Available online at www.sciencedirect.com
SCIENCE DIRECT®
Behaviour Research and Therapy 43 (2005) 1121–1130
BEHAVIOUR RESEARCH AND THERAPY
www.elsevier.com/locate/brat

Cognitive-behaviour therapy for depersonalisation disorder: an open study

Elaine C.M. Hunter*, Dawn Baker, Mary L. Phillips, Mauricio Sierra, Anthony S. David

Department of Psychology, Institute of Psychiatry, De Crespigny Park, London SE5 8AF, UK

Received 28 January 2004; received in revised form 30 July 2004; accepted 2 August 2004

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 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 report on an open study where 21 patients with DPD were treated individually with cognitive behavioural therapy (CBT). The therapy involved helping the patients re-interpret their symptoms in a non-threatening way as well as reducing avoidances, safety behaviours and symptom monitoring. Significant improvements in patient-defined measures of DP/DR severity as well as standardised measures of dissociation, depression, anxiety and general functioning were found at post-treatment and six-months follow-up. Moreover, there were significant reductions in clinician ratings on the Present State Examination (Wing, Cooper & Sartorius, 1974), and 29% of participants no longer met criteria for DPD at the end of therapy. These initial results suggest that a CBT approach to DPD may be effective, but further trials with larger sample sizes and more rigorous research methodology are needed to determine the specificity of this approach.

Keywords: Depersonalisation disorder; Dissociation; Cognitive-behavioural therapy

*Corresponding author. Tel.: +44-208-7848-0242; fax: +44-208-7848-5006.
E-mail address: e.hunter@iop.kcl.ac.uk (E.C.M. Hunter).

0005-7967/$ - see front matter © 2004 Elsevier Ltd. All rights reserved.
doi:10.1016/j.brat.2004.08.003

1. Introduction

Depersonalisation (DP) 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 (DR) 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 Behavioural Disorders (ICD-10), World Health Organisation, 1992). Sufferers often 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.

A review of epidemiological surveys of DP and/or DR (Hunter, Sierra, & David, 2004) found that transient experiences of these phenomena are almost universal in the general population. Moreover, community surveys employing standardised diagnostic interviews to measure clinically significant symptoms of DP/DR reported rates of between 1.2% and 1.7% for a one-month prevalence in two UK samples, and a 2.4% current prevalence rate in a North American sample. Current prevalence rates of disorder in samples of consecutive inpatient admissions are reported to be between 1% and 16%, with prevalence rates in clinical samples of specific psychiatric disorders reaching 83% in the case of panic.

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). In this, neither the therapeutic orientation of the treatment nor the methods used to assess outcome was specified. Case studies reporting successful outcomes have employed psychoanalytical techniques (Torch, 1987), psychoanalysis combined with abreaction by intravenous diazepam (Ballard, Mohan, & Handy, 1992), family therapy (Cattell & Cattell, 1974), behavioural methods (Blue, 1979) and imaginal exposure using tapes of grossly exaggerated narratives of previous DP episodes (Sookman & Solyom, 1978). A major limitation in these studies is that they rely on clinical judgement 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 2003. 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.

A recent cognitive-behavioural conceptualisation of DPD (Hunter, Phillips, Chalder, Sierra, & David, 2003) proposes that the chronic condition of DPD may result from catastrophic misinterpretations of the common, but normally transient, symptoms of DP/DR as indicative of serious mental illness and/or brain dysfunction. These misinterpretations serve to exacerbate and perpetuate the symptoms of DP/DR through the development of a range of cognitive biases and behaviours that form a maintenance cycle. This misinterpretation is similar to the process described in models of panic (Clark, 1986) or health anxiety (Warwick & Salkovskis, 1990). However, the cognitions in each condition may be disorder-specific, with panic and health anxiety patients focusing more on catastrophic misinterpretations of the physical symptoms of anxiety, whereas DPD patients may be more concerned about the cognitive symptoms of anxiety and suffer a form of 'mental-health anxiety'.

A cognitive-behavioural approach may prove useful in terms of progressing our understanding, and treatment, of DPD since cognitive behavioural therapy (CBT) models are amenable to empirical testing, and treatment protocols for other anxiety disorders have proved highly effective (Clark & Fairburn, 1997). This paper presents findings from a small-scale study of CBT for DPD, in which cognitive and behavioural interventions were adapted from the principles and techniques of CBT for anxiety disorders.

2. Method

2.1. Participants

All patients were recruited from the Depersonalisation Disorder Clinic, a national specialist research unit that was set up in 1997 at the Institute of Psychiatry, London, UK. Each patient was assessed for DPD, and the presence of co-morbid disorders, by two experienced psychiatrists at the clinic (MLP and MS) trained in using the Present State Examination (PSE): a semi-structured clinical interview to assess psychiatric symptoms (Wing, Cooper, & Sartorius, 1974). Both DP and DR were assessed separately through asking an initial question which was followed by a series of probes to elicit the severity, frequency and duration of symptoms in the past month. The PSE rates DP and DR symptoms as follows: 0 = no symptoms; 1 = moderately intense symptoms lasting for hours at a time during the past month; and 2 = intense symptoms persisting for hours at a time within the last month. Patients were diagnosed with DPD if they scored $\geqslant 2$ out of a possible maximum score of four.

A total of 21 patients took part in the trial (17 men and four women) over a two-year period. There was no selection process in recruitment to the trial; those who were currently in contact with the unit and new referrals were offered CBT. Four participants were given treatment but were excluded from the trial analysis due to, respectively, moving abroad during treatment, receiving a diagnosis of epilepsy, having long-standing DP/DR symptoms that did not meet current PSE criteria, or having DP symptoms that were delusional in quality.

2.1.1. Demographic and diagnostic information for participants

Table 1 summarises the demographic and diagnostic information for the 21 participants, along with their scores on the Dissociative Experiences Scale (DES), Beck Depression Inventory (BDI) and Beck Anxiety Inventory (BAI) at the start of treatment and medication during the course of therapy.

The mean age of the group was 38 years (s.d. = 12, range 23–74), with 17 men (81%). All participants were white British/European. The majority of participants were single ($n = 11$, 52%), with seven (33%) currently married and three (14%) divorced. In terms of employment, 38% ($n = 8$) were of a professional status, 19% ($n = 4$) were employed in associate professional/technical occupations, 14% ($n = 3$) were in clerical, craft or retail occupations, 23% ($n = 5$) were unemployed, and 5% ($n = 1$) were retired. The mean age of onset of the DPD was 22 years (s.d. = 12, range 5–65) and the mean duration of their disorder was 14 years (s.d. = 12, range = 1–42 years). All participants presented with DPD as their primary problem, which, conforming to DSM-IV criteria, could exist without co-morbid symptoms. However, 81%

Table 1
Demographic and diagnostic information for participants

Age	Sex	Partner status	Employment	Age at DPD onset	Co-morbid diagnosis	BDI score[a]	BAI score[a]	Medication
74	F	Divorced	Retired	65	Agoraphobia, depression	42[a]	20[a]	SSRI, lamotrigine and benzodiazapine
34	M	Single	Solicitor	31	None	13	2	Lamotrigine
32	M	Single	Unemployed	19	Panic, depression	23[a]	45[a]	SSRI, lamotrigine and benzodiazapine
37	M	Divorced	Unemployed	23	Generalised anxiety, depression	26[a]	41[a]	SSRI, lamotrigine
47	M	Married	Banking	5	None	4	16	None
37	M	Married	Unemployed	5	None	5	4	SSRI, lamotrigine
55	M	Married	Solicitor	16	Generalised anxiety, depression	10	0	SSRI, lamotrigine and benzodiazapine
30	M	Single	Journalist	17	OCD, social phobia	26[a]	29[a]	Benzodiazapine
46	F	Single	Student	30	Depression, generalised anxiety	35[a]	6	Anti-depressant
27	M	Single	Stockbroker	18	Generalised anxiety, depression	12	33[a]	SSRI, lamotrigine
37	M	Single	Unemployed	29	Generalised anxiety, depression	22[a]	23[a]	SSRI, lamotrigine
45	M	Married	Solicitor	19	None	28[a]	15	SSRI, lamotrigine
23	M	Single	Unemployed	18	Substance-induced anxiety	29[a]	25[a]	SSRI, lamotrigine, benzodiazapine
35	M	Single	Retail	19	Generalised anxiety, panic disorder	28[a]	31[a]	SSRI, lamotrigine
28	M	Single	Care worker	24	Social phobia	32[a]	25[a]	SSRI, lamotrigine
30	M	Single	Proof reader	15	Social phobia, depression	17	27[a]	SSRI, lamotrigine
50	M	Married	Administrator	23	Depression	22[a]	17	SSRI, lamotrigine
38	M	Divorced	Musician	22	Depression	36[a]	30[a]	SSRI, lamotrigine
27	M	Single	Architect	18	Generalised anxiety	19[a]	7	None
28	F	Married	Company Director	19	Generalised anxiety, depression	10	29[a]	None
27	F	Married	Accounts Manager	25	Generalised anxiety, Panic disorder with agoraphobia	29[a]	33[a]	None

[a]Scores on the BDI or BAI of >18 indicate, respectively, moderate to severe depression or anxiety (Source: Center for Cognitive Therapy, Philadelphia).

($n = 17$) had a co-morbid anxiety disorder and/or depression. In terms of clinically significant levels of depression and anxiety at the start of treatment, scores on the BDI and BAI of >18 indicate, respectively, moderate to severe depression or anxiety (Source: Center for Cognitive Therapy, Philadelphia). Using these cut-off scores, respectively 67% ($n = 14$) and 62% ($n = 13$), met criteria for moderate to severe depression or anxiety.

Only four participants (19%) were unmedicated during the trial. The rest were prescribed differing combinations of medications (see Table 1), which included antidepressants (mostly selective serotonin re-uptake inhibitors: SSRIs), benzodiazapines and lamotrigine. The latter is an anticonvulsant that a previous trial conducted at the same unit had found to improve DPD symptoms in 50% of cases, when used as an adjunct to an SSRI (Sierra et al., 2001). In the majority of cases who were taking medication (77%, $n = 13$), these regimes were unchanged throughout the duration of the CBT. However, in three cases the type of antidepressant was changed during the therapy and in one case the patient admitted to taking medication irregularly.

2.2. Measures

The following self-rated questionnaires were completed by all patients at pre-treatment, post-treatment and at six-month follow-up.

2.2.1. Disorder specific measures

Dissociative Experiences Scale (DES) (Bernstein & Putnam, 1986). This is the most widely used measure of current, adult dissociation. Participants rate the percentage of the time they experience 28 different dissociative phenomena to obtain a mean DES score. There is also a subscale specifically for DP/DR, which uses the mean score of six individual items.

Cambridge Repersonalisation Scale—State (CDS-S, Sierra & Berrios, 2000). The Cambridge Depersonalisation Scale (CDS) is a 29-item scale that measures the severity of trait DP/DR symptoms since the onset of the condition and results in a mean score ranging from 0 to 10. A state measure, derived from the CDS, uses a visual analogue scale to measure the intensity of 22 symptoms, as they are currently experienced.

2.2.2. Clinical measures

Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) and *Beck Anxiety Inventory* (BAI) (Beck, Epstein, Brown, & Steer, 1988). These are standardised measures of depression and anxiety, both with 21 items, giving a range of scores from 0 to 63, with total scores of <10 indicating no disorder, 10–18 indicating mild to moderate symptomatology, and >18 indicating moderate to severe depression or anxiety.

2.2.3. General functioning measures

Work and Social Adjustment Scale (WSAS) (Mundt, Marks, Shear, & Greist, 2002). Three items from this scale were employed to assess the extent to which the patient's problem interfered with their work, social activities and their relationships with others, on a scale of 0 (no impairment) to 8 (very severely impaired).

2.3. Treatment

Treatment was conducted by the first (E.C.M.H) and second (D.B) authors. All patients were seen as outpatients, with the majority of sessions in person, but with some sessions conducted by telephone for practical reasons due to the considerable distance some patients lived from London. The mean number of sessions was 13 (s.d. = 6, range = 4–20).

Treatment was implemented in three phases (see Hunter, Phillips, Chalder, Sierra, & David (2003) for a more detailed description of specific interventions). In phase 1, the reduction of distress was prioritised and therapy included psycho-education and normalising of the DPD symptoms, as well as standard CBT interventions for anxiety and depression symptoms. DPD diaries socialised patients to the CBT model by highlighting the role of mood, cognitions and behaviours on symptom fluctuations. In phase 2, DPD symptoms were specifically targeted with individually tailored programmes to reduce (a) avoidance of DPD provoking situations, (b) the use of 'safety behaviours' and (c) symptom monitoring and self-focused attention. In this phase, negative cognitions related to the DPD symptoms were challenged through evidence gathering and behavioural experiments. Finally, phase 3 focused on maintaining progress and relapse prevention strategies.

3. Results

To analyse the outcome of treatment for the group, a repeated-measures ANOVA was conducted, with the mean scores on each measure compared at each time period (i.e. pre-treatment, post-treatment and six-month follow-up). Table 2 summarises the results of this analysis for disorder specific, clinical and case specific measures for all 21 patients.The results in Table 2 show that on the three disorder specific measures there were significant reductions in the

Table 2
Summary of outcome measures for all participants ($n = 21$)

Measure (range of scores)	Pre-treatment mean (s.d.)	Post-treatment mean (s.d.)	Six-month follow-up	F value (df = 2,40)
Disorder Specific Measures				
DES Total scale (%)	22.2 (14.0)[a]	18.7 (10.8)[b]	15.3 (9.8)[c]	11.0***
DES DP/DR sub-scale (%)	39.2 (22.3)[a]	32.3 (19.4)[b]	26.5 (18.2)[b]	6.8**
CDS-state Version (%)	38.8 (21.8)[a]	29.9 (22.0)[b]	26.2 (19.5)[c]	11.0***
Clinical measures				
BDI (0–63)	22.3 (10.5)[a]	14.3 (10.0)[b]	12.8 (9.9)[b]	24.9***
BAI (0–63)	21.8 (12.7)[a]	14.8 (8.8)[b]	14.7 (11.5)[b]	8.6**
General functioning measure				
Work and social adjustment scale (0–8)	4.1 (1.3)[a]	3.6 (1.8)[a]	3.1 (2.0)[b]	7.6**

*$p < .05$; **$p < .01$; ***$p < .001$.
[a,b,c]Means that do not share the same superscript horizontally are significantly different ($p < .05$).

severity of symptoms reported from the start of treatment to the end of treatment. Moreover, at six-month follow-up the total DES score and the CDS showed further reductions that were significantly different from the end of treatment scores. However, on the DP/DR subscale, this improvement at follow-up only neared significance ($p = .06$). In terms of the clinical measures of depression and anxiety, there were significant improvements reported by patients from the start of treatment to the end of treatment, and these improvements were maintained at six-month follow-up. On the measure of general functioning, the difference from pre-treatment to post-treatment only neared significance ($p = .07$), but the improvement continued after treatment and at six-month follow-up was significant.

A delayed baseline analysis was conducted to determine how much, if any, change in symptom severity could have occurred naturally over time. All participants had completed measures (DES, BDI and BAI) on referral to the unit. The mean time difference between the completion of these initial baseline measures and the measures completed at the start of treatment was 9.9 months (s.d. = 6.6, range 2–23 months). Paired *t*-tests carried out on these measures at baseline and start of treatment showed increases on all three measures in the severity of reported symptoms from the initial baseline measure to the start of treatment, although none of these increases were significant (all *t*-values <2).

Changes in PSE ratings were also examined from the start of treatment to end of treatment (see Table 3). At the start of treatment all participants met criteria for DPD (i.e. PSE $\geqslant 2$). At the end of treatment the PSE ratings for the frequency and intensity of symptoms was significantly reduced ($t(20) = 7.6$, $p < .000$), and six participants (29%) no longer met DPD criteria.

Two secondary analyses were carried out. Firstly, the four patients who changed medication regime during therapy were excluded and the above analysis repeated. The pattern of results for the remaining 17 patients again showed a significant improvement in all measures from pre-treatment to follow-up. However, the planned contrasts showed that the differences only became significant at follow-up and not at the end of treatment. Moreover, although all four patients were rated lower on the PSE at the end of treatment, all still met criteria for DPD. Secondly, since there were significant improvements on measures of anxiety (BAI) and depression (BDI) in the group after treatment, the results were re-analysed co-varying for change in BAI and BDI scores from pre-treatment to six-month follow-up, to determine how much of the improvement in dissociation, DP/DR and general functioning could be attributable primarily to improvement in anxiety and depression. This re-analysis found that only the measure of dissociation remained significant (DES total scale, $F(2, 36) = 3.7$, $p = .04$), indicating that the effect of treatment on general level of dissociation was independent of anxiety and depression.

Table 3
PSE ratings pre and post treatment ($n = 21$)

PSE rating	Number of participants pre-treatment	Number of participants post-treatment
0	0	3
1	0	3
2	1	10
3	9	4
4	11	1

4. Discussion

To the authors' knowledge, this study is the first to report on the efficacy of a cognitive-behavioural approach to treating DPD. The findings of this study suggest that CBT for DPD may be effective, since patients reported significant improvements across all outcome measures from pre-treatment levels to post-treatment. These improvements were maintained or increased at six-month follow-up. Clinician ratings using the PSE also showed significant improvements, and 29% of patients no longer met criteria for DPD at the end of treatment. These results are noteworthy given the severity and chronicity of DPD in this sample, with a mean duration of 14 years, and a previous history of drug therapy, often with little alleviation of symptoms. Moreover, in 81% of cases ($n = 17$) the DPD co-existed with moderate to severe levels of depression and/or anxiety. The improvement in symptoms cannot be attributed merely to spontaneous improvements over time, given that participants reported a worsening of all symptoms between referral to the unit and the start of CBT. Nor can the improvement be attributable simply to medication, since although nearly all participants were medicated during the trial, their regimes were unchanged in 77% of cases and all measures remained significant when those with medication changes were excluded from the analysis.

However, it appears that the improvement in outcome measures is largely attributable to improvements in anxiety and depression as a result of the CBT. Although this makes the treatment package described in this study less specific for DPD than originally envisaged, it suggests that CBT may be highly effective in alleviating these symptoms indirectly by the treatment of the co-morbid anxiety and depression. Given the high level of co-morbidity of these disorders with DPD, this is valuable information to clinicians who are likely to have had considerable experience in treating depression and anxiety but may rarely have encountered DPD and lack confidence in their ability to treat this effectively. However, it is also possible that this result derives from a difficulty that patients have in discerning improvement on DP or DR symptoms as distinct from their symptoms of anxiety and depression, in that they are so intrinsically linked. Nevertheless, dissociation more generally was significantly improved independently to some extent, of improvement on anxiety and depression measures.

Despite the significant improvements across a range of measures and the reduction in symptoms when rated on the PSE, the majority of patients (71%) still met criteria for DPD at the end of treatment. However, since the therapy was instigated as a research trial with a maximum limit of 20 sessions decided at the onset of the trial, it is possible that, given the severity and chronicity of the disorder in many of those participating, more sessions may have been needed to reduce symptomatology to levels below clinical cut-offs.

Despite the overall success of this study, it clearly has numerous methodological limitations and as such the degree to which these results can be generalised is, as yet, unclear. The numbers of patients treated are limited and since the study is not randomised, and there is no other therapy as comparison, it is difficult to ascertain whether it is CBT that has been effective, or whether another form of psychological approach would have had a similar effect. Moreover, the patients were self-selecting for treatment and this is likely to have increased their motivation and consequently their perception of their symptoms as improved. The advantage of this is the low drop-out rate. Another methodological weakness of the study is that clinical ratings were not carried out independently of those providing the interventions and may have introduced an

element of bias. This was offset to some extent by the use of self-report measures and those with clear functional anchor points.

Further larger scale research with more rigorous research methodology in the form of a randomised control trial is needed to ascertain the efficacy for CBT in DPD. Nevertheless, the results of this study offer hope for the effectiveness of cognitive-behavioural approaches in the treatment of this distressing and disabling condition.

Acknowledgements

Support for this study came from the Col. W.W. Pilkington Will, Cecil Pilkington and A.P. Pilkington Pilozzo Charitable Trusts.

References

Ackner, B. (1954). Depersonalization—I. aetiology and phenomenology; II. clinical syndromes. *Journal of Mental Science, 100* (I) 838–853, (II) 854–872.

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

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

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.

Blue, F. R. (1979). Use of directive therapy in the treatment of depersonalization neurosis. *Psychological Reports*, *45*, 904–906.

Cattell, J. P., & Cattell, J. S. (1974). Depersonalization: psychological and social perspectives. In Arieti, S. (Ed.), *American Handbook of Psychiatry* (pp. 767–799). New York: Basic Books.

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

Clark, D. M., & Fairburn, C. G. (1997). *Science and practice of cognitive-behaviour therapy*. Oxford: Oxford University Press.

Hunter, E. C. M., Phillips, M. L., Chalder, T., Sierra, M., & David, A. S. (2003). DP 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 DP and DR a systematic review. *Social Psychiatry and Psychiatric Epidemiology*, *39*, 9–18.

Mundt, J. C., Marks, I. M., Shear, M. K., & Greist, J. H. (2002). The work and social adjustment scale: a simple measure of impairment in functioning. *British Journal of Psychiatry*, *180*, 461–464.

Sierra, M., & Berrios, G. E. (2000). The Cambridge DP Scale: a new instrument for the measurement of DP. *Psychiatry Research*, *93*, 153–164.

Sierra, M., Phillips, M. L., Lambert, M. V., Senior, C., David, A. S., & Krystal, J. H. (2001). Lamotrigine in the treatment of depersonalization disorder. *Journal of Clinical Psychiatry*, *62*(10), 826–827.

Sookman, D., & Solyom, L. (1978). Severe depersonalization treated by behavior therapy. *American Journal of Psychiatry*, *135*, 1543–1545.

Torch, E. M. (1987). The psychotherapeutic treatment of depersonalization 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.

Wing, J. K., Cooper, J. E., & Sartorius, N. (1974). *Measurement and Classification of Psychiatric Symptoms*. Cambridge: Cambridge University Press.

World Health Organisation, (1992). *The ICD-10 classification of mental and behavioral disorders: clinical descriptions and diagnostic guidelines*, Geneva.