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Attributions, appraisals and attention for symptoms in depersonalisation disorder

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ABSTRACT

A cognitive-behavioural model of depersonalisation disorder (DPD) suggests that catastrophic attributions and appraisals, and increased attention to symptoms, play important roles in the development and maintenance of the disorder. Empirical testing of this model was investigated in three groups: 25 patients with DPD, 21 patients with anxiety (obsessive-compulsive or panic disorder), and 22 psychiatrically healthy participants. Task 1 examined attributions for ambiguous symptoms. Task 2 used a questionnaire to compare the groups on the content, frequency, and conviction in appraisals when participants worried about their health. Task 3 employed four experimental manipulations designed to either increase, or decrease, attention to catastrophic appraisals and/or symptoms of DPD. Results indicate that the DPD group make less normalising attributions for symptoms (Task 1) and have more catastrophic appraisals (Task 2) than those in the Healthy Control group. The DPD and Anxiety groups were similar in their patterns of appraisals and attributions. In Task 3, the DPD group showed a perceived reduction in DPD severity when their attention was focussed on cognitively demanding tasks, whereas the other two groups showed an increase. The findings are consistent with the hypothesis that these cognitive processes play an important role in the development and maintenance of DPD.

Introduction

Depersonalisation disorder (DPD) is a chronic and distressing condition characterised by a sense of unreality about the self (Depersonalisation: DP) and/or the external world (Derealisation: DR). Those with DPD typically describe being detached from their experiences as if living in a dream, as well as feeling emotionally and physically numb. The world may appear artificial, two dimensional, lacking in significance and other people may appear like actors or robots. These experiences are not delusional since the person with DPD retains insight that these are subjective phenomena, rather than objective reality. Moreover, there is a relative absence of any notable aberrations in general cognitive functioning in those with DPD (Guralnik, Schmeidler, & Simeon, 2000).

Symptoms of DP/DR are common in non-clinical and psychiatric populations (Hunter, Sierra, & David, 2004). In non-clinical populations, DP/DR frequently occur as transient experiences, particularly under conditions of fatigue or trauma (Noyes & Kletti, 1977; Sedman, 1966; Shilony & Grossman, 1993) or when under the influence of recreational drugs such as 'ecstasy' or cannabis (Mathew, Wilson, Humphreys, Lowe, & Weithe, 1993; McGuire, Cope, & Fahy, 1994; Medford et al., 2003). Prevalence rates for clinically significant current levels of DPD in representative community surveys vary from 1 to 2% in the UK (Bebbington, Hurry, Tennant, Sturt, & Wing, 1981; Bebbington, Marsden, & Brewin, 1997; Lee, Kwok, Hunter, Richards, & David, 2013), 1.9% in Germany (Michal et al., 2007) and 2.4% in North America (Ross, 1991). Within psychiatric samples, symptoms of DP/DR have been reported in up to 16% of a sample of inpatients seen in order of admission (Latz, Kramer, & Hughes, 1995), 30% of war veterans with PTSD (Davidson, Kudler, Saunders, & Smith, 1990), 60% of patients with unipolar depression (Noyes, Hoenk, Kuperman, & Slymen, 1977) and 83% of patients with panic disorder (Cox, Swinson, Endler, & Norton, 1994).

A psychophysiological theory of DPD (Sierra & Berrios, 1998) suggests that extreme anxiety may trigger changes to the functioning of specific neurochemicals and/or brain regions that are involved in the control and expression of emotional responses. Psychoanalytic theories have suggested that DPD is a defence mechanism to protect the ego from internally generated

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psychodynamic conflicts (Horney, 1951; Schilder, 1939; Torch, 1987), whereas more recent psychodynamic theories suggest dissociative responses, including DPD, may protect the person from the impact of external factors such as childhood emotional abuse (see Simeon & Abugel, 2006 for a review).

Many studies have highlighted the strong associations between anxiety and DPD (see Hunter, Phillips, Chalder, Sierra, & David, 2003 for a review). These similarities are useful for the development of theoretical models and clinical interventions for DPD, as research and treatment of anxiety disorders are more advanced than for dissociative disorders. The cognitive-behavioural model of DPD (Hunter et al., 2003) is similar to misappraisal CBT models of anxiety disorders, particularly panic (Clark, 1986) and health anxiety (Warwick & Salkovskis, 1990), where the central process is the catastrophic misinterpretation of common and benign symptoms as more threatening than they really are. Just as anxiety symptoms can be maintained and exacerbated by negative cognitions and behaviours according to CBT models, so too can the common, transient symptoms of depersonalisation and/or derealisation become chronic depersonalisation disorder by similar processes.

In CBT models of anxiety, misinterpretations are characterised as catastrophic *appraisals* of the meaning and consequences of recently experienced symptoms and are linked to catastrophic *attributions* as to their cause (Salkovskis, 1996; Salkovskis, Warwick, & Deale, 2003). Empirical studies have demonstrated the validity of this approach to the understanding of anxiety related symptoms. For example, people with panic disorder were found to catastrophically appraise bodily sensations when compared to people with other anxiety disorders and non-patient controls (Butler & Mathews, 1983; Clark et al., 1997; Harvey, Richards, Dziadosz, & Swindell, 1993; McNally & Foa, 1987) and catastrophic appraisals increased the severity and number of symptoms (Westling & Öst, 1993). Similarly, the role of attributions about the possible cause of symptoms is likely to have a significant impact. Robbins and Kirmayer (1991) categorised the types of attributions that could be made for common physical symptoms into three types: normalising, somatic or psychological. Sensky and colleagues have carried out a series of studies to examine the role these types of attributions may play in anxiety (MacLeod, Haynes, & Sensky, 1998; Sensky, 1997; Sensky, MacLeod, & Rigby, 1996). These studies showed that anxious participants were less able to find normalising attributions for ambiguous symptoms presented to them but instead gave more psychological attributions. The most recent study from this group (MacLeod et al., 1998) found that the first attribution type generated was important. This may be as the search for further explanations tends to be terminated if the first response appears plausible (Shaklee & Fischoff, 1982).

Research into catastrophic appraisals and attributions in anxiety have demonstrated that these are not merely epiphenomena, nor a consequence, of the disorder, but represent a predisposing vulnerability and its "online" manifestation as active misinterpretation. In order to demonstrate this it is necessary to extend studies beyond correlational observations by showing that the induction and inhibition of catastrophic appraisals and attributions result in a respective increase, and decrease, in the symptoms experienced. Clark et al. (1988) induced significant levels of anxiety in participants with a history of panic attacks by asking them simply to read aloud a list of bodily symptoms that were paired with typical catastrophic appraisals of panic. Another experimental method of increasing anxiety by activating catastrophic appraisals and attributions is to use paradigms that increase symptom monitoring by asking participants to specifically focus on their bodily sensations (e.g. Haenen, Schmidt, Kroeze, & van den Hout, 1996). Conversely, one would predict that if participants engaged in a cognitively demanding task, this would inhibit their ability to generate catastrophic cognitions and symptom monitor, which would be reflected in a decrease in perceived symptomatology.

The cognitive-behavioural model of DPD (Hunter et al., 2003) uses a similar approach to the catastrophic misinterpretation models of anxiety described above, but with DPD specific cognitions and behaviours. This model has been updated and is shown in Fig. 1.

This DPD model shows that there are a number of triggers from empirical research that can give rise to the experiences of DP/DR. However, given that brief experiences of DP/DR are common in the general population, the question arises as to how these can become chronic as in DPD? One answer might be in the appraisals and attributions that are ascribed to these experiences. If the person attributes 'normalising' attributions to DP/DR phenomena, the latter will be viewed as benign, be ignored, and the phenomena are likely to decrease in severity. However, the DPD model suggests that if person generates catastrophic attributions and appraisals for the naturally occurring symptoms of DP/DR, these may lead to the development of a vicious cycle of emotional, behavioural and cognitive responses which are likely to maintain and exacerbate the initial symptoms. For example, these catastrophic attributions and appraisals may lead to emotional responses such as increased anxiety and depression that interact with, and exacerbate, the original DP/DR symptoms. Behavioural responses might include an avoidance of certain situations which the person predicts will worsen symptoms, as well as behaviours which he or she believes help prevent the feared outcome (i.e. 'safety seeking behaviours'). Moreover, catastrophic cognitions and emotional responses may provoke cognitive *processes*, such as changes to attention with an increased focus on symptoms. As research in anxiety disorders demonstrates, this increase in symptom monitoring may create a feedback loop in the model due to the increased likelihood in the initial perception of symptoms, and a reduced threshold for the perception of threat. In this way, transient experiences can develop into a chronic disorder.

However, it may be that some people have a predisposition to react to stressful and anxiety provoking situations with DP/DR, perhaps because of an unusual lack of autonomic responsiveness to arousing stimuli (Sierra et al., 2002), and given that our understanding of the psychobiology of DPD remains somewhat limited, there may be other mechanisms that trigger and maintain these symptoms. Nevertheless, it is also likely that the changes in emotions, behaviour and attention created by any catastrophic attributions and appraisal will exacerbate the DPD directly. As with other disorders which may have an unknown underlying physical and/or neurological aetiology (chronic fatigue syndrome being a good example), CBT models have been valuable in understanding the disorder-specific cognitions and behaviours which serve to exacerbate the initial symptoms.

Previous investigations to systematically examine and manipulate cognitive processes in DPD that might serve to maintain the problem have been extremely limited. Apart from experiments where DP/DR symptoms have been induced in healthy controls by asking participants to narrow their focus of attention by staring at a dot on a wall for a few minutes (Leonard, Telch, & Harrington, 1999; Miller, Brown, DiNardo, & Barlow, 1994), to the authors' knowledge, no previously published empirical study has attempted to induce, or inhibit, attributions, appraisals and attentional processes in those with DPD. The aim of this research therefore was to test empirically three aspects of the cognitive-behavioural model of DPD in a sample of participants with DPD, and compare these results with those from an anxiety disorder group and a demographically matched control group, who had been screened for current psychiatric disorders. It was designed in two parts, with three tasks in total. Part one (Tasks 1 & 2) aimed to investigate the