Depersonalisation

Founded in 1998, the **DEPERSONALISATION RESEARCH UNIT** *at the Institute of Psychiatry was the first in its field. Now the team describe their progress in the development of cognitive-behavioural and pharmacological treatments.*

DEPERSONALISATION is a psychiatric condition characterised by an alteration in the perception and experience of the self (Mellor, 1988). It was first described in the scientific literature over one hundred years ago (for a historical review see Sierra & Berrios, 1997) and has a prevalence in the general population that has been estimated to be as high as 3 per cent (Trueman, 1984).

Imagine being constantly out of touch with your own feelings and senses: this is similar to depersonalisation disorder. Sufferers of this disorder experience a sense of unreality and detachment from various aspects of themselves, which manifests itself as a sense of disconnection from one's own body, cognition or affective state (DSM-IV: APA, 1994). Clinical sufferers sometimes self-mutilate in an attempt to 'feel' themselves, such is the severity of the condition. Whilst depersonalisation can occur as a primary symptom (Ballard *et al*., 1992), it can also occur as secondary to neurological conditions such as epilepsy or Ménière's disease. It is also sometimes seen in conjunction with other psychiatric conditions, such as panic disorder, depression, schizophrenia and obsessive compulsive disorder (Sedman & Kenna, 1963; Sedman & Reed, 1963). In addition, depersonalisation can occur in healthy individuals after taking drugs such as cannabis or Ecstasy (McGuire *et al*., 1994; Szymanski, 1981) and has been reported during 'near-death' experiences (Noyes *et al*., 1976).

Surprisingly, there have been few contemporary investigations into depersonalisation and fewer still into the development of possible treatments. This lack of study led to the establishment in 1998 of the Depersonalisation Research Unit at the Maudsley Hospital in London. Its mission statement was twofold. First, to initiate research into the disorder; secondly, to develop effective treatment strategies, both pharmacological and psychological. In addition, collection and dissemination of information about depersonalisation is carried out. This is done through the unit's website (see Weblinks box) and through an information sheet that is sent to sufferers and clinical practitioners alike.

Some of the depersonalisation sufferers referred to the unit have participated in a drug trial to investigate the efficacy of treatment with the anticonvulsant lamotrigine through its potential anti-dissociation effects. A further group have participated in the development of a cognitive-behavioural therapy regime, whilst several other individuals have taken part in a number of functional neuroimaging and psychophysiological investigations.

In addition to these treatment programmes, a group of people suffering from the disorder were recruited through the internet, and a comparison was made between an 'online' population and one from the unit's clinic. The database of responses will help to characterise the disorder – this article summarises these investigations. But first we give examples of psychological theories that have been developed to describe depersonalisation.

Psychological theories of depersonalisation

Early psychologists believed that depersonalisation was caused by sensory disturbances in different modalities (the visual modality being the most frequently affected). However, contemporary theories ascribed depersonalisation to an alteration of body sensation *per se* (i.e. coenaesthesia). In recent years Damasio has proposed that the continuous bombardment of consciousness with bodily signals creates a 'sensory background' crucial to determining a sense of self. This would imply that a disruption of this 'sensory background' could result in depersonalisation (Damasio, 1996).

The view that depersonalisation could be explained as a consequence of a malfunction in individual mental functions (i.e. 'mental faculties') became dominant during the second half of the 19th century. Different researchers proposed theories based on a primary dysfunction of memory, emotions or body image (this being a variation of the coenaesthesia theory). All these theories had a major drawback – an inability to account for many of the components (e.g. affective dampening or visual disturbances) of the depersonalisation syndrome (see Sierra & Berrios, 1997, for a review).

The turn of the century marked a departure from the above reductionist views, and higher levels of complexity were adopted. Thus, depersonalisation was explained as the result of the loss of a brain mechanism that causes the *feeling* of mental experiences and their attribution to the self. A similar view, endorsed by the German philosopher and psychiatrist Karl Jaspers, related depersonalisation to a pathological change in the 'activity of the self' (a mechanism that is mediated by the feeling of self-belonging) (Jaspers, 1913/1963). These views are still present, in various forms, in the current understanding of depersonalisation as a 'dissociative' disorder.

However, being cast at such high level of complexity (i.e. that of the self and self-consciousness), these theories are hardly amenable to psychological or neurobiological testing. Psychological theories that *are* amenable to empirical investigation are those that consider depersonalisation to be a pre-formed functional response of the brain to threat or overwhelming anxiety. Such a response is assumed to enhance chances of survival in life-threatening situations by heightening alertness and dampening potentially disorganising levels of anxiety (Mayer-Gross, 1935; Noyes *et al*., 1977).

WEBLINKS

Depersonalisation Research Unit:
www.iop.kcl.ac.uk/depersonalisation
Depersonalisation bulletin board:
depersonalization.hypermart.net/

Indeed there now exists a substantial body of literature demonstrating a link between experiencing trauma and various types of dissociative response (e.g. Chu & Dill, 1990). Depersonalisation disorder, with its state of heightened arousal combined with a dampening of emotional response, is widely viewed as a defence mechanism in the face of severe stress, life-threatening situations or trauma. However, although this response may have been adaptive in the short or medium term, patients describe themselves as unable to 'switch off' this response and feel unable to return to their premorbid state.

To illustrate how the disorder affects individuals, two vignettes of patients seen in the clinic are now presented (see boxes). Details have been changed to protect their anonymity. Patient A presented with the syndrome as a complication of epilepsy; Patient B was more typical, with comorbid anxiety and depression.

The Maudsley database

To date the database contains information from well over 100 respondents. Of this number, two thirds are patients who have been referred to the unit, approximately 20 contacted the unit via our internet site, and a similar number contacted the unit in response to an article published in *The Times* in March 1998.

In our sample, depersonalisation is experienced in roughly equal proportions by both men and women. Those contacting the unit tend to be high academic achievers, with a figure of nearly 70 per cent going on to further education after secondary school suggesting possible selection or referral biases. However, nearly a third of the group are currently unemployed, a figure signifying the impairment in functioning which can result from depersonalisation disorder.

Clinical questionnaires indicated a moderate level of dysfunction, with mean depression and anxiety scores just above the clinical cut-offs. On the Dissociative Experiences Scale (DES) (Bernstein & Putnam, 1986) the group scored relatively highly with a mean score of 21 (compared with normal adult scores of 4–8). Comorbidity with other psychological disorders was high, with respondents reporting diagnoses of depression, anxiety disorders, and even schizophrenia.

The database is continually being updated from new referrals. As such it represents a valuable source of information concerning the disorder. In addition to demographic and clinical information, the database contains responses from a range of psychometric questionnaires. These provide details on many other factors such as visual imagery and self-perception, which may well prove to be related to depersonalisation and we hope will generate useful insights and avenues for future research.

It is widely acknowledged that depersonalisation can often coexist with DSM Axis I psychiatric conditions such as depression and anxiety (i.e. secondary depersonalisation) (Cassano *et al*., 1989). It is also accepted that symptoms of depression and anxiety may develop in those with primary depersonalisation, (Nemiah, 1989). However, the frequency with which primary and secondary depersonalisation occurs is not known.

The DES has three subscales, one of which assesses depersonalisation/derealisation. A recent analysis of the DES revealed eight items that are specific to the detection of depersonalisation (see box); the rest are specific to dissociation, amnesia, and so on. We found that the DES and its subscales correlated with depression and, to a lesser extent, anxiety (assessed

PATIENT B

A 30-year-old male with a family history of panic disorder and alcohol abuse developed depersonalisation after a panic attack. His depersonalisation was constant and was exacerbated by various factors, such as reading, where he reported being intensely depersonalised afterwards, and consuming alcohol, after which he reported being 'very blurred'. He also reported being 'very distant from the real world, and said when he touched someone '…it doesn't feel as if I am really touching them.'

This patient maintained that the depersonalisation feeling was '…always in my head, even when my eyes are closed', and reported the complete loss of visual imagery abilities. He was placed on the antidepressants fluoxetine and later sertraline, which resulted in improvement in depersonalisation. However, residual symptoms remained, which have interfered with work and social functioning.

PATIENT A

A 22-year-old right-handed woman with a family history of depression developed partial epilepsy at 17 years. The seizures were medically intractable, so she underwent a left-sided temporal lobectomy at the age of 18. Neuroimaging confirmed the compete removal of the medial temporal structures.

Eighteen months later her seizures recurred, consisting of a 'thumping' sensation in her head, fear, panic and depersonalisation. She felt that she 'wasn't there', that she was not real and that she was in a dream. She also felt that her surroundings and other people were not real, as though she was 'watching television'. Increasing her anticonvulsant medication stopped the episodes of depersonalisation but not the panic.

DISSOCIATIVE EXPERIENCES SCALE

The DES (Bernstein & Putnam, 1986) is scored by indicating the percentage of time a particular event happens to you. The items of the scale found to be specific to depersonalisation (Simeon *et al.,* 1998) are:

- Some people have the experience of finding themselves in a place and having no idea how they got there.
- Some people have the experience of finding new things among their belongings that they do not remember buying.
- Some people sometimes have the experience of feeling as though they are standing next to themselves or watching themselves do something, and they actually see themselves as if they were looking at another person.
- Some people are told that they sometimes do not recognise friends or family members.
- Some people have the experience of feeling that other people, objects, and the world around them are not real.
- Some people have the experience of feeling that their body does not seem to belong to them.
- Some people find that in one situation they may act so differently compared with another situation that they almost feel as if they were two different people.
- Some people find that they hear voices inside their head that tell them to do things or comment on things that they are doing.

using the Beck Anxiety and Depression Inventories). However, the DES and subscale scores were not able to differentiate between patients with primary and those with secondary depersonalisation. This generates doubt about the validity of this distinction based on psychopathological ratings alone, and reveals that some level of depression and anxiety is observed even in primary cases of depersonalisation (Lambert *et al*., in press).

Neuroimaging and psychophysiological studies

The active inhibition of emotional processing has been suggested in a model underlying some of the clinical features of depersonalisation (Sierra & Berrios, 1998). In brief, this model suggests that the medial prefrontal cortex exerts an inhibitory effect on subcortical limbic structures which are involved in generating normal emotional responses (including the insula) (Phillips *et al*., 1999). Patients will frequently state that although they are aware of the 'normal' emotional responses to particular situations, they do not really 'feel' the emotions and are 'going through the motions' of an emotion. This can be seen as part of the broader complaint of the disorder: that for sufferers the world around them lacks vividness and immediacy. To understand this further, functional neuroimaging and psychophysiological techniques were used in a number of studies to investigate the processing of affective material in those with depersonalisation disorder.

In the first study the skin conductance response of patients with chronic depersonalisation and age- and sex-matched normal controls was recorded when the participants were shown pictures with different emotional valence. These were neutral, pleasant or unpleasant (e.g. pictures of abstract art, a group of puppies playing, a shark attack). The amplitude of event-related responses to the unpleasant pictures was significantly reduced in the depersonalised patients. This suggests that people suffering from depersonalisation have a reduced autonomic response to unpleasant stimuli.

To investigate the possible neural underpinnings of this impairment in autonomic processing, functional neuroimaging experiments were also carried out. We compared depersonalisation disordered patients with both healthy volunteers and clinical control patients (i.e. individuals with obsessive compulsive disorder). We predicted that depersonalised patients would rate aversive scenes as less emotional, would demonstrate reduced activation in emotion-specific regions of the brain (such as the insula), and would demonstrate increased activation in regions implicated in the inhibition of the emotional response (inferior frontal cortex).

This pattern of results would provide some evidence for the symptom of depersonalisation as a coping response to overwhelming anxiety, with the ability to experience a state of high emotional arousal 'switched off' when a potentially distressing or threatening stimulus is encountered. This would lend some empirical support to the theoretical model proposed by Sierra and Berrios (1998).

The results gathered so far support these predictions. Patients suffering from depersonalisation rate the aversive pictures as producing the same intensity of

emotional experience as the neutral ones. The pattern of neural activity for both the healthy participants and the obsessive compulsive disorder patients was similar, with the aversive scenes activating the insula. However, the aversive scenes did not activate this region in the depersonalised patients. Instead a greater neural response (more activity) to the neutral scenes was demonstrated in these areas (Phillips *et al*., 2000).

A final study, currently in progress, aims to build on the finding described above by examining the neural processes underlying emotional memory both in healthy participants and in those with depersonalisation. Emotional memory has become a key area in affective neuroscience, due in part to its relevance in a range of psychiatric disorders including post-traumatic stress disorder, depression, and phobias. We are currently using stimuli specifically designed to examine the recall of emotional and neutral information from affective contexts. A previous pilot study carried out on healthy participants has shown a differential neural response to, on the one hand, direct emotional recall (recalling an emotional word, e.g. 'raped') and, on the other hand, contextual recall (recalling the sentence the word was read in, e.g. 'the sailor raped the girl in the alley') (Medford *et al*., 2000). We are currently using the same technique to examine these neural processes in the context of depersonalisation.

Using the internet as a research tool

Recent psychological investigations carried out on the internet have replicated results from standard laboratory and pen-and-paper studies (for reviews see Senior *et al*., 1999; Senior & Smith, 1999). These studies suggest that the internet is a viable medium for psychological research, although its role in clinical research has only just begun to be explored (see Senior *et al*., 1997; Smith & Senior, 2001).

We wanted to assess the utility of the internet for clinical research into depersonalisation. To do this we compared the scores of several clinical and demographic questionnaires between participants recruited on the depersonalisation bulletin board (see Weblinks) and the unit's outpatient clinic. We predicted that the general demographics and clinical psychometric scores between the two groups would be the same.

All participants were asked to provide details of their history of depersonalisation including duration of illness and medication, along with demographic details (age, sex, years in education). Patients attending the depersonalisation clinic were assessed by one of the unit's psychiatrists. Participants recruited via the internet were contacted and interviewed by telephone. Demographics, details of depersonalisation (duration and severity) and scores of the clinical questionnaires were equivalent, supporting the utility of the internet in the investigation of depersonalisation.

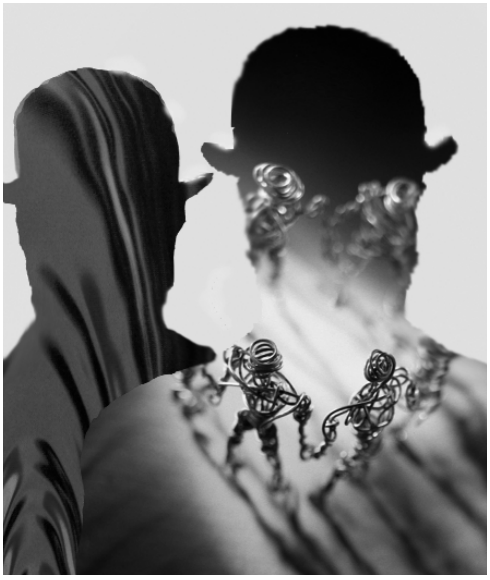
In addition to this, we wanted to determine whether useful clinical information regarding the aetiology, clinical characteristics and response to treatment could be obtained using the internet. This was carried out by posting a series of questions on the depersonalisation bulletin board. In general, the information acquired from the responses to the bulletin board postings was consistent with prior knowledge about the aetiology and phenomenology of depersonalisation.

In summary, the internet users found the depersonalisation website to be easy to access and helpful as a supportive network. The anonymity was an important aspect and many had used it to help establish or confirm their diagnosis and to discuss treatment. Some participants responding from the USA commented that they had not seen a specialist because of lack of funds. The internet would therefore appear to be an effective way of disseminating information as well as recruiting participants for research who would otherwise not have reached medical attention (Lambert *et al*., 2000).

Pharmacological and cognitive-behavioural treatment

Preliminary evidence suggests that lamotrigine (the anticonvulsant medication, used mostly to treat epilepsy) can reduce the intensity of ketamine-induced depersonalisation (Anand *et al*., 2000). Street users of ketamine experience out-of-body sensations that are similar to clinical depersonalisation. They are, however, transient, whilst a majority of the primary depersonalisation clinical cases that we see are permanent.

Based on these findings we tested lamotrigine in patients with depersonalisation disorder. Those from the unit's outpatient clinic with chronic, continuous depersonalisation disorder were asked to take part in a drug trial. Four patients volunteered and with their knowledge were given lamotrigine (dose range 50–250 mgs a day). Subsequently all four patients reported substantial improvements in their symptoms of depersonalisation with no adverse side-effects. The findings of this study, though preliminary, suggest an anti-depersonalisation effect of lamotrigine and warrant double-blind, placebo-controlled studies with lamotrigine as the only agent (Sierra *et al*., 2000).



Some experience a feeling of standing next to themselves or watching themselves do something, and they see themselves as if they were looking at another person

A further aim of the Depersonalisation Research Unit is to develop psychotherapeutic interventions for the treatment of depersonalisation disorder. Cognitive behaviour therapy (CBT) has been found to be highly effective in the treatment of a wide variety of psychological disorders. To our knowledge, no studies of CBT have been conducted with sufferers of depersonalisation, which is largely considered to be resistant to treatment. A pilot study is currently being conducted with patients who have been referred to the unit.

To date, nine patients with primary depersonalisation disorder have been included in this study. All of the participants were suffering from chronic depersonalisation, with a mean onset at age 25 years and a mean duration of 21 years. In the initial phase of treatment we employed a range of interventions that are non-specific to depersonalisation but do heighten activity, motivation and mood levels. Cognitive and behavioural techniques such as activity scheduling, graded exposure to social situations, and challenging negative automatic thoughts

through the use of cognitive diaries were also employed. Any comorbid disorders, such as panic or obsessive compulsive disorder were addressed during this initial phase.

The final phase of therapy involved the use of interventions that were specific to depersonalisation. We have found that in many patients, depersonalisation appears to have developed as a coping mechanism to avoid painful negative emotions arising from a variety of traumatic or aversive situations in childhood or adulthood. The resulting avoidance of emotional arousal has led to a globalised blunting of emotional response. Many sufferers of depersonalisation state that a major goal of treatment for them is to regain the ability to experience emotions, although they may also express concerns about their ability to cope, and fear being 'overwhelmed'. Treatment involves enabling the patient to gain confidence in experiencing negative emotions by the grading of exposure to emotional arousal under controlled conditions.

A second intervention, attention training (see e.g. Papageorgiou & Wells, 1998), specifically addressed alterations in attentional focus apparent in depersonalisation that may act as factors in maintaining the disorder. This is similar to where attention of a person suffering from panic or hypochondriasis has become focused towards symptom monitoring and as such is likely to intensify the problems. Attention training consists of exercises in selective attention, attention switching and divided attention, which help to develop attentional control and encourage focus to external, rather than internal, stimuli. This shift to an external focus of attention may also improve the sense of connection to the external world, which sufferers of depersonalisation often report as impaired.

Preliminary results from this CBT pilot study are promising, with improvements in general functioning and lessening of depersonalisation severity. However, the study is still at an early stage and further patients are being recruited. It is hoped that if CBT proves efficacious in the treatment of depersonalisation in this relatively small sample of patients, a larger randomised, controlled trial may follow in the near future.

What next?

Since the establishment of the Depersonalisation Research Unit, we are beginning to understand more about the many features of this underresearched psychiatric disorder. In particular, we are now in a position to report research findings that can help us to further understand the neurobiological basis of the disorder. We are also able to offer specific treatments for patients suffering from one or more of the symptoms of depersonalisation. However, it is not known how many people suffer from the disorder – finding this out is an important challenge for future studies, since then the heterogeneity of the symptoms can be established.

■ *The Depersonalisation Research Unit team who contributed to this article are Dr Carl Senior, Dr Elaine Hunter, Dr Michelle V. Lambert, Dr Nicholas C. Medford, Dr Mauricio Sierra, Dr Mary L. Phillips and Professor Anthony S. David. The Depersonalisation Research Unit is at the Institute of Psychiatry, De Crespigny Park, 103 Denmark Hill, London SE5 8AZ. Tel: 020 7848 0138; fax: 020 7848 0572.*

References

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

Anand A., Charney, D. S., Oren, D. A., Berman, R. M., Hu, X. ., Cappiello, A., & Krystal, J. H. (2000). Attentuation of the neuropsychiatric effects of ketamine with lamotrigine – Support for the hyperglutamatergic effects of N-methyl-D-aspartate receptor antagonists. *Archives of General Psychiatry, 57*, 270–276.

Ballard, C. G., Mohan, R. N., & Handy, S. (1992). Chronic depersonalisation neurosis au Shorvon: A successful intervention. *British Journal of Psychiatry, 160*, 123–125.

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

Cassano, G. B, Petracca, A., Perugi, G., Toni, C., Tundo, A., & Roth, M. (1989). Derealization and panic attacks: A clinical evaluation on 150 patients with panic disorder/ agoraphobia. *Comprehensive Psychiatry, 30,* 5–12.

Chu, J. A., & Dill, D. L. (1990). Dissociative symptoms in relation to childhood physical and sexual abuse. *American Journal of Psychiatry. 147,* 887–892.

Damasio, A. R. (1996). *Descartes' error*. London: Macmillan.

Jaspers, K. (1963) *General psychopathology*. (M. Hamilton & J. Honig, Trans.). Baltimore, MD: Johns Hopkins University Press. (Original work published 1913)

Lambert, M. V., Senior, C. Fewtrell, W. D., Phillips, M. L., & David, A. S. (in press). Primary and secondary depersonalisation disorder: A psychometric study. *Journal of Affective Disorders*

Lambert, M. V, Senior, C., Phillips, M. L., & David, A. S. (2000). Depersonalisation in cyberspace. *Journal of Nervous & Mental Disease, 188,* 764–771.

Mayer-Gross, W. (1935). On depersonalisation. *British Journal of Medical Psychology, 15*, 103–122.

McGuire, P. K., Cope, H., & Fahy, T. A. (1994). Diversity of psychopathology associated with use of 3,4-methylenedioxymethamphetamine ('Ecstasy'). *British Journal of Psychiatry, 165,* 391–395.

Medford, N., Brierley, B., David, A. S., & Phillips, M. L. (2000). Emotional memory: Content and context – A study using fMRI. *Journal of Cognitive Neuroscience, s12*, 56

Mellor, C. S. (1988). Depersonalisation and self perception. *British Journal of Psychiatry, s153*,. 15–19.

Nemiah, J. C. (1989). Dissociative disorders (hysterical neurosis, dissociative type). In H. Kaplan & B. Sadock (Eds.), *Comprehensive textbook of psychiatry* (5th ed.). (pp. 1028–1044). Baltimore, MD: Williams and Wilkins.

Noyes, R., Hoenk, P., Kuperman, S., & Slymen, D. J. (1977). Depersonalisation in accident victims and psychiatric patients. *Journal of Nervous and Mental Disorders, 164,* 401–407.

Papageorgiou, C., & Wells, A. (1998). Effects of attention training on hypochondriasis: A brief case series. *Psychological Medicine, 28*, 193–200.

Phillips, M.L., Young, A.W., Scott, S., Calder, A.J., Andrew, C., Giampietro, V., Williams, S.C.R., Bullmore, E.T., Brammer, M.J., & Gray, J. (1999). Neural responses to facial and vocal expressions of fear and disgust. *Proceedings of the Royal Society, 265*(B), 1809–1817.

Phillips, M.L., Medford, N.C., Senior, C., Bullmore, E.T., Suckling, J., Brammer, M.J., Andrew, C., Sierra, M., Williams, S.C.R., & David, A.S. (2000). Depersonalisation disorder: Thinking without feeling. *Biological Psychiatry, 47,* s313.

Sedman, G., & Kenna, J. C. (1963). Depersonalisation and mood changes in schizophrenia. *British Journal of Psychiatry, 109,* 669–673.

Sedman, G., & Reed, G. (1963). Depersonalisation phenomena in obsessional personalities and in depression. *British Journal of Psychiatry, 109,* 376–379.

Senior, C., & Smith, M. (1999). The internet... A possible research tool? *The Psychologist, 12*, 442–444.

Senior, C., Phillips, M., & David, A. S. (1997) Psychiatry and the WWW: Some implications. *Psychiatric Bulletin, 21,* 775–778.

Senior, C., Phillips, M. L., Barnes, J., & David, A. S. (1999). An investigation into the perception of dominance from schematic faces: A study using the world-wide web. *Behaviour Research Methods, Instruments, and Computers, 31,* 341–346.

Sierra, M., & Berrios, G. E. (1997). Depersonalisation: A conceptual history. *History of Psychiatry, 8,* 213–229.

Sierra, M., Berrios, G. E. (1998). Depersonalisation: Neurobiological perspectives. *Biological Psychiatry, 44*, 898–908.

Sierra, M., Phillips, M. L., Lambert, M.V., Senior, C., Krystal, J., & David, A. S. (2000). *Lamotrigine in the treatment of depersonalisation disorder: report of four cases*. Manuscript submitted for publication.

Simeon, D., Guralnik, O., Gross, S., Stein, D. J, Schmeidler, J., & Hollander, E. (1998). The detection and measurement of depersonalisation disorder. *Journal of Nervous and Mental Disease, 186*, 536–542.

Smith M, & Senior C. (2001). The internet and clinical psychology: A general review of implications. *Clinical Psychology Review, 21,* 129–136.

Szymanski, H.V. (1981). Prolonged depersonalisation after marijuana use. *American Journal of Psychiatry, 138,* 231–233.

Trueman, D. (1984). Depersonalisation in a non clinical population. *Journal of Psychology, 116,* 107–112.