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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.

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## 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

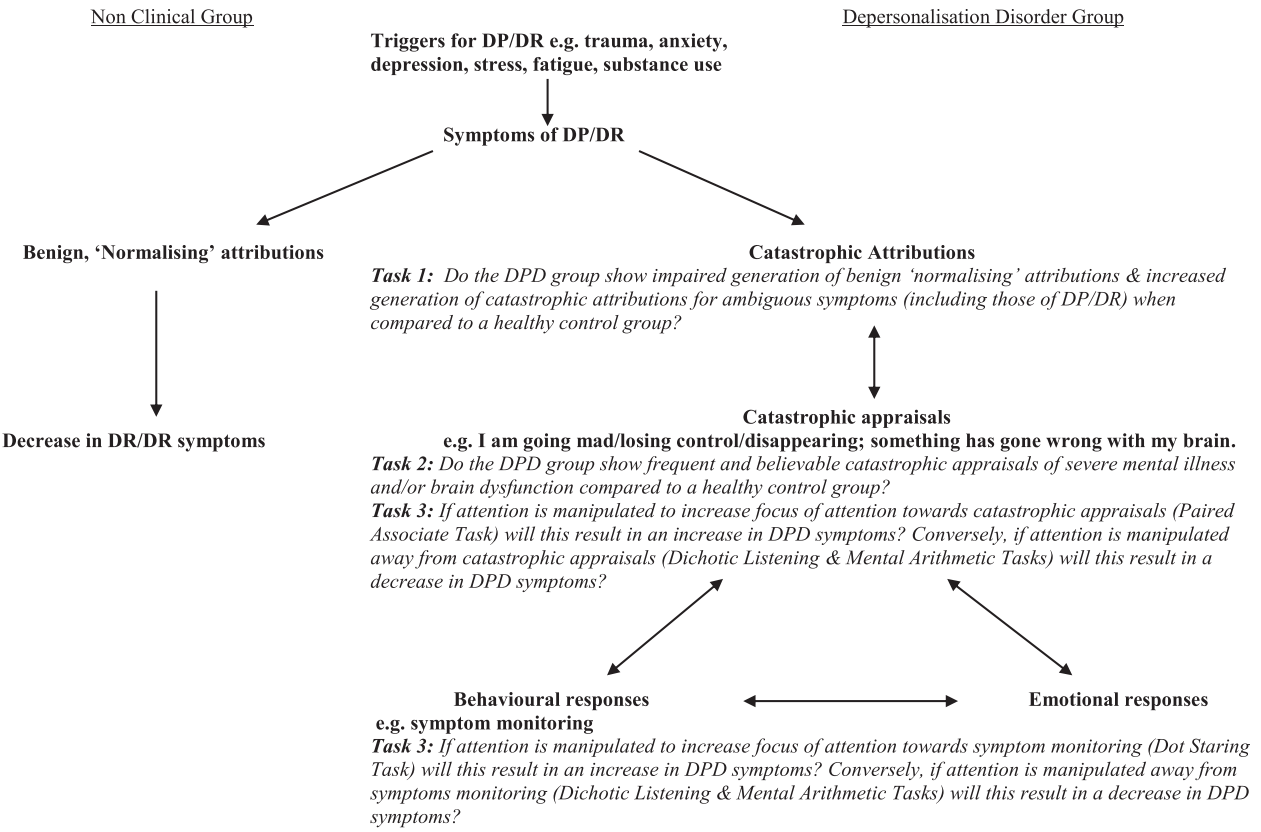


**Fig. 1.** Cognitive-behavioural model of depersonalisation disorder (based on Hunter et al., 2003), showing aspects of the model tested by the tasks in this study.

presence of catastrophic attributions and appraisals in patients with DPD. Part two of the study (Task 3) aimed to conduct a series of experimental manipulations to determine the impact on the perceived severity of DPD symptoms as a result of shifting attention to, and from, catastrophic cognitions and symptom monitoring.

These three tasks aimed to test specific hypotheses from the DPD model (see Fig. 1). Firstly, those with DPD will make less normalising attributions and more catastrophic attributions for a range of ambiguous symptoms, including those of DP/DR, than those in the control group (Task 1). Secondly, the DPD group would report significantly more frequent and convincing catastrophic appraisals (Task 2). Thirdly, experimental manipulations designed to focus attention onto DPD catastrophic cognitions (Paired Associates) or onto DPD symptoms (Dot Staring) should result in a corresponding increase in DPD symptoms. Conversely, if attention was diverted away from catastrophic cognitions and symptom monitoring by the use of cognitively demanding experimental tasks with externally generated stimuli (Dichotic Listening task) or internally generated stimuli (Mental Arithmetic task), this would be reflected in a decrease in perceived severity of DPD symptoms. Given that the CBT model of DPD supports strong associations between DPD and anxiety disorders, it was predicted that the two patient groups might appear very similar in their responding on these tasks, but with disorder specificity in the content of these.

## Method

### *Overview*

Depersonalisation disorder, anxiety disorder and psychiatrically healthy controls were recruited and screened using standardised diagnostic interviews and clinical questionnaires. Testing took place in a small plain laboratory room. Each participant started by completing measures of current emotional state and a DPD symptom checklist. The catastrophic attribution task (Task 1) was administered first to prevent contamination by other components of the investigation. The DPD symptom checklist was completed again after this task. The catastrophic appraisal questionnaire (Task 2) and the four experimental manipulations (Task 3) were presented in a randomised order, according to a Latin Square design. Filler tasks were administered between each experimental task in order to allow DPD symptoms to return to baseline levels. The DPD symptom checklist was completed immediately before and after each experimental task or manipulation. All participants were fully debriefed, assessed for any adverse reaction and paid a small sum of money for taking part.

### *Participants*

Three groups of participants were recruited for the tasks: a) participants with a diagnosis of Depersonalisation disorder (DPD); b) a mixed anxiety disorder group of participants with obsessive-compulsive disorder (OCD) or panic disorder; and c) psychiatrically healthy, demographically matched, controls.

DPD participants were recruited from a specialist DPD clinic (Depersonalisation Research Unit at the Institute of Psychiatry, London). All DPD participants had been given a prior, independent, diagnosis of DPD from a diagnostic, clinical interview based on the SCID structured interview schedule and conducted by an experienced psychiatrist in the DPD clinic. Referrals to this clinic are asked routinely to join a research register. The DPD group in this study responded to being sent information sheets describing the study and requesting their participation. A total of 70 letters were sent out with 32 responses (a response rate of 46%). Of these 32

potential participants, 2 did not meet inclusion criteria. Another 5 met criteria for inclusion, but following discussion about the nature of the study they did not take part as it was mutually agreed that testing might be too stressful for them at the present time. A total of 25 DPD participants took part.

Twenty-one participants were recruited into the Anxiety disorder group. Eighteen of these were recruited from the treatment waiting list of the Centre for Anxiety Disorders and Trauma at the Maudsley Hospital, London; thirteen of whom responded to being sent an information sheet and five who were given the information sheet by clinicians at the end of their assessment appointment. A further three participants were recruited from an OCD self help group when taking part in an unrelated research project.

Recruitment of the 22 participants in the Healthy Control group was via several methods. Eleven participants responded to adverts placed in local shops and sports centres; seven participants responded to information sheets distributed in a local shopping centre, and four participants were recruited from an existing subject pool in the psychology department of the Institute of Psychiatry, London.

The Structured Clinical Interview for DSM-IV (SCID: Spitzer, Gibbon-Miriam, & Williams, 1996) and Structured Clinical Interview for DSM-IV Dissociative Disorders Revised (SCID-D: Steinberg, 1994) were used to assess whether all participants met inclusion and exclusion criteria for the tasks. Participants from all groups were included if they were native English speakers between the ages of 18–75, who had a) no history of head injury that involved a significant loss of consciousness; b) no previous or current diagnosis of psychosis; c) no current diagnosis of acute stress disorder or post-traumatic stress disorder, since these include dissociative phenomena in their diagnostic criteria; or d) were not currently in receipt of cognitive-behavioural therapy which could influence cognitive Processes. Participants in the DPD group had to meet DSM-IV criteria (American Psychiatric Association, 1994) for current DPD, and those in the Anxiety disorder group had to meet DSM-IV criteria for current OCD or panic disorder, but not current DPD. Participants in the Control group were excluded if they met DSM-IV criteria for current or past histories of DPD, panic disorder, alcohol and/or substance abuse, as well as current agoraphobia without panic, social phobia, OCD, generalised anxiety disorder, health anxiety or major depressive episode.

## Measures

### Clinical measures

All participants completed the Beck Depression Inventory (BDI: Beck, Ward, Mendelson, Mock, & Erbaugh, 1961); Beck Anxiety Inventory (BAI: Beck, Epstein, Brown, & Steer, 1988); Dissociative Experiences Scale (DES: Bernstein & Putnam, 1986) and the Trait and State versions of the Cambridge Depersonalisation Scale (CDS-T & CDS-S: Sierra & Berrios, 2000). The BDI and BAI are two widely used self-report measures. Each consists of 21 items that assess cognitive, affective, behavioural and physiological symptoms of depression or anxiety over the previous week, with the total score representing the severity of symptomatology. The DES is the most widely used measure of current, adult dissociation. Participants rate the percentage of the time they experience 28 different dissociative phenomena in their everyday life, when not under the influence of alcohol or drugs. The CDS is the most widely used measure of depersonalisation/derealisation. The trait version (CDS-T) is a 29 item scale measuring the severity of trait DP/DR symptoms over the past 6 months. Each item is scored in terms of frequency and duration. The total score is out of 290. A cut-off score of 70 has been found to be indicative of DPD. The state version of the CDS (CDS-S) was designed specifically for intervention studies. It comprises 22 items from the original trait version, which are scored on a visual analogue scale and converted into a percentage. A mean overall score (0–100) is used. The psychometric properties of the CDS have been reported by Sierra and Berrios (2000). The scale showed high internal consistency and good reliability: Cronbach alpha and split-half reliability were 0.89 and 0.92, respectively.

However, given that ratings of current DPD symptomatology were conducted regularly during the testing procedure, the CDS-S with 22 items was deemed too long to use in our study. A shortened 12 item DPD checklist was derived using the most characteristic, and common, symptoms from the Cambridge Depersonalisation Scale. These items were cross-referenced with other diagnostic measures of DP/DR (i.e. SCID-D (Steinberg, 1994); DSM-IV (American Psychiatric Association, 1994); ICD-10 (World Health Organisation, 1992); and the Depersonalisation/derealisation items in the DES (Bernstein & Putnam, 1986)). For each of these 12 items, participants were asked to rate their current experience of each symptom as a percentage. The mean of the percentage rating of all 12 items was used in further analyses. A copy of the 12 item DPD symptom checklist can be obtained from the authors.

### Experimental measures/tasks

*Task 1: catastrophic attributions.* This task used an extended and modified version of the task used by MacLeod et al. (1998). Participants were presented with three symptoms in each of four categories: DPD (e.g. feeling cut off from things around you); physical symptoms of anxiety (e.g. heart pounding); cognitive symptoms deemed relevant to both DPD and Anxiety (e.g. confused); and neutral symptoms (e.g. hungry). Each symptom was presented individually in a randomised order, and for each symptom participants were asked to write down as many reasons as to why this might happen to them within a 1 min period. Responses were categorised into one of four types of attribution: 1) normalising (i.e. due to common environmental factors; common/mild mood states; minor impairments to cognitive functioning; or common physical illness e.g. colds or flu); 2) psychological (i.e. due to severe emotional states; mental health problems, including attributions directly related to DPD; psychologically distressing environmental situations; or moderate to severe cognitive impairments); 3) somatic (i.e. adverse but temporary physical conditions such as pain or allergy; or moderate to severe illness or injury); or 4) unclassifiable i.e. where there was insufficient information to determine classification the response was idiosyncratic (e.g. 'that's just me'). An independent rater, blind to the initial classification and to the group membership, categorised a random selection of 10% of responses and the level of agreement was high (kappa $> 0.8$).

*Task 2: catastrophic appraisals.* The Catastrophic Appraisals Questionnaire was designed specifically for this task and was derived from combining two questionnaires. The first of these had been used clinically to measure change in cognitions in a sample of 12 DPD patients who were having CBT with author EH, and the second was a questionnaire derived from the Agoraphobic Cognitions Questionnaire (Chambless, Caputo, Bright, & Gallagher, 1984) that had been modified by author PS to include further symptoms and belief ratings for panic. Participants were asked to indicate the frequency (on a 5-point Likert scale: never, rarely, half the time, usually and always) and percentage conviction rating (0% = not at all convinced to 100% = completely convinced) of 17 different catastrophic appraisals that they might have at the time of experiencing unusual or strange sensations, or when they worried about their health. Catastrophic appraisals fell into three categories: mental illness (5 questions e.g. I am going insane); brain

dysfunction (3 questions e.g. something has gone wrong with my brain); and physical illness (9 questions e.g. I have cancer). This measure showed acceptable internal reliability (Cronbach's alpha = 0.70).

*Task 3: experimental manipulations of attention to, and from, catastrophic appraisals and DPD symptoms*

*Dot Staring task (attention to DPD symptoms).* This manipulation aimed to experimentally increase attention to DPD symptoms in a similar way to that of previous empirical studies of DPD induction (Leonard et al., 1999; Miller et al., 1994). Participants were asked to focus their attention on a black dot (5 cm in diameter) placed on a plain white wall, approximately 2 m away, for 3 min duration.

*Paired Associates task (attention to DPD catastrophic appraisals).* This manipulation was adapted from a paradigm used by Clark et al. (1988) with panic disorder to activate catastrophic appraisals. In this exercise a list of DPD symptoms was paired with typical DPD-related catastrophic appraisals (e.g. spaced out = insanity, unreality = madness, detached = brain disease). Participants were asked to read aloud the list slowly for 1 min, and to pause after each pair to think about the meaning of the words.

*Mental Arithmetic task (attention from DPD symptoms and appraisals by internally generated task).* In order to instigate an internally generated task that would focus attention away from cognitive processes and symptom monitoring, participants were asked to perform mental arithmetic calculations aloud (reverse serial 7's starting from 500) as quickly as possible for 1-min duration.

*Dichotic Listening task (attention from DPD symptoms and appraisals by externally generated task).* A Dichotic Listening task was employed as an externally generated task to focus attention. The task was adapted from a study of people with chronic pain (Rode, Salkovskis, & Jack, 2001) but with neutral, rather than threat-related stimuli. Participants were asked to listen to a series of neutral sentences. Ten words that formed a sentence and ten distractor words (different forms of vehicle) were heard simultaneously through headphones. The words in the sentence were presented in alternate ears and participants had to try to 'shadow' the sentence by saying the words aloud. Participants were instructed that the words in the sentences always started in the right ear with a person's forename, in order to orientate them to the channel to be shadowed. After this name, the words in the sentence would be presented into alternate ears e.g.

| | | | | | | |
|---|---|---|---|---|---|---|
| Channel 1: | **Carole** | aeroplane | **swimming** | motorcycle | **the** | truck |
| Channel 2: | Car | **goes** | helicopter | **at** | gondola | **local** |
| Channel 1: | **pool** | bicycle | **Monday** | convertible | | |
| Channel 2: | cart | **each** | lorry | **morning** | | |

Sentences and distractor words were spoken by two different female voices at a rate which had been found in a pilot study to result in 20% of the distractor words being reported (at 2 s intervals). There was a gap of 5 s between each sentence. All participants were given 5 practice sentences before the task started. The task lasted for 3 min duration. Stimuli for this task were generated on a Power Macintosh 7500/100 running SoundEdit™ 316 plus Dexk II programmes; Headphones were of the GEN*EXXA* manufacture.

*Neutral 'filler' tasks*

Four neutral tasks were employed between the experimental manipulations as "fillers". These tasks were included to allow time for any temporary increase or decrease in symptom severity caused by the experimental manipulations to return to baseline levels. These tasks involved either searching for specific symbols among a series of symbols or copying symbols/numbers from a template. Each 'filler' task was of 3 min duration.

**Table 1**
Demographic and clinical characteristics of participants.

| | DPD group ($n = 25$) | | Anxiety group ($n = 21$) | | Healthy control group ($n = 22$) | |
|---|---|---|---|---|---|---|
| | *M* | S.D. | *M* | S.D. | *M* | S.D. |
| *Characteristic* | | | | | | |
| Age (years) | 35.1 | 9.5 | 37.5 | 11.3 | 38.5 | 14.0 |
| Education (years) | 14.2 | 2.4 | 13.2 | 2.5 | 14.7 | 2.4 |
| BDI total score | 15.6[a] | 7.8 | 19.7[a] | 14.0 | 2.4[b] | 2.2 |
| BAI total score | 14.7[a] | 9.1 | 21.8[b] | 10.7 | 3.1[c] | 2.3 |
| DES total score | 27.8[a] | 15.3 | 18.3[a] | 17.0 | 5.7[b] | 4.3 |
| CDS-trait total score | 124.4[a] | 48.2 | 46.8[b] | 34.1 | 9.1[c] | 6.7 |
| CDS – state mean % score | 31.2[a] | 18.0 | 9.7[b] | 9.9 | 1.4[c] | 2.1 |
| | *n* | | *n* | | *n* | |
| Gender (female/male) | 13/12 | | 13/8 | | 13/9 | |
| *Ethnicity* | | | | | | |
| Black British | 0 | | 3 | | 2 | |
| British Asian | 1 | | 1 | | 0 | |
| Caucasian | 24 | | 16 | | 20 | |
| Mixed race | 0 | | 1 | | 0 | |
| Living with partner (yes/no) | 9/16 | | 9/12 | | 6/16 | |
| Currently employed (yes/no) | 19/6 | | 15/7 | | 18/4 | |

*Note.* BDI = Beck Depression Inventory; BAI = Beck Anxiety Inventory; DES = Dissociative Experiences Scale; CDS = Cambridge Depersonalisation Scale.
[a,b,c]Means that do not share the same superscript horizontally are significantly different ($p < .01$).

*Data analysis*

Demographic and clinical characteristics of the three participant groups were compared using Chi-squared tests and one-way ANOVAs, with planned Bonferroni comparisons. In Task 1, the distribution of data was analysed to see if they conformed to expectations of normality according to the Kolmogorov–Smirnov test. Any variable that was not normally distributed was analysed using non-parametric tests. The analyses were conducted in two stages. The first stage compared the type of the first attribution (i.e. normalising, psychological, somatic or unclassifiable) given for all 12 symptoms in each of the three participant groups. The second stage of analyses investigated whether there was a between-groups effect for the specificity of the different types of symptoms (i.e. DPD, cognitive, anxiety, neutral) on the first attribution given. Simple main effects ANOVAs were conducted with planned tests (LSD) to compare group means.

In Task 2, the frequency and percentage conviction rating of catastrophic appraisals was analysed using a $3 \times 3$ repeated measures ANOVA, with type of catastrophic cognition (mental illness, brain dysfunction or physical illness) as the dependent variable and group as the between subjects factor (DPD, anxiety, control). If Mauchly's Test of Sphericity was significant, the Greenhouse–Geisser correction was applied to the degrees of freedom to assess the observed F ratio. If there was a significant interaction a simple main effects analysis was conducted with planned tests (Least Significant Difference: LSD) to compare group means. Secondary analyses were conducted, excluding the panic participants ($n = 5$) to determine if the difference in frequency and conviction ratings in physical illness catastrophic appraisals in the

Anxiety disorder group was due to the influence of appraisals relating to the physical symptoms of panic, rather than a general effect of anxiety.

In Task 3, the difference between pre-task and post-task scores on the DPD symptom severity checklist was calculated as a difference score. One-way ANOVAs were conducted for the four experimental tasks with Tukey LSD planned tests for multiple comparisons.

## Results

### *Participants*

As shown in Table 1, each patient group was demographically comparable to the healthy comparison group in terms of age, gender, ethnicity, living with a partner, years of education, or if currently employed (all significance levels $p > .1$).

In terms of participants' scores on the clinical measures, one-way ANOVAs yielded significant differences among the three groups on the BDI, $F\ (2,\ 65) = 21.2$, $p < .01$; BAI, $F\ (2,\ 65) = 28.6$, $p < .01$; DES, $F(2, 65) = 16.0$, $p < .01$; CDS-T, $F(2, 65) = 66.4$, $p < .001$ and CDS-S, $F\ (2,\ 65) = 37.0$, $p < .01$. Planned comparisons using Bonferroni tests showed that on the BDI and the DES, the DPD and Anxiety groups were significantly different from the Healthy Control group, but not from each other. On the BAI and CDS, all three groups were significantly different from each other, with the Anxiety group having the highest scores on anxiety and the DPD group having the highest score for Depersonalisation.

### *Task 1: catastrophic attributions*

#### *Stage 1: all symptoms*

Table 2 shows the means and standard deviation scores in each of the three participant groups for the number of first attributions in each attribution type (i.e. normalising, psychological, somatic and unclassifiable) from the 12 symptoms presented. In the DPD group, there were a small number of attributions made that were specific for DPD (e.g. 'my depersonalisation'). These were for DPD symptoms (mean = 1.00, S.D. = 1.15) and cognitive symptoms (mean = 0.28, S.D. = 0.68). Since these attributions were for mental illness, they were incorporated into the broader psychological attribution category.

A simple main effects ANOVA was conducted with planned post-hoc tests (LSD). The results from these analyses show that the healthy controls assigned significantly more normalising first attributions to the symptoms than did both the DPD and Anxiety Disorder groups, $F\ (2,\ 65) = 5.95$, $p < .01$, whereas the DPD and Anxiety Disorder group assigned significantly more psychological first attributions to the symptoms presented in the task than did the Healthy Control group, $F(2, 65) = 5.11$, $p < .01$. The three groups did not differ significantly in the number of somatic first attributions, $F\ (2,\ 65) = 0.76$, $p > .05$, nor for unclassifiable responses $F\ (2,\ 65) = 0.48$, $p < .05$. However, analysis of the distribution of the somatic first attributions showed this did not conform to expectations of normality according to the Kolmogorov–Smirnov test. Transformations of this variable did not normalise these data, so a non-parametric analysis was also conducted (Kruskal–Wallis test). The results from this analyses again showed a non-significant difference between the groups ($\chi^2$(d.f. = 2) = 0.78, $p > .05$).

#### *Stage 2: specificity of symptoms*

A second, more detailed, analysis examined whether there was a between-group effect for the *specificity of the symptoms* presented in the task, i.e. to determine whether the number of first attributions for the different types of symptoms presented in the task (i.e. DPD, Cognitive, Anxiety, Neutral) differed in the three participant groups.

**Table 2**
Task 1: catastrophic attributions.

| | DPD group ($n = 25$) | | Anxiety group ($n = 21$) | | Healthy control group ($n = 22$) | |
|---|---|---|---|---|---|---|
| | *M* | S.D. | *M* | S.D. | *M* | S.D. |
| **Attribution type** | | | | | | |
| *Mean number of first attributions (0–12)* | | | | | | |
| Normalising | 6.6[a] | 2.4 | 6.5[a] | 2.5 | 8.5[b] | 1.4 |
| Psychological | 3.8[a] | 2.3 | 3.5[a] | 2.5 | 1.9[b] | 2.0 |
| Somatic | 1.3 | 1.5 | 1.5 | 1.4 | 1.0 | 1.3 |
| Unclassifiable | 0.4 | 0.6 | 0.5 | 0.7 | 0.6 | 1.1 |
| **Symptom type** | | | | | | |
| *Mean number of first attributions (0–3)* | | | | | | |
| DPD | | | | | | |
| Psychological | 1.56[a] | 1.16 | 0.91[b] | 1.0 | 0.68[b] | 0.95 |
| Somatic | 0.28 | 0.61 | 0.33 | 0.80 | 0.32 | 0.57 |
| Normalising | 1.12 | 1.06 | 1.48 | 0.93 | 1.77 | 1.02 |
| Cognitive | | | | | | |
| Psychological | 0.76[a] | 0.83 | 1.14[b] | 0.96 | 0.36[a] | 0.58 |
| Somatic | 0.48 | 0.65 | 0.29 | 0.46 | 0.18 | 0.40 |
| Normalising | 1.72[a] | 0.89 | 1.52[a] | 0.98 | 2.36[b] | 0.58 |
| Anxiety | | | | | | |
| Psychological | 1.36 | 1.08 | 1.24 | 1.14 | 0.73 | 0.77 |
| Somatic | 0.20 | 0.50 | 0.48 | 0.68 | 0.36 | 0.49 |
| Normalising | 1.44 | 1.08 | 1.29 | 1.06 | 1.82 | 0.85 |
| Neutral | | | | | | |
| Psychological | 0.12 | 0.33 | 0.19 | 0.40 | 0.14 | 0.35 |
| Somatic | 0.32 | 0.57 | 0.38 | 0.50 | 0.14 | 0.35 |
| Normalising | 2.28 | 0.74 | 2.24 | 0.77 | 2.50 | 0.51 |

*Note*. [a,b]Means that do not share the same superscript horizontally are significantly different ($p < .05$).

The number of first attributions given in each symptom category by each group, was analysed using simple main effects ANOVAs with planned comparison tests (LSD). Table 2 shows the means and standard deviations for the number of first attributions in each symptom type by group. There were significant between-group differences on the attributions for DPD symptoms, $F(2, 65) = 4.54$, $p < .05$. Tukey LSD tests showed that the DPD group gave significantly more psychological attributions for DPD symptoms presented in this task when compared to the Anxiety and healthy Control groups, who did not differ from each other. There were also between-group differences on attributions for cognitive symptoms, with significant differences in normalising attributions, $F\ (2,\ 65) = 6.05$, $p < .005$, and psychological attributions, $F\ (2,\ 65) = 5.0$, $p < .01$. Tukey LSD tests indicated that the Control group generated significantly more normalising first attributions for the cognitive symptoms than either the DPD or the Anxiety Groups. Moreover, the Anxiety group generated significantly more psychological first attributions for the cognitive symptoms relative to the DPD or the Control groups.

### *Task 2: catastrophic appraisals*

#### *Frequency of catastrophic appraisals*

There was a significant main effect for the frequency of each type of catastrophic appraisal (mental illness; brain dysfunction; physical illness), $F\ (2,\ 130) = 15.2$, $p < .001$, and of Group, $F\ (2,\ 65) = 17.6$, $p < .001$. These effects were modified by a significant interaction between the frequency of each type of Catastrophic Appraisal and Group, $F\ (4,\ 130) = 14.0$, $p < .001$. A simple main effects analysis was therefore conducted with planned tests (LSD) to compare group means. Table 3 shows the means and standard deviation scores in each group for the frequency of each type of catastrophic appraisal.

The results presented in Table 3 show that the DPD and Anxiety disorder groups reported significantly more frequent

catastrophic appraisals about mental illness and brain dysfunction than the Healthy Control group, respectively $F(2, 65) = 18.6$, $p < .001$, and $F(2, 65) = 13.4$, $p < .001$. In terms of physical illness, the Anxiety Disorder group reported significantly more of these catastrophic appraisals than the DPD and Healthy Control groups ($F(2, 65) = 17.4$, $p < .001$), who did not differ significantly from each other.

*Conviction in catastrophic appraisals*

There was a significant main effect of the percentage conviction rating of each type of catastrophic appraisal (mental illness; brain dysfunction; physical illness), $F(2, 130) = 12.4$, $p < .001$, and of Group, $F(2, 65) = 16.1$, $p < .001$. These effects were modified by a significant interaction between the percentage conviction rating of each type of Catastrophic Appraisal and Group, $F(4, 130) = 14.0$, $p < .001$. A simple main effects analysis was conducted with planned tests (LSD) to compare group means. Table 3 shows the means and standard deviation scores in each group for the percentage conviction rating of each type of catastrophic appraisal.

The results presented in Table 3 show that the DPD and Anxiety disorder groups reported significantly more conviction in their catastrophic appraisals about mental illness and brain dysfunction than the Healthy Control group, respectively $F(2, 65) = 18.9$, $p < .001$, and $F(2, 65) = 12.9$, $p < .001$. In terms of conviction ratings of catastrophic appraisals of physical illness, the Anxiety Disorder group reported significantly higher conviction in these than the DPD and Healthy Control groups ($F(2, 65) = 14.0$, $p < .001$), who did not differ significantly from each other.

A secondary analysis, excluding the panic participants ($n = 5$) and including only OCD participants, showed the same pattern of results, with significantly more frequent and convincing physical illness catastrophic appraisals, than the DPD and Healthy Control groups.

*Task 3: experimental manipulations of attention to, and from, catastrophic appraisals and DPD symptoms*

This part of the study involved four experimental manipulations (Dot Staring, Paired Associates, Mental Arithmetic & Dichotic Listening). These were analysed using separate simple main effects ANOVAs, as each manipulation had a different hypothesised method of changing the participant's perceived severity of DPD symptoms. Tukey LSD planned tests for multiple comparisons were conducted. Participant's pre and post scores on the severity of symptoms on the DPD checklist were converted into a difference by subtracting pre from post scores. Table 4 shows the means and standard deviation scores for each group on mean pre- and post-task DPD symptom severity scores and the discrepancy between these scores for each experimental task.

Simple main effects ANOVAs showed that the three groups were not significantly different on the Dot Staring task ($p = .07$), but there was a significant group difference on the Paired Associate task, $F(2, 65) = 4.0$, $p < .05$. Post-hoc tests showed that the DPD group showed a significant increase in perceived DPD symptom severity compared to the healthy control group, but not the anxiety group. The anxiety group did not differ significantly from the healthy control group on this task.

In the Mental Arithmetic task and the Dichotic Listening task the analysis showed a significant main effect (respectively, $F(2, 65) = 11.5$, $p < .001$ and $F(2, 65) = 14.0$, $p < .001$). Planned post-hoc tests showed that on the Mental Arithmetic task the DPD group showed a significant decrease in DPD symptoms compared to the anxiety and healthy control groups, who were not significantly different from one another and who both showed a slight increase in reported DPD symptom severity. On the Dichotic Listening task, the DPD group again showed a significant decrease in the reported severity of DPD symptoms compared to the anxiety and healthy control groups. The control groups both showed an increase in reported DPD symptom severity and were significantly different from each other.

## Discussion

This series of tasks aimed at systematically testing three parts of the CBT model of DPD (Hunter et al., 2003) deemed to be important in the development and maintenance of symptoms; namely the role of attributions, appraisals and attention to symptoms (see Fig. 1). Although there have been anecdotal reports in the existing literature for catastrophic cognitions in DPD, this is the first study that has examined this phenomenon systematically. These three processes were tested in a sample of people with a diagnosis of

**Table 3**
Task 2: catastrophic appraisals: frequency and percentage conviction rating of type of catastrophic appraisal by group.

| | DPD group ($n = 25$) | | Anxiety group ($n = 21$) | | Healthy control group ($n = 22$) | |
|---|---|---|---|---|---|---|
| | *M* | S.D. | *M* | S.D. | *M* | S.D. |
| *Frequency of type of catastrophic appraisal (0–4)* | | | | | | |
| Mental illness | 1.7[a] | 1.3 | 1.6[a] | 0.9 | 0.2[b] | 0.3 |
| Brain dysfunction | 1.5[a] | 0.9 | 1.0[a] | 1.0 | 0.2[b] | 0.2 |
| Physical illness | 0.4[a] | 0.4 | 1.2[b] | 0.8 | 0.3[a] | 0.3 |
| *Percentage conviction rating of type of catastrophic appraisal* | | | | | | |
| Mental illness | 48.6[a] | 33 | 48.0[a] | 28 | 6.0[b] | 14 |
| Brain dysfunction | 41.7[a] | 27 | 31.2[a] | 32 | 5.1[b] | 11 |
| Physical illness | 15.8[a] | 16 | 40.2[b] | 29 | 9.6[a] | 13 |

*Note*. [a,b]Means that do not share the same superscript horizontally are significantly different ($p < .01$). Those that do are not significantly different ($p > 0.05$).

**Table 4**
Comparison of pre- and post-task scores for each experimental manipulation by group.

| Task type | DPD group ($n = 25$) | | Anxiety group ($n = 21$) | | Healthy control group ($n = 22$) | |
|---|---|---|---|---|---|---|
| | *M* | S.D. | *M* | S.D. | *M* | S.D. |
| Paired Associates | | | | | | |
| Pre-task scores | 36.4 | 20.5 | 14.8 | 21.8 | 0.7 | 1.4 |
| Post-task scores | 43.4 | 20.8 | 18.6 | 24.8 | 1.6 | 2.2 |
| Discrepancy in % ratings in pre–post scores | 7.0[a] | | 3.7[ab] | | 0.9[b] | |
| Dot Staring | | | | | | |
| Pre-task scores | 40.0 | 21.3 | 15.2 | 22.6 | 0.8 | 1.1 |
| Post-task scores | 48.9 | 20.9 | 31.6 | 29.6 | 7.9 | 11.4 |
| Discrepancy in % ratings in pre–post scores | 8.9 | | 16.4 | | 7.0 | |
| Mental Arithmetic | | | | | | |
| Pre-task scores | 38.4 | 19.4 | 15.4 | 23.7 | 0.7 | 1.3 |
| Post-task scores | 29.5 | 21.0 | 19.2 | 29.7 | 1.3 | 1.7 |
| Discrepancy in % ratings in pre–post scores | −8.9[a] | | 3.8[b] | | 0.6[b] | |
| Dichotic Listening | | | | | | |
| Pre-task scores | 39.4 | 20.2 | 16.3 | 22.9 | 0.4 | 0.8 |
| Post-task scores | 32.2 | 19.1 | 26.0 | 31.0 | 1.9 | 2.3 |
| Discrepancy in % ratings in pre–post scores | −7.2[a] | | 9.7[b] | | 1.5[c] | |

*Note*. [a,b,c]Means that do not share the same superscript horizontally are significantly different ($p < .05$).

DPD, compared to a healthy control group. An Anxiety disorder control group was also included for comparison, given the similarities between DPD and Anxiety proposed by this model. Task 1 examined whether those with DPD were more likely to generate a catastrophic, rather than normalising, attribution for ambiguous symptoms, compared to the healthy control group. Task 2 looked at the content, frequency and believability of catastrophic appraisals in DPD. Task 3 used four experimental manipulations to direct attention *to* DPD symptoms and catastrophic cognitions (respectively, Dot Staring and Paired Associates tasks) and *from* DPD symptoms and catastrophic cognitions by using cognitively demanding tasks with either internally generated stimuli (Mental Arithmetic task) or externally generated stimuli (Dichotic Listening task). In summary, this study found empirical support for all three parts of the CBT model under investigation when the DPD group were compared to the healthy controls, and many similarities between the DPD and Anxiety groups although with some distinct differences between the two patient groups.

The first two tasks in this study indicated that people with a diagnosis of DPD show significant differences in their attributions and appraisals of symptoms when compared to healthy controls. In the first task, participants were presented with a range of ambiguous symptoms and asked to generate reasons as to what might be the possible cause of these. The control group were significantly more likely to give a benign reason for ambiguous symptoms as their first response and in doing so normalise these experiences, when compared to those with DPD. By contrast, those with DPD were more likely to give a catastrophic psychological reason as their first response, believing the symptom was likely to be caused by significant mental health problems. The two groups did not differ in terms of somatic attributions. Moreover, when the results were examined in terms of their responses to specific types of symptoms presented, those in the DPD group were significantly more likely to give a serious psychological attribution to DPD symptoms than the healthy controls, and were less likely to find a normalising reason for cognitive symptoms. These findings from Task 1 fit with the predictions from the CBT model of DPD, which suggest that those with DPD are impaired in their ability to generate benign, normalising attributions for symptoms and instead generate psychological attributions that are catastrophic in nature. This appears to be specifically for DP/DR and cognitive symptoms, and does not apply to anxiety nor neutral symptoms. This finding might offer some explanation as to why although epidemiological surveys show that transient symptoms of DP/DR are very common in the general population, that only a minority of people go onto develop the clinical disorder of DPD. It appears that in those who develop DPD there is a tendency to interpret these experiences in a psychologically catastrophic way which is only likely to exacerbate the original symptoms.

The presence of catastrophic appraisals is the lynchpin of cognitive models, including the cognitive model of DPD proposed by Hunter et al. (2003). If DPD patients were found to have no more frequent catastrophic appraisals than controls, nor a greater belief in these, then there would be little point in further investigation. However, the results from the second task showed that the hypothesis that people with a diagnosis of DPD would report significantly more frequent and convincing, catastrophic appraisals when compared to a healthy control group was confirmed in terms of appraisals of mental illness and brain dysfunction, but not physical illness. Those with a diagnosis of DPD reported having disturbing thoughts such as 'I'm losing control of my mind' or 'something has gone wrong with my brain' nearly half the time when they experienced unusual or strange sensations or when they worried about their health, and these catastrophic thoughts were between 40 and 50% convincing at the time. In comparison, healthy controls reported having catastrophic cognitions ranging from never to rarely, and if they did experience these types of thoughts their conviction rates did not exceed 10%. The importance of these findings from tasks 1 and 2 to the cognitive-behavioural model of DPD is that these types of catastrophic appraisals and attributions are likely to trigger off a vicious cycle of negative emotions (such as anxiety), as well as behavioural and cognitive strategies (such as increased symptom monitoring), which only serve to exacerbate the original symptoms. In this way a maintaining cycle can be instigated, so that transient symptoms of DP/DR can become chronic DPD (see Fig. 1).

In task three, experimental manipulations directed participant's attention *towards* catastrophic attributions/appraisals associated with DPD (Paired Associate Task), or DPD symptoms (Dot Staring Task), or *away from* both of these processes with highly demanding cognitive tasks requiring internally generated stimuli (Mental Arithmetic task) or externally generated stimuli (Dichotic Listening task). The results of three out of four of these manipulations supported the predictions that would be made from the CBT model of DPD. However, in the Dot Staring task there were no significant differences between the DPD, Anxiety disorder and healthy control groups, with all three groups showing an increase in DPD symptom severity following the task. It may be that this task failed to differentiate between the groups because those with DPD already had a moderately high baseline level of symptom monitoring that did not increase significantly with this task, whereas in the two control groups the task was relatively effective in inducing DPD symptoms. In the Paired Associate task, the DPD group reported a significant increase in their symptom severity compared to controls after reading aloud a list of DPD symptoms paired with typical DPD catastrophic appraisals for just 1 min. The findings from these two tasks suggests that although the processes of both catastrophic cognitions and symptom focussed attention are likely to play a part in increasing DPD symptoms, the role of cognitions may be more important.

The two tasks, Mental Arithmetic and Dichotic Listening, which aimed to direct attention away from negative cognitions and symptom monitoring had very interesting results. Whereas these cognitively demanding, and stressful, tasks increased reported DPD symptoms in the Anxiety group and to a lesser degree in the Control group, they had the opposite effect in the DPD group. After both the internally focussed (i.e. Mental Arithmetic) or externally focussed (i.e. Dichotic Listening) tasks, the DPD participants reported DPD symptom severity ratings as reduced. These results appear to confirm the predictions for Task 3 from the CBT model, in that when attention was manipulated away from catastrophic cognitions and symptom monitoring in the DPD group, these processes in the vicious cycle part of the model in Fig. 1 were inhibited, and participants reported a perceived reduction in DPD symptoms.

The study is interesting also in terms of the predicted similarities between DPD and Anxiety disorders as described by Hunter et al. (2003). One of the hypotheses in the study was that the two patient groups would be very similar in their responding in terms of catastrophic cognitions. Results of tasks 1 and 2 are largely consistent with this view. In the Attribution task, most of the significant differences between the DPD group and the healthy controls were mirrored by the Anxiety group. Both patient groups were less likely to normalise a range of ambiguous symptoms and give more psychological explanations for these than those in the healthy control group. However, the DPD group, as predicted by the model, were more likely to attribute a catastrophic psychological reason for DPD symptoms. The Anxiety group, on the other hand, reported more psychological attributions for cognitive symptoms than the DPD group. In Task 2, the groups were again very similar in terms of the frequency and conviction in appraisals of mental illness and

brain dysfunction, with the Anxiety group showing significantly more physical illness appraisals than the DPD group, which would be expected from a group of patients with panic and/or OCD. However, the major significant difference between the two patient groups was in their response to the Dichotic Listening and Mental Arithmetic tasks where the Anxiety group showed an increase in reported DPD symptoms following these, whereas the DPD group reported a decrease. One interpretation of these results, as predicted by the CBT model of DPD, is that the DPD group could not focus on symptom monitoring nor catastrophic attributions/appraisals during these tasks and so reported a decrease in symptoms. On the other hand, the Anxiety group found these tasks stressful and this increased perceived DPD symptoms. The results from the manipulation tasks suggest that the two disorders might be differentiated by their responses to cognitively demanding tasks.

The study has several limitations. Firstly, the sample size in each group was relatively small. There were also potential sampling biases, in that both the DPD and the Anxiety Disorder participants were recruited from tertiary services, where there is likely to be a greater severity of symptomatology. Participants in the Anxiety disorders group were comprised of patients with clinically significant levels of anxiety but with primary diagnoses of Panic disorder or OCD. Ideally, the anxiety disorders group would have been comprised of a sample with a single diagnosis but given difficulties in recruitment, a pragmatic sample was used. The tasks were potentially also open to demand effects, given that the patient groups were aware of the basis for their selection for the study. Moreover, the primary rater for attribution type was not blind to the group status of participants and this may have introduced a bias, although the second rater was blind to group status. The same participants were used in all the tasks; using different samples of participants for each of the three tasks would have strengthened the research design. The Dot Staring task requires effort to produce depersonalisation, yet people with the disorder experience the symptoms as involuntary. It may be that the phenomenon in the laboratory works through a different mechanism, even though this task increased DPD symptom severity in all three groups and has previously been demonstrated as the most effective experimental method to produce the experience of DPD. Given the number of tasks in the study, the testing time for participants was quite lengthy and this meant restricting data collection to only that which was essential. As a result, the study did not collect information on other data which may have been interesting, such as changes in mood during the testing procedure.

However, despite these limitations, the results from this series of tasks provide empirical support for the CBT model of DPD. This has important implications for CBT approaches to the treatment of DPD as carried out in a small scale open trial by Hunter, Baker, Phillips, Sierra, and David (2005). Tasks 1 and 2 showed that that those with DPD find it hard to generate benign explanations for a range of symptoms, and there are high frequencies and conviction ratings in catastrophic appraisals triggered by unusual symptoms or worry in those with DPD. From our clinical work with DPD, clients frequently reported that psycho-education about the high prevalence of transient DP and DR, and hearing that other people can recover from their symptoms, was one of the most helpful aspects of contact with professionals, as it helped to normalise their experiences and offered hope. Similarly, giving information about the differences between DPD and schizophrenia, as well as the lack of empirical research evidence for serious brain abnormalities in DPD was reported as reassuring by clients and helped to counter existing, catastrophic appraisals. The findings from this study show why these interventions would be helpful to clients. Including a shared, individualised, CBT formulation derived from the DPD model in therapy, could help clients see how their catastrophic appraisal of the DP/DR symptom, rather than the symptom per se, is likely to have led to increased distress. Moreover, helping clients with DPD to find alternative, non-pathological, explanations for their symptoms and testing these through a series of behavioural experiments would clearly be of benefit. Regular re-rating of conviction in the catastrophic cognitions through the course of therapy would assess the effectiveness of these interventions.

The results from Task 3 showed that activating catastrophic cognitions associated with DPD, increased symptom severity ratings. Conversely, when participants were engaged in tasks which broke these vicious cycles they noticed an immediate reduction in symptoms. It follows from these findings that helping the client understand how the cycle of rumination and self focussed attention serves to exacerbate their problems and working collaboratively to find cognitively challenging but interesting strategies to break this vicious cycle would be beneficial. The open study of CBT in patients by Hunter et al. (2005), employed both Attention Training (Wells, White, & Carter, 1997) and Task Concentration Training (Bogels, Mullens, & De Jong, 1997) to help clients enhance their ability to shift their attention away from internally focussed symptom monitoring to an external focus.

In conclusion, the findings from the study presented here are valuable in furthering our understanding of the psychological processes in DPD, and particularly the significant role that attributions, appraisals and attention to symptoms play in the development and maintenance of this disorder. The CBT model of DPD and the interventions suggested by this offer hope that effective treatments for this distressing and disabling condition will be advanced.

## References


American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.) Washington, D.C.

Bebbington, P., Hurry, J., Tennant, C., Sturt, E., & Wing, J. K. (1981). Epidemiology of mental disorders in Camberwell. *Psychological Medicine, 11*, 561–579.

Bebbington, P., Marsden, L., & Brewin, C. R. (1997). The need for psychiatric treatment in the general population: the Camberwell needs for care survey. *Psychological Medicine, 27*, 821–834.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: psychometric properties. *Journal of Consulting and Clinical Psychology, 56*, 893–897.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4*, 561–571.

Bernstein, E. M., & Putnam, F. W. (1986). Development, reliability and validity of a dissociation scale. *Journal of Nervous and Mental Disease, 174*(12), 727–735.

Bogels, S. M., Mulkens, S., & De Jong, P. J. (1997). Task concentration training and fear of blushing. *Clinical Psychology and Psychotherapy, 4*(4), 251–258.

Butler, G., & Mathews, A. (1983). Cognitive processes in anxiety. *Advances in Behaviour Research And Therapy, 5*(1), 51–62.

Chambless, D. L., Caputo, G. C., Bright, P., & Gallagher, R. (1984). Assessment of fear of fear in agoraphobics – the body sensations questionnaire and the agoraphobics questionnaire. *Journal of Consulting and Clinical Psychology, 52*(6), 1090–1097.

Clark, D. M. (1986). A cognitive model of panic. *Behaviour Research and Therapy, 24*, 461–470.

Clark, D. M., Salkovskis, P. M., Gelder, M., Koehler, C., Martin, M., Anastasiades, P., et al. (1988). *Tests of a cognitive theory of panic*. In I. Hand, & H. U. Wittchen (Eds.), *Panic and phobias* (Vol. 2); (pp. 149–158). Berlin: Springer-Verlag.

Clark, D. M., Salkovskis, P. M., Ost, L. G., Breitholtz, E., Koehler, K. A., & Westling, B. E. (1997). Misinterpretation of body sensations in panic disorder. *Journal of Consulting and Clinical Psychology, 65*, 203–213.

Cox, B. J., Swinson, R. P., Endler, N. S., & Norton, G. R. (1994). The symptom structure of panic attacks. *Comprehensive Psychiatry, 35*, 349–353.

Davidson, J. R. T., Kudler, H. S., Saunders, W. B., & Smith, R. D. (1990). Symptom and co-morbidity patterns in World War II and Vietnam veterans with post-traumatic stress disorder. *Comprehensive Psychiatry, 31*, 162–170.

Guralnik, O., Schmeidler, J., & Simeon, D. (2000). Feeling unreal: cognitive processes in depersonalisation. *American Journal of Psychiatry, 157*, 103–109.

Haenen, M. A., Schmidt, A. J. M., Kroeze, S., & van den Hout, M. A. (1996). Hypochondriasis and symptom reporting – the effect of attention versus distraction. *Psychotherapy and Psychosomatics, 65*(1), 43–48.

Harvey, J. M., Richards, J. C., Dziadosz, T., & Swindell, A. (1993). Misinterpretation of ambiguous stimuli in panic disorder. *Cognitive Therapy and Research, 17*, 235–248.

Horney, K. (1951). *Neurosis and human growth: The struggle towards self-realization*. London: Routledge & Kegan Paul Ltd.
Hunter, E. C. M., Baker, D., Phillips, M. L., Sierra, M., & David, A. S. (2005). Cognitive behaviour therapy for depersonalisation disorder: an open study. *Behaviour Research and Therapy, 43*(9), 1121–1130.
Hunter, E. C. M., Phillips, M. L., Chalder, T., Sierra, M., & David, A. S. (2003). Depersonalisation disorder: a cognitive-behavioural conceptualisation. *Behaviour Research and Therapy, 41*(12), 1451–1467.
Hunter, E. C. M., Sierra, M., & David, A. S. (2004). The epidemiology of depersonalisation and derealisation: a systematic review. *Social Psychiatry and Epidemiology, 39*, 9–18.
Latz, T. T., Kramer, S. I., & Hughes, D. L. (1995). Multiple personality disorder among female inpatients in a state hospital. *American Journal of Psychiatry, 152*, 1343–1348.
Lee, W. E., Kwok, C. H. T., Hunter, E. C. M., Richards, M., & David, A. S. (2013). Prevalence and childhood antecedents of depersonalization syndrome in a UK birth cohort. *Social Psychiatry and Psychiatric Epidemiology, 47*(2), 253–261.
Leonard, K. N., Telch, M. J., & Harrington, P. J. (1999). Dissociation in the laboratory: a comparison of strategies. *Behaviour Research and Therapy, 37*(1), 49–61.
MacLeod, A. K., Haynes, C., & Sensky, T. (1998). Attributions about common bodily sensations: their associations with hypochondriasis and anxiety. *Psychological Medicine, 28*, 225–228.
Mathew, R. J., Wilson, W. H., Humphreys, D., Lowe, J. V., & Weithe, K. E. (1993). Depersonalisation after marijuana smoking. *Biological Psychiatry, 33*, 431–441.
McGuire, P. K., Cope, H., & Fahy, T. A. (1994). Diversity of psychopathology associated with methylenedioxymethamphetamine ('ecstasy'). *British Journal of Psychiatry, 165*, 391–395.
McNally, R. J., & Foa, E. B. (1987). Cognition and agoraphobia: bias in the interpretation of threat. *Cognitive Therapy and Research, 11*, 567–581.
Medford, N., Baker, D., Hunter, E., Sierra, M., Lawrence, E., Phillips, M. L., et al. (2003). Chronic depersonalisation following illicit drug use: a controlled analysis of 40 cases. *Addiction, 98*, 1731–1736.
Michal, M., Beutel, M. E., Jordan, J., Zimmermann, M., Wolters, S., & Heidenreich, T. (2007). Depersonalisation, mindfulness, and childhood trauma. *Journal of Nervous and Mental Disease, 195*(8), 693–696.
Miller, P. P., Brown, T. A., DiNardo, P. A., & Barlow, D. H. (1994). The experimental induction of depersonalisation and derealisation in panic disorder and non-anxious subjects. *Behaviour Research and Therapy, 32*(5), 511–519.
Noyes, R., Hoenk, P. R., Kuperman, S., & Slymen, D. J. (1977). Depersonalisation in accident victims and psychiatric patients. *Journal of Nervous & Mental Disease, 164*, 401–407.
Noyes, R., Jr., & Kletti, R. (1977). Depersonalisation in response to life-threatening danger. *Comprehensive Psychiatry, 18*, 375–384.
Robbins, J. M., & Kirmayer, L. J. (1991). Attributions of common bodily symptoms. *Psychological Medicine, 21*, 1029–1045.
Rode, S., Salkovskis, P. M., & Jack, T. (2001). An experimental study of attention, labelling and memory in people suffering from chronic pain. *Pain, 94*(2), 193–203.
Ross, C. A. (1991). Epidemiology of multiple personality disorder and dissociation. *Psychiatric Clinics of North America, 14*, 503–517.
Salkovskis, P. M. (1996). The cognitive approach to anxiety: threat beliefs, safety seeking behaviour and the special case of health anxiety and obsessions. In P. Salkovskis (Ed.), *Frontiers of cognitive therapy*. New York: Guilford Press.
Salkovskis, P., Warwick, H. M. C., & Deale, A. C. (2003). Cognitive-behavioural treatment for severe and persistent health anxiety (hypochondriasis). *Brief Treatment and Crisis Intervention, 3*, 353–368.
Schilder, P. (1939). The treatment of depersonalisation. *Bulletin of the New York Academy of Medicine, 15*, 258–272.
Sedman, G. (1966). Depersonalisation in a group of normal subjects. *British Journal of Psychiatry, 112*, 907–912.
Sensky, T. (1997). Causal attributions in physical illness. *Journal of Psychosomatic Research, 43*(6), 565–573.
Sensky, T., MacLeod, A. K., & Rigby, M. F. (1996). Causal attributions about common somatic sensations among frequent general practice attenders. *Psychological Medicine, 26*, 641–646.
Shaklee, H., & Fischoff, B. (1982). Strategies of information search in causal analysis. *Memory and Cognition, 10*, 520–530.
Shilony, E., & Grossman, F. K. (1993). Depersonalisation as a defence mechanism in survivors of trauma. *Journal of Traumatic Stress, 6*(1), 119–128.
Sierra, M., & Berrios, G. E. (1998). Depersonalisation: neurobiological perspectives. *Biological Psychiatry, 44*, 898–908.
Sierra, M., & Berrios, G. E. (2000). The Cambridge depersonalisation scale: a new instrument for the measurement of depersonalisation. *Psychiatry Research, 93*, 153–164.
Sierra, M., Senior, C., Dalton, J., McDonough, M., Bond, A., Phillips, M. L., et al. (2002). Autonomic response in depersonalisation disorder. *Archives of General Psychiatry, 59*, 833–838.
Simeon, D., & Abugel, J. (2006). *Feeling unreal: Depersonalisation disorder and the loss of the self*. Oxford University Press.
Spitzer, M. B., Gibbon-Miriam, R. L., & Williams, J. B. W. (1996). *Structured clinical interview for DSM IV axis I disorders, clinician version (SCID-IV)*. Washington, D.C.: American Psychiatric Press.
Steinberg, M. (1994). *Structured clinical interview for DSM-IV dissociative disorders (SCID-D)*. Washington, D.C.: American Psychiatric Press.
Torch, E. M. (1987). The psychotherapeutic treatment of depersonalisation disorder. *Hillside Journal of Clinical Psychiatry, 9*(2), 133–143.
Warwick, H. M. C., & Salkovskis, P. M. (1990). Hypochondriasis. *Behaviour Research and Therapy, 28*, 105–117.
Wells, A., White, J., & Carter, K. (1997). Attention training: effects on anxiety and beliefs in panic and social phobia. *Clinical Psychology and Psychotherapy, 4*(4), 226–232.
Westling, B. E., & Öst, L. G. (1993). Relationship between panic attack symptoms and cognitions in panic disorder patients. *Journal of Anxiety Disorders, 7*, 181–194.
World Health Organisation. (1992). *The ICD-10 classification of mental and behavioral disorders: Clinical descriptions and diagnostic guidelines*. Geneva.