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Depersonalization: A selective impairment of self-awareness ☆

Mauricio Sierra *, Anthony S. David

Depersonalization Disorder Unit, Institute of Psychiatry, King's College, London, United Kingdom

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ABSTRACT

Depersonalization is characterised by a profound disruption of self-awareness mainly characterised by feelings of disembodiment and subjective emotional numbing.

It has been proposed that depersonalization is caused by a fronto-limbic (particularly anterior insula) suppressive mechanism – presumably mediated via attention – which manifests subjectively as emotional numbing, and disables the process by which perception and cognition normally become emotionally coloured, giving rise to a subjective feeling of 'unreality'.

Our functional neuroimaging and psychophysiological studies support the above model and indicate that, compared with normal and clinical controls, DPD patients show increased prefrontal activation as well as reduced activation in insula/limbic-related areas to aversive, arousing emotional stimuli.

Although a putative inhibitory mechanism on emotional processing might account for the emotional numbing and characteristic perceptual detachment, it is likely, as suggested by some studies, that parietal mechanisms underpin feelings of disembodiment and lack of agency feelings.

1. Introduction

Depersonalization is a fascinating and intriguing phenomenon, which challenges commonly held assumptions regarding the nature of self. The condition manifests as a pervasive disruption of self-awareness at its most basic, preverbal level (i.e. what it feels like to be an entity, to exist), unlike dissociative conditions such as psychogenic amnesia, or dissociative identity disorder, which typically impair identity at levels involving autobiographical memory, self-narratives, and personality. 'The person affected with depersonalization complains spontaneously that his or her mental activity, body, and surroundings are changed in their quality, so as to be unreal, remote, or automatized. Among the varied phenomena of the syndrome, patients complain most frequently of loss of emotions and feelings of estrangement or detachment from their thinking, their body, or the real world. In spite of the dramatic nature of the experience, the patient is aware of the unreality of the change. The sensorium is normal and the capacity for emotional expression intact' (World Health Organization, 1992).

Although *'feelings of unreality'* is still commonly used as a short-hand to describe the phenomenon in clinical practice, most patients stress the ineffable nature of the experience and make use of metaphors which usually take two forms. A first kind makes reference to a sense of being cut-off, alienated from oneself and surroundings. For example, patients would often talk about 'being in a bubble', or being 'separated from the world by an invisible barrier such as a pane of glass, a fog, or a veil' (Sierra, 2009). A second group of metaphors emphasise instead a qualitative change in the state of consciousness, and the feeling as if in 'a dream'...'stoned', 'not awake' or an indescribable 'muzzy feeling', etc. This ineffable aspect of

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* Corresponding author.
E-mail address: mauricio.sierra-siegert@kcl.ac.uk (M. Sierra).

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depersonalization sets it apart from other 'neurotic' conditions such as 'hypochondriasis', or 'conversion disorders', where vivid, detailed and often dramatic descriptions are commonplace.

> *"What has really been changed or diminished with the onset of depersonalization cannot be expressed in speech. Even educated people (as in some cases in the literature) have given no clearer description, they only used metaphors. Now here is, I think, the point to which the interest of the psychopathologist should be directed. Where normal speech proves unable to deal with an event in consciousness, one may assume that something important is there. Perhaps an underlying brain anomaly makes itself perceptible in this way. Psychopathologists have not bothered very much about this remarkable fact" (Mayer-Gross, 1935, p. 106).*

Another commonly observed feature in patients' accounts of their experience is the frequent use of the expression *'as if'* to qualify their descriptions (e.g. *'I have the feeling as if I am not really here, and as if these were not my hands'* etc.). Such expressions have been traditionally interpreted as evidence of the non-delusional (i.e. nonpsychotic) nature of depersonalization. However, the use of *'as if'* expressions is more likely to be intended as a critique regarding the adequacy of the description used, rather than a critique of the reality of the experience itself. Thus, while it is true that patients remain painfully aware of the anomalous nature of their experience, they remain convinced that a fundamental, albeit ineffable change has taken place in them.

Another conceptual problem with the use of 'unreality feelings' as a general descriptor of depersonalization is that the term introduces a negative definition which has poor explanatory value as it alludes to something missing from normal experience without clarifying its nature (Radovic & Radovic, 2002; Sierra & Berrios, 2001). Historically, there has been disagreement as to the nature of this putative 'missing experience', and different writers proposed that depersonalization stemmed from either perceptual, emotional, memory or body image related impairments. Underlying all these hypotheses is the notion that the phenomenological complexity of depersonalization could be reduced to the impairment of a single mental function.

An alternative view, that depersonalization could be best conceptualised as a syndrome rather than a symptom, became well established in the first half of the 20th century (Shorvon, 1946; Sierra & Berrios, 1997). The following description by Schilder (1928), illustrates this:

> "To the depersonalized individual the world appears strange, peculiar, foreign, dream like. Objects appear at times strangely diminished in size, at times flat. Sounds appear to come from a distance. The tactile characteristics of objects likewise seem strangely altered, but the patients complain not only of the changes in their perceptivity but their imagery appears to be altered. Patients characterise their imagery as pale, colourless and some complain that they have altogether lost the power of imagination. The emotions likewise undergo marked alteration. Patients complain that that they are capable of experiencing neither pain or pleasure; love and hate have perished with them. They experience a fundamental change in their personality, and the climax is reached with their complaints that they have become strangers to themselves. It is as though they were dead, lifeless, mere automatons. The objective examination of such patients reveals not only an intact sensory apparatus, but also an intact emotional apparatus. All these patients exhibit natural affective reactions in their facial expressions, attitudes, etc.; so that it is impossible to assume that they are incapable of emotional response".

In the above description Schilder describes four main and distinct experiential components; namely: (1) an experience of feeling cut-of or alienated from surroundings (i.e. derealization); (2) difficulties remembering or imagining things; (3) inability to feel emotions; and (4) a feeling of disembodiment, described as a feeling of being dead, or automaton-like. Interestingly, such four symptom-domains would seem to broadly correspond with those very mental functions historically deemed relevant to the genesis of depersonalization (Sierra & Berrios, 1997).

Further evidence supporting the view that depersonalization is characterised by several distinct symptoms was marshalled by a study, which compared 200 historical cases of chronic depersonalization published in the neuropsychiatric literature since the late 19th century, with 45 current patients with depersonalization disorder (DPD). The study revealed the presence of five symptoms which showed little variation between the historical and modern clinical samples (Sierra & Berrios, 2001): (1) complaints of changes in body experience; (2) automaton-like feelings (i.e. loss of feelings of agency); (3) emotional numbing; (4) changes in the subjective experience of imagery and autobiographical recollections; and (5) complaints of changes in visual perception of surroundings.

In spite of its apparent symptom diversity, it might still be the case that depersonalization could result from a single, pervasive experience of detachment equally affecting all aspects of experience. When described separately with regard to emotions, body experiencing, etc., this pervading detachment experience might give rise to the illusion of multiple symptoms. However, the fact that not all symptoms are always present; that some seem more stable than others, or show differential intensity (Sierra & Berrios, 2001), suggests that at least some of these symptoms belong to different experiential domains, with potentially distinct underlying mechanisms (Sierra & Berrios, 1998; Sierra, Lopera, Lambert, Phillips, & David, 2002). Furthermore, two recent exploratory factor analysis studies using the Cambridge Depersonalization Scale (CDS), support the view that, rather than being a one-dimensional construct, 'depersonalization' represents the expression of several distinct underlying dimensions (Sierra & Berrios, 1999; Sierra, Baker, Medford, & David, 2005; Simeon et al., 2008).

The first study was carried out on 145 DPD patients, most of whom had long-standing, constant depersonalization feelings (Sierra et al., 2005). Four well differentiated factors were found and were labelled as follows: (1) Anomalous body experience. (2) Emotional numbing. (3) Anomalous subjective recall. (4) Alienation from surroundings (i.e. derealization).

Moreover, the fact that an oblique rotation (a statistical factoring model which assumes correlation among factors), yielded a better solution than an orthogonal rotation (a model which assumes independent factors), suggests that the different components of depersonalization represent a cohesive clinical entity rather than the mere coexistence of unrelated phenomena. Recently, Simeon et al. (2008) used the CDS to carry out a confirmatory factor analysis on 450 affected subjects and obtained a strikingly similar factorial solution. Four of their five factors clearly overlapped with those found by Sierra et al. (2005). In summary, converging evidence from both historical and contemporary phenomenological analysis of depersonalization, suggests that rather than being a unitary experience (i.e. feelings of unreality), the condition is likely to represent a clinical composite of several distinct symptoms: (1) feelings of disembodiment, (2) emotional numbing, (3) anomalous subjective recall, and (4) derealization (i.e. a feeling of alienation from surroundings). According to this syndromal view, 'Depersonalization' is a generic term encompassing all the above symptoms including 'derealization'. This represents a departure from the prevalent assumption, which considers depersonalization and derealization are distinct independent conditions. What follows describes each of the constituent symptoms of depersonalization in some detail.

1.1. Disembodiment feelings

Patients with depersonalization complain of a variety of related changes in body experience, which can be conceptualised generically as 'disembodiment'. These are (1) Lack of body ownership feelings. (2) Feelings of loss of agency, which refer to the feeling that actions happens automatically without the intervention of a willing self. (3) Experience of disembodiment, which can range from a non-specific feeling of not being in the body, and heightened self-observation, to out of-body experiences, and autoscopic hallucinations. The latter two, however, are rare in depersonalization (Gabbard, Twemlow, & Jones, 1982). (4) Somatosensory distortions usually affecting the size of body parts or feeling very light have not been found to be characteristic of depersonalization and may be useful in the differential diagnosis with conditions such as schizophrenia, epilepsy or migraine, where somatosensory distortions are said to be frequent (Priebe & Rohricht, 2001; Rohricht & Priebe, 2002; Watanabe, Takahashi, & Tonoike, 2003). Interestingly, these profound subjective distortions in body image do not seem accompanied by objective changes in body schema (i.e. implicit regulation of posture and movement in relation to surrounding space (Cappon & Banks, 1965).

1.2. Emotional numbing

Most patients with depersonalization report different degrees of attenuated emotional experience such as loss of affection, pleasure, fear or disgust. Some patients describe an absolute inability to experience emotional states, others describe a more subtle impairment characterised by an inability to experience emotional feelings which normally colour perception and mental activity. It has been suggested that the latter impairment may be causally related to descriptions of things looking 'unreal' (Sierra & Berrios, 1998). Indeed, the narratives of patients often suggest that this might be case: "[as I hear music] there is no response in me. Music usually moves me, but now it might as well be someone mincing potatoes ... I seem to be walking about in a world I recognise but don't feel. I saw Big Ben alight last night, normally a moving sight to me, but it might have been an alarm clock for all I felt ... My husband and I have always been happy together but now he sits here and might be a complete stranger. I know he is my husband only by his appearance – he might be anybody for all I feel towards him" (Bockner, 1949). Such statements would seem to suggest that what seems more affected in depersonalization is the ability to imbue perceived objects or concrete situations with emotional feeling, rather than a general inability to experience emotional states (Sierra & Berrios, 1998). A related complaint is that of an inability to experience empathy and compassionate feelings. Lawrence et al. (2007) compared 16 DPD patients with 48 healthy controls along a series of tests designed to provide a measures of two types of empathy: cognitive and affective. In short, while cognitive empathy reflects the capacity to understand another person's emotional state, 'affective empathy' reflects the ability to experience a congruous emotional response. The main findings of this study was that while patients with depersonalization showed an intact performance on cognitive empathy, there was evidence of a disruption in implicit physiological concomitants of affective empathy.

Comparable findings emerged from a study looking at the emotional responses to emotive pictures of patients with DPD as compared with normal controls and anxiety disorder patients. Although patients with depersonalization did not experience any difficulties when rating the unpleasantness of pictures on a scale, they showed attenuated autonomic responses to arousing pictures and rated them as subjectively less arousing (Sierra et al., 2002). Just as it seems to be the case with anomalies of body experience, subjective complaints of emotional numbing are usually accompanied by a normal array of emotional motor expression. Such dissociation is important in the differential diagnosis given that in other conditions in which emotional numbing can be seen, such as in schizophrenia, depression or PTSD, subjective complaints are accompanied by impoverished emotional expression. In this regard, emotional numbing in depersonalization has shown itself to be a distinct and robust psychopathological concept which can be differentiated from anhedonia (Mula et al., 2010).

1.3. Anomalous subjective recall

Patients with DPD often complain of subtle subjective impairments affecting recall and imagery. Although the ability to retrieve information seems unaffected, patients frequently complain that memories, particularly of personal events (i.e. episodic memory) seem to have lost any personal meaning: *"I can remember things, but it seems as if what I remember did not*

really happened to me''. Such complaints would seem to correspond to a dissociation between what have been termed the 'know/remember' components of autobiographical memories (Gardiner & Java, 1991). In short, in addition to the retrieval of factual information about a personal event (i.e. a factual or 'know' component), the act of remembering also entails an awareness or particular feeling, that the experience recalled actually happened in the past and is not just being imagined or the memory of a dream. Unlike the case with 'psychogenic amnesias', the 'factual' aspect of the memory is preserved in depersonalization while it is the 'remembering' component which becomes disrupted in some patients. A recent study on 14 patients with DPD found that although patients did not differ from controls in a free recall performance task after watching a movie clip, they exhibited subjective and objective memory fragmentation as measured by their inability to sequence in temporal order a series of scenes extracted from the watched clip (Giesbrecht, Merckelbach, van Oorsouw, & Simeon, 2010).

Another common clinical observation is that autobiographic memories in depersonalization are usually remembered from a vantage point outside of the body. That is, the event is visualised as if it had been witnessed from outside, rather than through the person's own eyes. This type of memory distortion, which has been called 'observers perspective' remembering (Nigro & Neisser, 1983), has been shown to affect the recall of traumatic situations, or situations which were experienced as threatening (Sierra and Berrios, 1999). Kenny and Bryant (2006), investigated the relationship between memory vantage point and avoidance following trauma in 60 trauma survivors with differing levels of avoidance. It was found that avoidant individuals were more likely to remember their trauma from an observer perspective than individuals with a lower level of avoidance. Interestingly, avoidance did not influence vantage point for positive or neutral memories. These results support the view that the adoption of the observer vantage point for unpleasant memories may serve an avoidant function for people affected by trauma. Similar results have been reported in regards to distressing memories in depression (Williams & Moulds, 2006), and memories related to social interactions in social phobics (D'argenbeau et al., 2006).

A related complaint affecting memory is that depersonalized patients often characterise their imagery as pale, colourless or completely absent. Lambert et al. (2001b) assessed visual imagery in 28 patients with depersonalization disorder using the Vividness of Visual Imagery Questionnaire (VVIQ) and the Vividness of Movement Imagery Questionnaire (VMIQ). The former is a 16-item scale consisting of descriptions of visual scenes that the subject is asked to imagine, and rate on a 5-point scale ranging from 1 = 'perfectly clear and as vivid as normal vision' to 5 = 'no image at all'. The VMIQ, in turn is a 24-item scale consisting of movements that the subject is requested to imagine. Using the same 5-point scale as above, the items of this questionnaire request subjects to imagine somebody else performing a movement, and then to repeat the items this time imagining that they are themselves making the movements. As compared with a group of age and sex matched normal controls the depersonalization patients were found to have a significant impairment of imagery on both the VVIQ and the VMIQ measures. Interestingly, patients showed more impairment on the VVIQ with those items requesting to imagine situations involving people as opposed to objects or scenery. On the VMIQ patients were more impaired at imagining *themselves* making movements, as compared with imagining *another person* making the same movement. In fact, this difficulty to imagine oneself making movements was found to correlate significantly with the intensity of depersonalization as measured by the DES-Taxon. There was however a potential confounding contribution from depressed mood as the latter also correlated with impaired ability to generate visual images. A subgroup of 10 patients was further tested on a neuropsychological battery of visual perception tests and found to be unimpaired compared with normal controls and patients with obsessive compulsive disorder, despite subjective impairments in imagery (Lambert, Senior, Fewtrell, Phillips, & David, 2001a).

1.4. Derealization

Most patients with depersonalization describe feelings of being cut-off from the world around, and of things around seeming 'unreal'. Such an experience is frequently described in terms of visual metaphors (e.g. looking through a camera, mist, veil, etc.). The term derealization was coined in 1935 and ascribed to Mapother by Mayer Gross. Although it has been suggested that apparent phenomenological differences between depersonalization and derealization might simply reflect different descriptive angles of the same experience rather than different phenomena (Sierra et al., 2005), it might be argued that there are genuine phenomenological differences between symptoms pertaining to body, emotional and memory experiencing, and that of perception of surroundings.

'Derealization' commonly accompanies all the other symptom domains of DPD, and its isolated occurrence has been questioned or reported as extremely rare. Thus, Coons reported to have found only two papers which suggested that derealization can occur alone (Rosen, 1955; Krizek, 1989), but careful reading of these two case reports suggests that symptoms of derealization and depersonalization occurred together in both patients. Lambert et al. (2001a) found that among 44 patients with depersonalization derealization syndrome only four suffered from ''pure derealization''.

Depersonalization has been shown to correlate with anxiety measures (Trueman, 1984), and patients with a diagnosis of DPD, a condition characterised by chronic depersonalization are often found to have high levels of anxiety (Baker et al., 2003). Additionally, it has been observed that the onset of depersonalization often coincides with stressing life-events or even life threatening situations. This has been interpreted as suggesting that depersonalization represents an anxiety-triggered 'hard wired' inhibitory response intended to ensure the preservation of adaptative behaviour during situations normally associated with overwhelming and potentially disorganizing anxiety (Sierra & Berrios, 1998). It has been proposed that

such inhibitory response is mediated by a fronto-limbic suppressive mechanism, which would generate a state of emotional numbing, and disable the process by means of which perception (including that of one's own body), as well as cognition become emotionally coloured. Such 'decolouring' will result in a "qualitative change" of conscious awareness, which is then reported by the subject as "unreal or detached". In patients with DPD this response would become abnormally persistent and dysfunctional (Sierra & Berrios, 1998). Studies carried out during the last decade seem supportive of this model.

2. Psychophysiological studies

Lader and Wing (1966) first reported anecdotal observations in patients with pathological anxiety in whom the onset of depersonalization coincided with a dramatic flattening of their previously labile galvanic skin responses. An even earlier work, looking at the psychophysiological effects of repeated electrical shocks on healthy subjects, had noted that, at the time of receiving high intensity shocks, subjects often described feelings of derealization or of becoming detached observers of themselves. Coinciding with this, there was a blunting in their skin conductance recordings (Oswald, 1959). A less dramatic confirmation of such findings has recently been obtained. Sixty-nine undergraduate students were exposed to a succession of 19 aversive auditory probes while their skin conductance responses were measured. It was found that the occurrence of acute dissociative experiences (including depersonalization), during the experiment was associated with a fast attenuation of skin conductance responses (Giesbrecht, Merckelbach, ter Burg, Cima, & Simeon, 2008).

Kelly and Walter (1968), were probably the first to study the sympathetic autonomic system in patients suffering with continuous, chronic depersonalization states. Using forearm blood flow as a measurement of sympathetic autonomic function they found that a group of 8 'depersonalized patients' had the lowest 'basal' recordings as compared with a whole range of clinical control groups including chronic anxiety, and depression. Although the highest basal autonomic activity was observed in the chronic anxiety group (almost four times higher than observed in the depersonalization group), both groups had similarly high subjective anxiety. The authors concluded: "The evidence suggests that the discrepancy between subjective and objective signs of anxiety is the fundamental characteristic of patients with depersonalization. In physiological terms, anxiety is experienced but is not translated into defence reaction arousal" (Kelly & Walter, 1968).

Sierra, Senior, et al. (2002) tested the prediction that the observed attenuation of sympathetic autonomic arousal was selective to emotional stimuli as compared with neutral and non-specific stimuli. The skin conductance responses of 15 patients with DPD, 15 controls, and 11 individuals with anxiety disorders were recorded in response to non-specific elicitors of electrodermal responses (an unexpected clap and taking a sigh) and in response to pictures with neutral and both pleasant and unpleasant emotional contents. It was found that the depersonalization patients had selectively reduced autonomic responses to unpleasant pictures but not to neutral or pleasant ones (the response to these was also reduced but the difference was not statistically significant). Further, the latency of response to these stimuli was significantly prolonged in the group with DPD. In contrast, latency to non-specific stimuli (clap and sigh) was significantly shorter in the depersonalization and anxiety groups than in the healthy controls. These findings suggested the presence of both inhibitory and facilitatory mechanisms on autonomic arousal, which suggests a specific disruption in emotion processing rather than a non-specific dampening effect on autonomic reactivity.

Another related study compared the skin conductance responses (SCR) of DPD patients with those of anxiety disorder patients and normal controls as they watched pictures and video clips of facial expressions of disgust and happiness (Sierra, Senior, Phillips, & David, 2006). In keeping with Kelly and Walter's study it was found that while patients in the anxiety group had increased autonomic reactivity to disgust expressions, depersonalization patients had very similar responses to those of the healthy controls in spite of showing similarly high anxiety scores to those of the anxiety group. In other words it would seem that the blunting effect on autonomic responses seen in depersonalization is not absolute but relative to anxiety levels. Another recent study compared the skin conductance level (SCL) of DPD patients and healthy controls as they watched an arousing scene from the horror movie 'The Silence of the Lambs'. As compared with healthy controls, depersonalized participants exhibited an earlier peak followed by subsequent flattening of SCLs which failed to return to baseline levels after termination of the stimulus. The existence of both excitatory and inhibitory influences on autonomic responses in depersonalization finds further support in a study, which compared levels of urinary norepinephrine of patients with DPD and healthy controls. In keeping with their higher anxiety levels, patients with depersonalization were found to have higher levels of norepinephrine than healthy controls. However, within the depersonalization group there was a striking negative correlation between norepinephrine and depersonalization scores (Simeon, Guralnik, Knutelska, Yehuda, & Schmeidler, 2003). It seems plausible to suggest that autonomic responses in patients with depersonalization are likely to reflect a balance between two opposing tendencies. One excitatory determined by anxiety levels, and an inhibitory one determined by depersonalization intensity.

3. Functional neuroimaging studies

Over the last decade a number of functional neuroimaging studies have revealed abnormal brain activation patterns which seem functionally related to both the autonomic changes and subjective experiences already discussed. One of the first studies used positron emission tomography (PET) to compare patterns of brain activation of 8 patients with DPD patients with normal controls as they performed a verbal memory task (Simeon et al., 2000). Although patients showed reduced metabolic activity in some association areas such as the right superior and middle temporal gyri, other areas, such

as the parietal, and occipital lobes were more active than the controls. Interestingly, one of the most significant findings was that of an abnormally increased activation in the angular gyrus of the right parietal lobe, which correlated ($r = 0.7$) with ratings of depersonalization intensity. The potential significance of abnormal parietal functioning in depersonalization is further suggested by a recent open label trial using low frequency repetitive transcranial magnetic stimulation (TMS) on the right temporoparietal junction in 12 patients with DPD (Mantovani et al., 2010). It was found that after 3 weeks treatment half of the patients showed significant improvement.

Experimental neuroimaging studies on the neural correlates of embodiment and agency feelings, have identified a network of parietal regions, which appear to play an important role in the generation of embodiment and agency feelings: the inferior parietal cortex, the temporoparietal junction, and the posterior insula. Increased activation in the angular gyrus has been observed in patients experiencing a lack of agency feelings regarding movement or the experience that movements are being controlled by an external agency (Farrer et al., 2004; Frith, Blakemore, & Wolpert, 2000). As mentioned above, subjective experiences of lack of agency feelings are often reported by patients with depersonalization. It is currently believed that the right angular gyrus computes discrepancies between intended action and subsequently experienced movement, allowing any detection of mismatch to be consciously experienced (Farrer et al., 2004, 2008). It is likely that the experience or observation of movements which do not feel as arising from the self elicits an attentional orientation response, similar to that elicited by unexpected events.

In addition to the angular gyrus, the posterior insula has also been shown to play a significant role in the integration of different input signals related to self-awareness. For example it has been shown that decreased activity in this region corresponds with a decreasing feeling of movement control. Thus, subjects with minimal posterior insula activation report such a striking absence of feelings of agency that when they move it feels to them that they are watching movements of another person (Farrer & Frith, 2002; Farrer et al., 2003). In keeping with these findings, studies in stroke patients have shown that lesions to the posterior right insula are associated with lack of ownership feelings regarding the existence, or activity of contralateral limbs (Baier & Karnath, 2008; Karnath et al., 2005).

In summary, studies on the neurobiological underpinning of agency feelings have shown that while posterior insula activation correlates with the degree of self-attribution of movement, the angular gyrus in the inferior parietal cortex shows the opposite pattern so that the lower the sense of agency the greater the activity in the right inferior parietal lobe (Farrer & Frith, 2002; Farrer et al., 2003, 2008). Another related and partially overlapping parietal region, the temporoparietal junction, has been shown to play an important role in the experience of embodiment, and both pathology and electrical stimulation of this area can generate out-of-body experiences (Blanke, Landis, Spinelli, & Seeck, 2004; De Ridder, Van Laere, Dupont, Menovsky, & Van de Heyning, 2007).

Other studies using functional neuroimaging have been designed to explore the role of emotional numbing in depersonalization. The first of those studies used functional magnetic resonance imaging (fMRI) to compare the neural response of patients with DPD with that of healthy volunteers and patients with obsessive compulsive disorder. Participants were scanned as they watched a series of aversive and neutral pictures extracted from a well known and standardised picture set (the international affective picture system; IAPS). Attesting to the presence of subjective emotional numbing, depersonalized patients stated that although they could understand the content of the pictures they failed to experience any subjective emotional response. It was found that while healthy volunteers as well as obsessive compulsive disorder (OCD) patients showed activation of the anterior insula in response to unpleasant and disgusting pictures, such activation was not observed in DPD patients. Intriguingly, the anterior insula has been found to play an important role in the neurobiological underpinnings which allow autonomic body states to be consciously experienced as emotional feelings (Craig, 2009).

Another key finding of this study was that depersonalization patients, but not the controls, showed an area of activation in the right ventrolateral prefrontal cortex (BA 47). This region seemed functionally coupled with the insula. Indeed, during the presentation of unpleasant stimuli there was evidence of an inverse correlation, so that prefrontal activation only occurred in the absence of insula activation. Interestingly, the prefrontal area in question has been implicated in the evaluation of negative or aversive information and on exerting control over both emotional experience and its impact on decision making (Beer, Knight, & D'Esposito, 2006).

A recent fMRI study has marshalled further evidence of a fronto-limbic inhibitory mechanism in DPD, and also has shown how it might relate to the finding of attenuated autonomic responses (Lemche et al., 2007, 2008). The researchers used event-related fMRI and simultaneous skin conductance measures to compare the neural responses of 9 patients with DPD and 12 healthy controls as they viewed pictures of faces showing different intensities of sadness and happiness. The first finding of the study was that depersonalized patients showed decreased activity in the amygdala and hypothalamus which was greater with increased emotional expression. Normal controls, in turn, yielded an opposite pattern. The second key finding was that, in the depersonalization patients but not in the controls, activity in a region of the dorsolateral prefrontal cortex (BA 9) previously implicated in emotional regulation was negatively correlated with autonomic response, hence suggesting an inhibitory role on limbic functioning.

Yet another fMRI study has provided further evidence that, in keeping with their subjective complaints of emotional numbing, patients with DPD show abnormalities in the processing of emotionally salient material (Medford et al., 2006). The authors predicted that patients would not show the well established enhancing effect that emotion has on memory. It was also expected that patients would not show activation of emotion-related brain regions during encoding and recognition of emotional words. The authors compared 10 DPD patients with healthy controls while they performed an emotional memory test. It was found that while controls activated a number of emotion-relevant brain regions including the right

amygdaloid complex, hippocampus and the left temporal gyrus and anterior insula, none of these areas were activated in depersonalization patients. In fact this group showed no differences in brain activation in response to emotional vs. neutral words.

In summary, in keeping with patients' subjective complaints of non-existent or attenuated subjective emotional experiencing, fMRI findings suggest that depersonalization is consistently characterised by reduced activity in emotional-related areas such as the amygdala and the anterior insula as well as attenuated autonomic responses to arousing emotional stimuli.

An inhibitory mechanism mediated by the prefrontal cortex on limbic structures and the anterior insula might explain some of the experiential aspects of depersonalization. In particular, an impairment of the process whereby emotional experience becomes integrated with perception might result in a 'qualitative change' on subjective conscious experiencing best described by those affected as 'feelings of unreality'. One hypothesis arising from this model is that disabling this inhibitory network with, for example TMS, should improve the symptoms of depersonalization.

The idea that the 'immediacy', and 'vividness' (i.e. 'feelings of reality') of experience might be determined by concomitant emotional feelings has been previously suggested in the literature. For example, as early as 1925 MacCurdy wrote: 'the feeling of the reality attaching to any idea is proportionate to our emotional interest in it. Loss of the feeling of reality is, then only a manifestation of loss of interest which is, in turn, related to the loss of energy and stimulus susceptibility' (MacCurdy, 1925, p. 126). This idea has found support in neurophysiological and cognitive studies. For example, it has been shown that a reduction in the affect attached to a memory can cause it to be experienced as if one had been a detached external observer at the time rather than a direct participant (Robinson & Swanson, 1993). Similarly, as described above, when evoking personal recollections patients with depersonalization often complain that memories feel as if they really didn't happen to them. In other words, autobiographic memories retain their factual aspect (i.e. their informational load) but seem devoid of the distinct feeling that accompanies the act of remembering. A feeling that seems emotional in nature to a significant extent (Sharot, Delgado, & Phelps, 2004).

It is worth noticing that lack of 'emotional colouring' might also be related to feelings of 'unreality' regarding one's own body. In this regard, it is perhaps not surprising that those body parts which tend to evoke a greater emotional resonance (e.g. face, hands) are the ones most commonly reported as 'feeling unreal' by patients with depersonalization (Shorvon, 1946), as the following case reported by Schilder (1935, p. 139): *"[Depersonalization] occurs, as I have shown, especially in organs which have previously been of a great erotic significance. I have observed a singer who showed depersonalization concerning speech and concerning the mouth, an organ to which she paid special attention, in herself as well as in others".*

The view that 'emotional feelings' may be a core experiential component of perception rather than just a reaction to it has been a neglected idea in neuropsychology. It is likely, however, that in addition to a pathway of information processing leading to semantic recognition (a 'what is it? perceptual function), there is a parallel pathway in charge of assigning emotional significance to percepts (Halgren & Marinkovic, 1994). Such an 'emotion colouring' mechanism is likely to be a major contributor to feelings usually described in terms of 'immediacy', 'atmosphere' or 'vividness' (Gloor, 1990). There is evidence suggesting that these two parallel functions take place pre-consciously (Halgren & Marinkovic, 1994), which may explain why, when perception becomes conscious, it is already 'emotionally coloured' (Halgren, 1992). Neuropsychological evidence is compatible with the view that these cognitive and emotional components of perception are independent of one another. On the one hand, a disruption of perceptual identification with preservation of emotional response has been demonstrated for example in subjects with prosopagnosia, who in spite of not being able to consciously recognise pictures of relatives, show evidence of implicit emotional recognition as measured by autonomic responses (Tranel & Damasio, 1985). Similarly, it has been reported that electrical stimulation targeting temporal lobe structures such as the amygdala, often trigger hallucinatory phenomena, which although fragmentary and lacking in perceptual detail, are experienced vividly and with a strong feeling of reality and personal relevance. This has been explained in terms of the simultaneous presence of an emotional component that colours the experience (Gloor, 1990). On the other hand, a failure to display normal autonomic responses in the face of intact perceptual recognition has been shown in patients with Capgras syndrome (Brighetti, Bonifacci, Borlimi, & Ottaviani, 2007; Ellis, Young, Quayle, & De Pauw, 1997; Hirstein & Ramachandran, 1997). Thus, while these patients do not experience any problems at identifying relatives, they claim that the person in question does not feel genuine and must be an impostor. The accompanying lack of autonomic responses is revealing in that recent evidence shows them to be instrumental to the conscious experiencing of familiarity feelings (Morris, Cleary, & Still, 2008). It has been suggested that the lack of familiarity feelings in Capgras arises from a functional disconnection between face-processing areas in the temporal lobe and the limbic system (Hirstein & Ramachandran, 1997). Similar phenomena have been described with regard to a wide range of objects in addition to people, such as buildings, places, and objects. (Abed & Fewtrell, 1990; Benson, Gardner, & Meadows, 1976).

Patients with depersonalization seem to experience a similar, non-delusional version of this phenomenon. "When I look at my parents I know who they are, but at the same time they feel different, as if they were people I don't really know". In fact a noted high prevalence of depersonalization in patients with Capgras syndrome or reduplicative paramnesia has been interpreted as suggesting that the latter represent a delusional elaboration of depersonalization experiences (Christodoulou, 1986).

The neurobiological structures and mechanisms in charge of assigning emotional significance to percepts, and the ensuing generation of 'emotional colouring', is still far from being completely understood. It is clear, however, that the amygdala plays an important role. It has been clearly established that the amygdala is crucial for the perception of threat as well as the integration of fear responses. Humans with amygdala lesions show impaired aversive conditioning learning (Bechara et al., 1995; LaBar, LeDoux, Spencer, & Phelps, 1995), seem incapable of recognising facial expressions of fear (Adolphs,

Tranel, Damasio, & Damasio, 1994; Calder et al., 1996; Young et al., 1995), voice intonation expressing fear and anger (Scott et al., 1997), and the recognition of sad or scary music (Gosselin, Peretz, Johnsen, & Adolphs, 2007). In addition to fear-related functions, the amygdala also seems to have a more generic role in the processing and assignment of emotional significance, as well as to play a modulatory role on cognitive functions such as attention, perception and memory (LeDoux, 2007). Neuroimaging studies also show activation of the amygdala during the recall of emotionally charged memories (Cahill, Haier, Fallon, et al., 1996), and it would seem that such activation contributes to the 'feeling' of remembering (Sharot et al., 2004). The fact that in patients with depersonalization a lack of subjective emotional feelings coexists with adequate behavioural emotional expressions gives support to the idea that in depersonalization there is a disruption of the process which allows emotions to gain conscious representation, rather than a global dysfunction of emotion processing.

Recent research has identified the anterior insula as a crucial cortical region necessary for the experience of emotional feelings (Critchley, 2005; Morris, 2002). Such findings are in keeping with the reduced insula activation found in patients with DPD (Phillips et al., 2001). From an anatomical perspective the insula seems to be well placed to integrate signals from a variety of sources. It receives visceral, somatosensory, visual, auditory and gustatory inputs, and has extensive reciprocal connections with the amygdala, hypothalamus, cingulate gyrus and orbitofrontal cortex (Höistad & Barbas, 2008; Mesulam & Mufson, 1985). It has been proposed that one of the main functions of the anterior insula would be that of integrating peripheral autonomic responses with central 'cognitive' processing, allowing visceral responses to gain conscious representation in the form of subjective feelings (Morris, 2002). It has been shown that subjects with a condition known as pure autonomic failure, who are unable to generate autonomic responses, have a reduced capacity to experience conscious feeling including empathy (Chauhan, Mathias, & Critchley, 2008; Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004). Furthermore, it has been experimentally shown in fMRI studies that the ability to experience feelings in response to emotional pictures is directly related to activity in the anterior insula. In particular, it would seem that an inability to become aware of feelings is related to hypoactivity in the anterior insula (Silani et al., 2008). Anterior insula activation has been related to whole range of emotional feelings such as disgust, sadness, fear, reward experiences, categorization of facial emotional expressions, craving, and hunger or satiety states (Morris, 2002). It is also been involved in the experience of socially laden feelings such as the sense of 'fairness' (Moll et al., 2007). Such a vast array of activation correlates suggests that the role of the anterior insula in the generation of feelings is generic rather than specific to any particular emotion.

In light of the evidence reviewed so far it would seem as if there are two distinct neural networks that may be relevant to the neurobiology of depersonalization. The first system, relevant to the experience of emotional feelings, includes the amygdala, the anterior insula and possibly other limbic-related structures such as the hypothalamus and the anterior cingulate. The activity of this emotional system is strongly regulated by the prefrontal cortex, and it is suggested that in depersonalized subjects abnormal prefrontal regulatory suppression might be responsible for emotional numbing and the related inability to colour experience with feelings. This hypothesis is supported with the findings discussed above of attenuated autonomic responses, underactive amygdala and anterior insula responses, as well as related increased activation in prefrontal regions in depersonalized subjects. Of the four different symptom domains of the depersonalization syndrome reviewed above, it would seem that three of them, namely 'derealization', 'anomalous subjective recall', and 'emotional numbing', might be related to fronto-limbic suppression.

A second neural network relevant to the experience of embodiment and feelings of agency, may be implicated in feelings of disembodiment, lack of body ownership and lack of agency feelings experienced by patients with DPD. The idea that the depersonalization syndrome might be mediated by anomalies in two distinct albeit related networks (i.e. fronto-limbic and parietal) is supported by the observation that patients with distinct neurological lesions may complain of either derealization or 'disembodiment'. The phenomenological similarities between visual hypoemotionality, a neurological syndrome, which allegedly results from a cortico-limbic disconnection involving visual pathways (Bauer, 1982), and derealization, supports the idea that a disruption of the processes by which perception becomes emotionally coloured may be an underlying mechanism in both conditions. Likewise, phenomenological overlaps with asomatognosia suggest that the 'disembodiment' component of depersonalization might result from parietal mechanisms disrupting the experience of body ownership and agency (Sierra, Lopera, et al., 2002).

It any event, the evidence suggest that the neural networks supporting body and emotional representation are closely linked. For example, a study using structural neuroimaging on 108 subjects with focal brain lesions found that the perception of intensity in facial emotional expressions required the integrity of the right somatosensory cortices (Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000). Future studies on neurobiological mechanisms of depersonalization should address the relationship between emotion regulation systems and those underpinning embodiment and agency feelings.

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