
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8 1, but only in non-improved patients at time 2.
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15 These findings are of great interest because they link in with key issues in the contemporary
16 neuroscience of emotional experience, emotion regulation, and self-related processes. The
17 anterior insula appears to be underactive during emotional stimulation in DPD, and this area
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21 has been identified as a key brain region in the generation of subjective feeling states. The
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25 influential work of Damasio and colleagues has centred around the idea that feeling states are
26 produced by the integration of bodily sensations into conscious awareness (e.g. Damasio
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29 2003), so that subjective feelings arise out of signals from afferent somatosensory systems,
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32 which in combination produce 'interoception', definable as 'a sense of the internal milieu'
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35 (Craig 2002). A wealth of converging evidence suggests that the anterior insula is a crucial
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38 'hub' for this process (see Craig 2002, Craig 2009, Medford and Critchley 2010 for reviews).
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41 Across functional neuroimaging studies, anterior insula activation has been reported in a wide
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44 range of experimental contexts involving physical sensation or the induction of feeling states,
45 including tactile stimulation, sexual arousal, visceral distension, happiness, anger, fear,
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48 sadness, and feelings of romantic love. Studies that have probed interoceptive awareness
49 directly (e.g. by asking participants to make judgements about the timing of their own
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52 heartbeats, as in Critchley et al, 2004) have identified anterior insula activity as directly
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55 correlated with such awareness, and statistically significant correlations between
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58 interoceptive awareness and self-reported emotional experience have also been demonstrated
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61 (Feldman Barrett et al 2004).

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

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52 As detailed above, fMRI data suggest a role for right VLPFC (Brodmann Area 47) in the
53 suppression of emotional responses in DPD. Studies of healthy participants have identified
54 this area as involved in the control of emotion (Ochsner and Gross, 2005; Ohira et al, 2006).
55 A recent study of the regulation of both positive and negative emotion found this area to be
56 involved in emotion regulation in general, but most particularly when participants were
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3 attempting to decrease the experience of negative emotional responses (Kim and Hamann,
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6 2007). Perhaps most significantly, a study of cognitive reappraisal (a mental strategy of
7
8 deliberate conscious reframing or reinterpretation of emotional material in such a way as to
9
10 reduce its affective impact) found that right VLPFC activity was closely associated with
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12 successful (i.e. emotion-reducing) reappraisal, and identified a pathway from right VLPFC
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14 through nucleus accumbens and ventral striatum, which appears to be specialised for the
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16 inhibition of responses to aversive stimuli (Wager et al, 2008). This is of potential relevance
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18 to DPD, firstly because the area of right VLPFC identified by these authors is anatomically
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20 very close to that repeatedly identified in DPD, but also because in DPD, there is a clinical
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22 impression that patients unwittingly tend towards a ruminative intellectualisation of
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24 emotional issues and situations (Torch, 1978; Hunter et al, 2003, Medford et al, 2005), which
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26 may be analogous to cognitive reappraisal. All these studies examine the *voluntary*
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28 suppression of emotional responses: in DPD such suppression is apparently involuntary (and
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30 largely resistant to volitional control), but it is reasonable to suppose that this will
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32 nevertheless engage similar inhibitory networks.
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43 Future directions

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46 While this paper has focused on primary DPD, symptoms of depersonalization are common
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48 outside of this clinical context, occurring in a range of neuropsychiatric conditions. With
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50 regard to schizophrenia in particular, there is a longstanding current of thought that regards
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52 DP/DR as important early symptoms in the development of schizophreniform psychosis (e.g.
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54 Huber's notion of 'basic symptoms' in schizophrenia, see Gross 1997). Yet there has been
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56 very little empirical research examining the prevalence and character of DP/DR symptoms in
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58 this wider clinical arena, or how such symptoms relate to other aspects of specific conditions.
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3 More research along these lines has the potential to substantially improve our understanding
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5 of the experiences of patients, and to suggest new psychotherapeutic and pharmacological
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7 treatment strategies.
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21 Closing remarks
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27 Depersonalization, both as a primary disorder (DPD) and as a phenomenon in general,
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29 provides an unusual and valuable real-life sounding board for important ideas about the
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31 nature of first-person experience, the processes through which feelings are generated, and the
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33 ways in which subjective experiences shape an individual's sense of themselves. Empirical
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35 studies of DPD can shed light on the psychological and biological processes that underpin
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37 these sometimes abstract concepts. It is to be hoped that this hitherto little-studied condition
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39 will become a focus of interest for neuroscientists and philosophers working on these issues,
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41 and yield insights of relevance not only to psychiatry but also to the wider understanding of
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46 the human condition.
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