Depersonalisation disorder: a cognitive–behavioural conceptualisation

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Abstract

Depersonalisation (DP) and derealisation (DR) are subjective experiences of unreality in, respectively, one's sense of self and the outside world. These experiences occur on a continuum from transient episodes that are frequently reported in healthy individuals under certain situational conditions to a chronic psychiatric disorder that causes considerable distress (depersonalisation disorder, DPD). Despite the relatively high rates of reporting these symptoms, little research has been conducted into psychological treatments for this disorder. We suggest that there is compelling evidence to link DPD with the anxiety disorders, particularly panic. This paper proposes that it is the catastrophic appraisal of the normally transient symptoms of DP/DR that results in the development of a chronic disorder. We suggest that if DP/DR symptoms are misinterpreted as indicative of severe mental illness or brain dysfunction, a vicious cycle of increasing anxiety and consequently increased DP/DR symptoms will result. Moreover, cognitive and behavioural responses to symptoms such as specific avoidances, 'safety behaviours' and cognitive biases serve to maintain the disorder by increasing awareness of the symptoms, heightening the perceived threat and preventing disconfirmation of the catastrophic misinterpretations. A coherent model facilitates the development of potentially effective cognitive and behavioural interventions.
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Depersonalisation is an experience in which the individual feels a sense of unreality and detachment from themselves. This is often accompanied by the symptom of derealisation in which the external world also appears unfamiliar (Diagnostic and Statistical Manual of Mental Disorder (DSM-IV), American Psychiatric Association, 1994; ICD–10 Classification of Mental and Behav-

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ioural Disorders (ICD-10), World Health Organisation, 1992). Sufferers describe their experiences of unreality as if they are living in a dream, and their sense of detachment from the world as though they are viewing life from behind glass. These experiences are not delusional since the sufferer retains insight that these are subjective phenomena, rather than objective reality. Alongside these core diagnostic criteria, sufferers often report a wide spectrum of distortions and impairments to affective, cognitive and physiological/perceptual functioning (see Fig. 1).

Epidemiological surveys have found that transient experiences of depersonalisation and/or derealisation are common. The incidence of transient life-time experiences of DP/DR is estimated to be between 34 and 70% in non-clinical populations, often occurring under conditions of stress, fatigue or drug use (Dixon, 1963; Sedman, 1966, 1970; Trueman, 1984). Furthermore, a 1 year prevalence rate of 23% was reported for the symptoms of either depersonalisation or derealisation in a US rural community sample (Aderibigbe, Bloch, & Walker, 2001) and a 1 month prevalence of a clinically significant depersonalisation syndrome measured by standardised psychiatric interviews was found to be between 1.2 and 1.7% from two urban samples in the UK (Bebbington, Hurry, Tennant, Sturt, & Wing, 1981; Bebbington, Marsden, & Brewin, 1997). Surveys of psychiatric populations have found lifetime experiences of DP/DR reported in 80% of inpatients, of

Affective
- Emotional numbing (for both positive and negative affect)
- Lack of empathy
- Sense of isolation
- Depression
- Anxiety
- Dream-like state
- Loss of motivation
- Loss of a sense of the consequences of one's behaviour

Cognitive
- Impaired concentration
- Mind 'emptiness' or 'racing thoughts'
- Memory impairments
- Impaired visual imagery
- Difficulty in processing new information

Physiological / Perceptual
- Partial or total physiological numbing
- Feelings of weightlessness / hollowness
- Lack of a sense of physical boundaries
- Sensory impairments (e.g. taste, touch, microscopia and/or macroscopia)
- Sensory distortions (e.g. sound, loss of colour)
- Dizziness
- External world appears flat and 2 dimensional
- Objects do not appear solid
- Loss of sense of recognition to one's own reflection and voice.
- Changed perception of time

Fig. 1.   Main symptoms of depersonalisation disorder.

whom 12% describing this as severe and unremitting (Brauer, Harrow, & Tucker, 1970). Current symptoms of DP/DR in psychiatric inpatients have been reported in 8% (Parikh, Sheth, & Apte, 1981) and 56% of those surveyed (Davidson, 1966). The symptoms of depersonalisation disorder are the same as those experienced in transient episodes, except that in DPD the symptoms are experienced chronically, cause significant distress and result in functional impairment (Shorvon, Hill, Burkitt, & Halstead, 1946; Ackner, 1954). Depersonalisation disorder can occur as a primary disorder in the absence of other co-morbid conditions, although the prevalence of this is unknown.

Despite the relatively high rates of reporting of these symptoms, the published literature on psychological treatments for DP/DR is confined to single case studies, except for one larger series (Ackner, 1954). Case studies reporting successful outcomes have employed psychoanalytical techniques alone (Torch, 1987), psychoanalysis combined with abreaction by intravenous diazepam (Ballard, Mohan, & Handy, 1992), or family therapy (Cattell & Cattell, 1974). Behavioural methods such as negative reinforcement (Blue, 1979) and imaginal exposure (Sookman & Solyom, 1978) also resulted in a reduction of symptomatology, but in vivo exposure proved ineffectual (Sookman & Solyom, 1978). However, these studies are limited in that they rely on clinical judgement alone to assess outcome rather than quantitative methods. A literature search using Medline, Psychlit and Web of Science databases, found no published randomised controlled trials of psychological intervention for DP/DR up to the end of 2001. Consequently, there is no standard treatment protocol and despite some success with individual cases the consensus view remains that DPD has a poor prognosis for psychotherapeutic intervention (Schilder, 1939; Simeon & Hollander, 1993; Roth, 1960). Given the considerable success of cognitive–behavioural approaches in the treatment of emotional disorders, the application of this approach to depersonalisation disorder seems timely. However, in order to develop effective treatments a conceptual framework is necessary. In this paper we will suggest that although DPD is classified as a dissociative disorder in DSM-IV, it is also strongly associated with the anxiety disorders, particularly panic, and that although DPD may be conceptualised from either perspective, an anxiety disorder framework facilitates a cognitive–behavioural model of DPD (e.g. Beck, 1976) and may provide a more fruitful basis for interventions. Such a model will be presented in which the chronic condition of DPD results from the catastrophic misinterpretations of the normally transient symptoms of DP/DR. Following a discussion of the supporting literature, we will make recommendations for treatment derived from the model.

1. Depersonalisation disorder: associations with anxiety

Depersonalisation disorder is classified as a dissociative disorder in DSM-IV (American Psychiatric Association, 1994), alongside dissociative amnesia, fugue and identity disorder (DID). The definition of the dissociative disorders is that there is a 'disruption in the usually integrated functions of consciousness, memory, identity, or perception of the environment' (DSM-IV, p. 477). In this respect, DPD has dissociative features in that sufferers experience a disruption in their previous integrated sense of self, as well as subjective detachment from the external world and from their own mental processes. However, according to the DSM-IV definition, five of the characteristics of the other dissociative disorders are atypical of DPD. Firstly, sufferers of DPD do not typically experience significant periods of memory loss as seen in dissociative amnesia,

fugue states or dissociative identity disorder. Secondly, although there may be a sense of detachment from the external world, there is no loss of conscious awareness of the self or the external environment in DPD. Indeed, it is an acute awareness of the discrepancy between the sufferer's current altered perception of themselves and their environment in comparison to their pre-morbid state that causes marked distress. Moreover, unlike the other dissociative disorders where there is typically a pattern of alternating between non-dissociative and dissociative states, in DPD the most common pattern is of unremitting symptomatology, with little fluctuation in severity (see Baker et al., 2003). Fourthly, there is a strong association between trauma and dissociative disorders. Sufferers typically report a precipitating trauma and those with DID often give histories of physical or sexual abuse (Putnam et al., 1996; Ross et al., 1990). Early trauma histories, particularly of childhood sexual abuse, have also been associated with increased frequencies of dissociative tendencies (Chu & Dill, 1990; Neumann et al., 1996; Sanders & Giolas, 1991; van Ijzendoorn & Schuengel, 1996). Yet despite support for the temporal relationship between traumatic events (e.g. road traffic accidents) and transient symptoms of depersonalisation and/or derealisation (Mayou et al., 2001; Noyes & Kletti, 1977; Shilony & Grossman, 1993), no such strong association between childhood trauma and the chronic condition of DPD has been established. Roth (1960) found no significant differences between DPD patients and controls in the incidence of childhood trauma and in a more recent sample of 30 cases of depersonalisation disorder (Simeon et al., 1997), the incidence of trauma was low, although a more recent study found childhood emotional abuse to be a predictor of DPD (Simeon et al., 2001). Finally, sufferers of DPD report absent to mild amnesia and do not experience alternate personalities (Steinberg, 1991), hence co-morbidity with other dissociative disorders is rare, although conversely those diagnosed with DID will commonly report depersonalisation and/or derealisation symptoms (Dell, 2002; Ross et al., 1990; Saxe, van der Kolk, Berkowitz, Chinman, Hall, Lieberg, & Schwartz (1993); Steinberg, Rounsaville, & Cicchetti, 1990). Some authorities propose that depersonalisation lies on a 'continuum of dissociation' (Braun, 1997), while others suggest the existence of 'distinct dissociative types' (Putnam et al., 1996), such as dissociative detachment and compartmentalisation (Allen, 2001; Holmes, Mansell, Brown, Fearon, Hunter, Frasquilho et al., submitted manuscript).

Alternatively, there is substantial support for the conceptualisation of DPD as an anxiety disorder. Cognitive symptoms experienced by DPD sufferers are typically those of increased arousal, such as having 'racing thoughts' or 'mind emptiness', with subjective deficits in concentration and attention (Wells & Matthews, 1994). According to DSM-IV criteria (American Psychiatric Association, 1994) several diagnostic features of DPD are akin to those of acute stress disorder and post traumatic stress disorder, such as 'emotional anaesthesia', a sense of detachment or of 'being in a daze', and estrangement from others. Similarly, both sufferers of panic and DPD report symptoms of feeling dizzy or faint or experiencing parasthesias, and the behavioural consequences of experiencing DPD frequently result in the avoidance of situations that evoke greater levels of anxiety, such as social situations or crowds as in agoraphobia.

There is a high incidence of co-morbidity of DP/DR symptoms with the anxiety disorders, such as panic (Cassano et al., 1989; Marshall et al., 2000; Mayer-Gross, 1935; Segui et al., 2000; Simeon et al., 1997), 'free-floating anxiety' (Roth, 1960), generalised anxiety disorder (Simeon et al., 1997), post-traumatic stress disorder (Bremner, Krystal, Putnam, Southwick, Marmar, Charney et al., 1998), obsessive compulsive disorder (Roth, 1960; Sedman & Reed, 1963; Shorvon,

Hill, Burkitt & Halstead, 1946; Simeon et al., 1997) and hypochondriasis (Shorvon et al., 1946). DP/DR symptoms are also reported in patients with depression (Ackner, 1954; Mayer-Gross, 1935; Roth, 1960; Sedman & Reed, 1963) and the dissociative disorders (Ross et al., 1990; Steinberg, Rounsaville, & Cicchetti, 1990), where there may be relatively high levels of co-morbid anxiety.

Further support for the association between DPD and anxiety comes from a neurobiological perspective, since it has been suggested that depersonalisation is the manifestation of a 'hard-wired' brain response, which may be triggered in certain circumstances, especially in states of extreme anxiety, where at a certain threshold, the symptoms of anxiety diverge into a state of depersonalisation/derealisation (e.g. Mayer-Gross, 1935). Recent theorists (e.g. Sierra & Berrios, 1998) have suggested that depersonalisation may be a result of changes to the functioning of those brain regions which are involved in the control and expression of emotional responses, namely, the amygdala, the anterior cingulate and the medial prefrontal cortex. They conclude that this pattern is likely to be a mechanism for 'dealing with extreme anxiety combining a state of increased alertness with a profound inhibition of the emotional response system' (p. 903). A pattern of reduced 'limbic' and increased prefrontal response to emotional stimuli has recently been demonstrated in patients with DPD (Phillips, Medford, Senior, Bullmore, Brammer, Andrew et al. (2000). These findings have been interpreted as evidence for an increased regulation by prefrontal regions of neural regions important for emotion processing in DP/DR. DP/DR may therefore result from an abnormally increased regulatory response to severe arousal and anxiety.

Within the anxiety disorders, there appear to be strong links between the symptoms of DP/DR and panic. Depersonalisation and derealisation are two of the symptoms of panic according to the diagnostic criteria for DSM IV, and two studies of panic sufferers have found, respectively, 22 and 34% reporting depersonalisation and/or derealisation during their attacks (Segui et al., 2000; Cassano et al., 1989). Moreover, those who experienced DP/DR symptoms reported a greater severity of panic (Marshall et al., 2000; Segui et al., 2000). In a survey of 204 people with a putative diagnosis of DPD who were recruited to the Depersonalisation Research Unit at the Institute of Psychiatry in London via clinical referral ($n = 130$), unit's website ($n = 55$), media articles ($n = 14$) and patient support organisations ($n = 5$), 71% reported that they had previously been diagnosed with an anxiety disorder, 73% of respondents reported current 'panic attacks' and 59% stated that they were 'afraid to go out alone' (Baker et al., 2003). Several authors have noted an association between panic and the initial onset of DPD (Mayer-Gross, 1935; Roth, 1960; Shorvon et al., 1946). Mayer-Gross (1935) reported that in 39% of his patients the symptoms of DP or DR 'appear suddenly without any warning … a patient sitting quietly reading by the fireside is overwhelmed by it in full blast *together with an acute anxiety attack* ' (p. 116, italics added). Shorvon et al. (1946) found that 92% of the 66 cases of DPD he studied reported symptoms starting in a similar manner.

So why do some panic patients develop DP/DR and not others? One explanation is that DPD patients show a 'paradoxical' autonomic response to stimuli or situations which normally increase arousal. Such patients show reduced levels of arousal as indexed by skin conductance responses (Lader, 1975; Sierra, Senior, Dalton, McDonough, Bond, Phillips et al., 2002). A similar pattern has been demonstrated in a subgroup of women shortly after a violent sexual assault (Griffin, Resick, & Mechanic, 1997). Women rated as higher on a peritraumatic dissociation scale showed reduced galvanic skin and cardiovascular responses when recalling the traumatic events when

compared to neutral events. Hence there appears to be a link between dissociation and DP/DR, but it is mediated through a response to anxiety-provoking situations. We speculate that it is this reduced physiological reaction in response to anxiety that results in an unpleasant 'unreal' state. This may be interpreted as impending madness and may lend itself to attributions of damage or malfunctioning of the nervous system.

Other common precipitants for DP and DR include acute intoxication or withdrawal from alcohol and/or a variety of drugs, especially 'ecstasy' (McGuire, Cope, & Fahy, 1994), marijuana (Moran, 1986) and hallucinogens such as LSD (Waltzner, 1972). These symptoms may be prolonged if usage took place during a time of stress alongside co-morbid psychiatric diagnoses (Keshaven & Lishman, 1986; Szymanski, 1981) even with only infrequent use (Simeon et al., 1997). Depression is also a common precipitating factor, with Mayer-Gross (1935) reporting 50% of sufferers describing the onset of their depersonalisation during the course of an episode of depression and Ackner (1954) and Sedman (1972) defining concepts of a 'depressive depersonalisation' syndrome. In these cases it is possible that the main focus of concern for the sufferer is the sense of detachment and emotional numbing which result from the depression. Finally, in a minority of cases no precipitating factor can be elicited (Simeon & Hollander, 1993). This may be due to the onset of symptoms occurring following, rather than during, a period of extreme stress (Shorvon et al., 1946) or because of lack of disclosure (Roth, 1960).

In summary, there is strong circumstantial evidence for an association between DPD and the anxiety disorders, particularly panic. This is fortunate since cognitive–behavioural theories of anxiety disorders are more advanced than those for dissociative disorders and hence provide a coherent framework and rationale for treatment.

2. A cognitive model of depersonalisation disorder

Transient symptoms of DP/DR are common phenomena in normal populations, as are symptoms of anxiety. Cognitive models of anxiety disorders such as those of panic (Clark, 1986) or health anxiety (Warwick & Salkovskis, 1990) suggest that it is the interpretation of these common symptoms that determines whether they develop into a chronic disorder. More specifically, it is the catastrophic misinterpretation of these symptoms as indicating threat that leads to a vicious cycle of increasing symptoms, since the secondary anxiety about the meaning of the symptoms adds to the initial anxiety.

Clark (1986) and Wells (1997) propose that many of the symptoms of panic are associated with specific cognitions. These include misinterpreting a difficulty in breathing as indicative of potential suffocation, palpitations as early indicators of a heart attack, and dizziness as the imminent forerunner of fainting. They also identify that the DP/DR symptoms that are common in patients with panic disorder are associated with the belief that these are a sign of madness or potential loss of control. Such beliefs are likely to increase the person's fear in the situation, which in turn will increase their anxiety, and so the cycle perpetuates itself.

Further support for these types of cognitions in DPD comes from the few authors who have investigated patients' attributions for their symptoms. Shorvon et al. (1946) described how the symptoms of depersonalisation 'frequently sets up a panic state, and it is common for patients to complain … of fears of insanity, of impending death …' (p. 782). Roth (1960) noted the relation-

ship between DPD and anxiety states given that patients with DPD commonly experience free-floating anxiety, where 'attention becomes focussed on some somatic accompaniment of anxiety ... which [is] elaborated into fears of heart disease, cancer or brain disease with impending insanity' (p. 299). Other examples of the misinterpretation of DP/DR symptoms reported by sufferers seen in our clinic have included beliefs about having permanent brain damage, especially in those with a history of drug and alcohol use. In a questionnaire on attributions for symptoms given to 15 consecutive patients assessed in our clinic prior to treatment, 12 (80%) endorsed the statement 'something has gone wrong with my brain' (for example, Creutzfeldt-Jakob Disease (CJD)) as being a likely cause of their symptoms. Those with symptoms of physiological numbing, perhaps a true reflection of autonomic under-arousal, often report fears of becoming invisible or disappearing, as was also noted by Ackner (1954).

Ackner (1954) suggested that these, what we would now call catastrophic attributions, reduced the threshold for the perception of sensations that could be interpreted as threatening. He noted that those with DPD developed an 'increased responsiveness for anxiety of internal origin, whereas that of external origin was reduced' (p. 852). Palmer (1941) reported that panic sufferers with co-morbid obsessional features tended to focus during anxiety attacks on the 'psychic components, especially the unreality, of the anxiety attacks', and that this emphasis on the symptoms of DP/DR continued to be a perpetuating factor after the anxiety had subsided. Furthermore, Trueman (1984) commented 'this cycle of anxiety followed by depersonalization or derealization may become semi-autonomous; thoughts of impending episodes arouse anxiety and consequent dissociative experiences' (p. 91).

Cognitive theories of emotional disorders (e.g. Beck, 1976) predict that negative automatic thoughts are likely to be derived from underlying assumptions and core beliefs about the self. There is some evidence that those who develop DPD may have pre-morbid concerns regarding their mental health. Ackner (1954) questioned a series of 15 consecutive patients with DPD and found that those who reported the most distress were those who feared their symptoms indicated impending insanity or death. In the eight patients who feared going mad, half reported psychosis in a close family member and six reported a preoccupation with fears of insanity prior to the onset of their DPD. Whereas the majority of people who experience transient DP/DR symptoms will attribute their symptoms to situational explanations (e.g. tiredness, stress, the effects of drug intoxication, or fear in a specific situation) and believe that when their circumstances change their DR/DR symptoms will dissipate, it is possible that those who develop the chronic condition of DPD may have pre-morbid concerns regarding their vulnerability to mental illness, and that this makes the transient DP/DR symptoms more threatening since these experiences appear to confirm their worst fears. In the survey of 204 cases of DPD (Baker et al., 2003), 10% reported a family member with a history of DPD, and 30% reported a history of psychiatric disorder in a first degree relative. Moreover, the latter group were found to score significantly higher on a standardised measure of dissociation (the dissociative experiences scale, Bernstein & Putnam, 1986) than those without a reported familial psychiatric history. Although the evidence for catastrophic beliefs in DPD is extremely limited in existing studies, and thus the model of DPD presented here is largely speculative, the findings from the few studies cited above that have investigated cognitions and attributions in DPD sufferers appear to support the model. However, further empirical testing of the model is urgently needed.

In summary, the cognitive model of DPD proposes that the catastrophic misinterpretation of

the transient symptoms of DP/DR (common in the population at large and in around a third of panic sufferers) as indicative of madness, loss of control, becoming invisible and/or brain disease is likely to lead to an increase in anxiety (although this may be accompanied by a 'paradoxical' decrease in autonomic arousal) which serves to exacerbate and perpetuate the symptoms of DP/DR. Moreover, cognitive models of anxiety disorders suggest that the person is also likely to develop a range of behaviours and cognitive biases that form a maintenance cycle to further perpetuate the symptoms. For example, there may be an avoidance of certain situations which have triggered the symptoms; there may be behaviours which the sufferer adopts which he or she believes help prevent the feared outcome (i.e. 'safety behaviours'), and there may be cognitive or attentional biases, such as an increase in symptom monitoring leading to an increased likelihood in the perception of symptoms and a reduced threshold for the perception of threat (see Fig. 2).

In the case of DPD, avoidances are likely to include any situation that increases anxiety, with social situations being the most commonly cited. For example, nearly half of the patients with DPD in the study by Simeon and colleagues (1997) were diagnosed with co-morbid social phobia. Roth and Argyle (1988) noted that since crowded places may intensify the depersonalisation symptoms this too could lead to a decrease in socialising. Moreover, the sense of detachment experienced in DPD frequently leads to de-motivation, with a decrease in activities and productivity (Simeon & Hollander, 1993). De-motivation, combined with an inability to derive
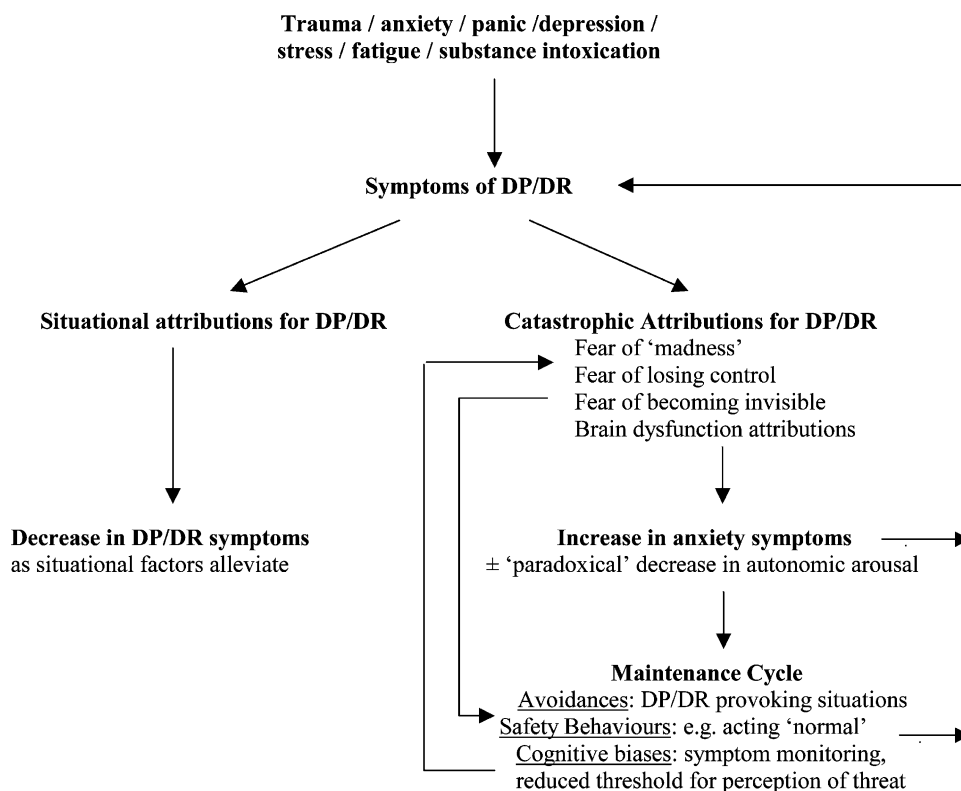

Fig. 2.   Cognitive–behavioural model of depersonalisation disorder

pleasure from previously enjoyable activities (Simeon et al., 1997) can in turn result in low mood and a sense of frustration leading to greater levels of avoidance.

The belief that the DP/DR symptoms are indicative of madness (Oberndorf, 1950; Simeon & Hollander, 1993), combined with the social stigmatisation of being identified as having a mental illness, leads many patients with DPD to disguise their symptoms by developing the safety behaviour of attempting to appear 'normal'. Horney (1951), states that the DPD sufferer aims to 'feel what he should feel, wish what he should wish and like what he should like. In other words, the tyranny of the should drives him frantically to be something different from what he is or could be ... that his real self fades' (p. 159). Ironically however, acting a role is likely to further increase the sufferer's sense of detachment. Some investigators have noted that patients with DPD share a tendency to increased introspection (Mayer-Gross, 1935) and over-intellectualisation as seen in obsessive personalities (Simeon & Hollander, 1993). Ackner (1954) suggested that over-intellectualisation was likely to maintain affective distancing.

The association with obsessive–compulsive symptomatology and depersonalisation, especially with an excessive focus on the self, can lead to a pattern of symptom monitoring and checking behaviour (Simeon & Hollander, 1993). This has been described in DPD sufferers as 'compulsive self-scrutiny' (Roth, 1960) and 'hypochondriacal pre-occupation' (Sedman, 1972). Mayer-Gross (1935) reported that all 26 patients he investigated displayed obsessional symptoms during the course of their depersonalisation. However, as with hypochondriasis, increased monitoring is likely to lead to attentional biases that heighten the detection and severity of symptoms (Warwick & Salkovskis, 1990) and may act to maintain the disorder. Interestingly, one of the few pharmacological interventions found to be effective in a controlled trial is Clomipramine— an agent used in the treatment of OCD (Simeon, Stein, & Hollander, 1998). Experimental research supports this clinical observation since induction of the symptoms of DP/DR in controls was achieved by the sustained focussing of attention (Miller, Brown, DiNardo, & Barlow, 1994). It has been suggested that the increase in the level of self-focussed attention, where the concept of the 'self' and the world has become the subject of obsessive focus may be responsible for the experience of both the self and the external world appearing unreal or unfamiliar (Jaspers, 1963).

3. Implications of the model for treatment

The cognitive–behavioural model of DPD described above, based on models of anxiety disorders, provides a framework for the construction of an individual conceptualisation of those factors that are likely to have predisposed and precipitated the presenting problem, and which continue to perpetuate the symptoms. Each of these individual factors should be addressed in therapy, with initial emphasis placed on effecting change on those factors deemed to be maintaining the disorder and later focus on predisposing and precipitating factors to prevent relapse. Below we outline some interventions that are likely to be useful in the treatment of DPD.

3.1. Psycho-education and normalising

Engagement of the patient through the shared understanding of a clear rationale for their symptoms and treatment is a cornerstone of effective CBT. This is particularly emphasised with DPD

sufferers who have been found to have an average time of 7–10 years before being given their correct diagnosis of DPD (Steinberg, Cicchetti, Buchanan, Hall, & Rounsaville, 1993) or around 12 years in a UK clinic (Baker et al., 2003; Phillips, Sierra, Hunter, Lambert, Medford, Senior et al., 2001) and are likely to have had conflicting information about their problems during this time. Psycho-education and normalising of their symptoms therefore can form an important part of treatment. Many patients report the enormous sense of relief that comes from being told that their often bewildering array of symptoms form a well-defined syndrome. Information regarding how commonly the transient symptoms of DP/DR are experienced in the population, particularly during periods of stress or threat, is reassuring to the sufferer. If the patient has a history of panic, the high proportion of panic sufferers who report transient symptoms of DP/DR during panic states can be presented. The 'normalising' description of the role of DPD as a protective method for dealing with these overwhelming feelings by effectively distancing oneself from them finds favour with most sufferers. If during assessment the patient has identified anxiety producing situations as increasing their current symptomatology, this can be used as additional evidence to support the links with anxiety.

3.2. Diary keeping

This can be useful in highlighting to the patient the variability of their symptoms, since many sufferers have a tendency to view their DPD as unremitting. It will also emphasise that changes to their behaviour and their thoughts can have an impact on their DPD and increases the belief that their symptoms are controllable and not necessarily a sign of permanent neurological damage, as well as introducing the concept of 'homework'. For the therapist, the diary may also highlight symptom-led behaviour by indicating a 'boom and bust' pattern, with extremes of activity and inactivity similar to that frequently seen in chronic pain and chronic fatigue syndrome where the person adjusts their behaviour according to the severity of their symptoms (Deale, Chalder, Marks, & Wessely, 1997). If this is the case, an average level of activity can be calculated, and achievable targets agreed upon with the patient. The therapist may also want to increase activity levels if the diary clearly indicates an overall low level of activity that may be contributing to lowered mood and motivation with increased introspection and symptom monitoring.

3.3. Reducing avoidance

Behavioural interventions are likely to include reducing avoidance through graded exposure. Typical situations that are avoided are those that result in increased anxiety, such as socialising, crowded public places and driving due to fear that the unreality of the situation will lead to accidents. A review of techniques to overcome social anxiety will be valuable in the majority of cases, for example videotaping of social interactions may be useful for the sufferer to reassure them how little their symptoms are detectable to others. Some 'safety behaviours' might include maintaining a fixed expression, keeping very still, making minimal eye contact, trying to say the 'right thing', which leaves the sufferer with the feeling that they are simply going through the motions of social interaction. Role playing with, and without, the use of safety behaviours will allow the patient to see for themselves whether their safety behaviour serves to help or hinder their performance.

3.4. Reducing self-focussed attention

Refocusing and grounding techniques are used to combat a range of dissociative phenomena including DP/DR (Kennerley, 1996). Refocusing through the use of specific, predetermined, words, objects, images or self-statements can help the person increase their contact with reality, orientate them to their immediate environment, and break the cycle of increasing self-focussed attention on their symptoms. However, these are likely to be useful strategies only when the DP/DR reported is intermittent so that the sufferer can intervene before symptoms worsen. However, their continual usage in those with severe and relatively non-fluctuating symptoms would be likely to interfere with normal functioning and could develop into a maladaptive 'safety behaviour'. Sufferers reporting severe and chronic DP/DR symptoms may instead be helped by techniques that reduce the degree of self-focussed attention or symptom focussing, since this has been found to worsen symptoms in social phobia (Clark & Wells, 1995), panic (Wells, 1990) and hypochondriasis (Salkovskis and Bass, 1997). Two such interventions are attention training (Wells, 1990; Wells, White, & Carter, 1997) and task concentration training (Bogels, Mullens & De Jong, 1997). In attention training, the patient enhances their ability to control their sustained attention, attention shifting and divided attention skills through a series of exercises. Similarly, in task concentration training, patients are encouraged to gain insight into the proportion of their attention which is focused on (1) internal stimuli; (2) external, irrelevant stimuli; or (3) external, task-related stimuli. Through a series of exercises the patient is trained to increase the degree of externally focussed, task-related, attention, initially in non-threatening situations and subsequently in threatening situations.

3.5. Challenging catastrophic assumptions

Cognitive interventions such as thought records (Beck, Rush, Shaw, & Emery, 1979) can be used to identify specific negative automatic thoughts which occur in anxiety producing situations, or when the symptoms of DPD increase. These negative cognitions can then be reality tested and more balanced thoughts offered as replacements.

During assessment it is important to elicit as many of the feared consequences that the patient may have imagined might occur as the worst outcome from their DPD (see Fig. 2). Once the therapeutic alliance is well-established and progress has been made with more behaviourally orientated interventions, these catastrophic misinterpretations can be gradually challenged through education, experimentation and evidence gathering. For example, if a patient fears that an increase in their DPD would result in them losing control, a detailed list of what this would entail should be constructed. The patient is then encouraged to test this hypothesis by carrying out behaviours that have previously increased the DPD severity to discover if their predictions are realised. If this in vivo experimentation cannot be conducted through difficulty in increasing symptoms or patient reluctance to do so, the therapist and patient can instead examine the patient's worst episodes to determine why the feared consequences were not realised. Obtaining belief ratings for each feared consequence at regular intervals through therapy will monitor the success of these interventions.

However, if challenging the catastrophic attributions does not effect change in symptomatology, this may be due to the sufferer having developed a repertoire of safety behaviours that they

believe prevent the feared outcome. As in other cognitive behavioural treatments the setting up of behavioural experiments with clearly defined predictions where sufferers 'drop' their safety behaviours allows them to test the efficacy of these behaviours in preventing the feared catastrophe.

Finally, physiological interventions can also be incorporated in the treatment of patients with DPD, such as applied relaxation training (Ost, 1987) and education regarding the role of hyperventilation in anxiety and panic, with training in diaphragmatic breathing. However, over-reliance on relaxation techniques may exacerbate DP/DR symptoms in some cases by inducing an under-aroused state (Fewtrell, 1984). Since many patients acknowledge fatigue as a trigger for worsening of the DPD, discussion on sleep hygiene, exercise and diet is worthy of inclusion.

In our unit at the Institute of Psychiatry in London, an initial trial has been conducted into the efficacy of CBT with patients with primary DPD (manuscript in preparation). Eleven patients (nine men, mean age 41 years, mean duration of symptoms 18 years) completed a course of CBT that included the above interventions. A range of measures were administered at pre- and post-treatment, and at 6 months follow-up. Significant improvements in patient-defined measures of problem severity, standardised measures of general functioning and depersonalisation/derealisation severity were found at post-treatment and 6 months follow-up. These initial results suggest that a cognitive–behavioural approach to DPD may be effective, but further trials with larger sample sizes and more rigorous research methodology are needed. A case example is included in the Appendix.

4. Conclusions

From the existing literature there appears to be compelling evidence to support an association between DPD and the anxiety disorders. The conceptualisation of DPD within an anxiety disorders framework provides us with an empirically testable model and a rationale for treatment. However, the current lack of empirical research on DPD means that parts of the model proposed in this paper are still very speculative and testing of the model is planned for the near future. It is hoped that further development and refinement of the model will not only provide a better understanding of this disorder that causes considerable distress to many sufferers, but will further increase the efficacy of treatment.

Acknowledgements

Appendix

Case example 1

Case example 1 was a 29-year-old, single man, employed as a journalist, who reported a 12-year history of depersonalisation disorder. He described feeling detached from the world as though

he was living 'inside a bubble' and found it difficult to concentrate since he felt as though his brain had been 'switched off'. His body no longer felt solid and he could not feel himself walking on the ground. The world appeared two-dimensional and he reported his sense of direction and spatial awareness to be impaired. He described himself as having lost his 'sense of himself' and felt that he was acting on 'auto-pilot'. He also reported symptoms of depression and some symptoms of OCD, which took the form of counting and stepping on cracks in the pavement, although he did not report the latter as a problem.

Prior to the onset of his DPD, he experienced transient DP symptoms when intoxicated with cannabis. At the age of 17, he started at a new school and felt very anxious and experienced DP symptoms when not under the influence of cannabis. He described the first time this happened as 'terrifying' since he felt he had 'gone into another world'. He reported difficulty with breathing and believed he may have a brain tumour or that his 'brain was traumatised into a state of panic'. From the age of 17 to 19, the episodes of DP became more frequent until they became constantly present. He reports the symptoms as 'enormously restricting' his life in that he felt frustrated since he has been 'unable to express or enjoy myself'.

Since he rated his depersonalisation as constantly at 90% intensity, diary keeping was initiated. This highlighted the fluctuating nature of his DPD and helped him identify fatigue, low mood and caffeine as triggers for increasing symptomatology. He was encouraged to participate in social activities again, which he had been avoiding, since he found large groups of people anxiety provoking and had found that this made his symptoms worse. As he tended to go home and lie down with the lights off if his symptoms were severe, he was asked to attempt to remain in the situation until the DP/DR reduced. In order to increase his concentration and reduce his self-focussed attention, he was instructed in task concentration training exercises. His catastrophic belief was that the DPD would eventually worsen to the point where he would be unable to function or work since he would 'be so utterly withdrawn that he couldn't speak'. This belief was challenged by examining the evidence for this in terms of his ability to function and communicate in previous, and current, periods when the DP/DR symptoms had been at their very worse. Thought records were used to challenge negative automatic thoughts relating to the DPD and/or his co-morbid depression. He had a total of eight sessions, with 1, 3 and 6 month follow-up appointments.

At the start of treatment the client was assessed using a range of case-specific (problem severity scale: Marks, 1986), general functioning (work and social adjustment scale: Mundt, Marks, Shear, & Greist, 2002), depression (Beck depression inventory (BDI): Beck, Ward, Mendelson, Mock & Erbaugh, 1961) and depersonalisation-specific measures (Cambridge depersonalisation scale (CDS): Sierra & Berrios, 2000; dissociative experiences scale (DES): Bernstein & Putnam, 1986). Prior to treatment, he rated how much his problems upset or interfered with his normal activities as 5.5 on the 0–8 point problem severity scale, where 0 represented no interference at all, 2, slightly/sometimes; 6, markedly/very often; and 8, very severely/continuously. At the end of treatment this had reduced to a 2.5 rating (i.e. just above 'slightly/sometimes'). The work and social adjustment scale was used to assess the extent to which his problem interfered with his life on a similar 0–8 scale in five separate domains of work, home management, social leisure activities, private leisure activities and relationships with others. At the start of treatment his mean score across these five domains was 4.4 out of 8 and this reduced to 2.8 by the end of his eight sessions. His pre-treatment depression score of 26 on the BDI (i.e. in the moderate to severe

range of 19–29) was reduced to 11 (i.e. at the lower end of the mild-moderate range of 10–18). The Cambridge depersonalisation scale includes both a state depersonalisation measure (22 items measuring current severity of DP/DR symptoms on a % visual analogue scale) and a trait depersonalisation measure (29-item scale measuring severity of DP/DR since onset of symptoms). His state CDS score at the beginning of treatment was 33%, which was reduced to 9% at the end of treatment and his mean CDS trait score had reduced from 5 to 3.5 (from a possible range of 0–10). Finally, the dissociative experiences scale (DES, Bernstein & Putnam, 1986) was used as a self-report measure on the % frequency of a range of current, adult dissociative reactions (total scale of 28 items). His score on the DES reduced from 22% initially to 13% by the end of the eight sessions. At 6-month follow-up these gains were maintained, with some further reductions in his trait CDS score. Moreover, he reported that his life was now 'moving on', since he had recently married and taken on new work commitments. He felt the depersonalisation, although still present, had reduced to a tolerable level and that the CBT had been helpful in that he was now able to challenge his negative thoughts and had developed better ways of dealing with his difficulties.

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