

A critical analysis of the use of DSM-iv in the understanding of borderline personality disorder.

Joanna L. Ringrose.

Currently, borderline personality disorder (BPD) is by far the most common personality disorder diagnosis, one of ten personality disorders currently classified in diagnostic and statistical manual of mental disorders version four (DSM-iv, Proctor & Shaw, 2004). DSM-iv criteria for diagnosis state that five of the following “symptoms” must be present, these include; 1.) impulsivity or unpredictability in at least two areas that are potentially self-damaging; 2.) intense interpersonal relationships; 3.) inappropriate intense anger or lack of control of anger; 4.) identity disturbance; 5.) affective instability; 6.) problems tolerating being alone / fears of abandonment; 7.) physically self damaging acts / suicidal and self mutilating behaviours; 8.) chronic feelings of emptiness or boredom and lastly, 9.) paranoia or dissociation. This last criteria was not included in the earlier DSM-iii-R version. Sanislow, Grilo, Morey, Bender, Skodol, Gunderson, Shea et al, (2001) note that given that meeting any five of the nine criteria qualifies a person for the BPD diagnosis, there are 151 different combinations of criteria from which it is possible to achieve a BPD diagnosis.

BPD was originally used to describe “patients” who neither fell into the category neurotic, or psychotic but lay somewhere in-between (Mongomery, 1987; Gunderson, 2001). The original diagnosis has undergone many revisions in the DSM. Perhaps “the most notable revision was an attempt to cleave the psychotic end from the category, with the introduction of schizotypal personality disorder into DSM-iii in 1980” (Sanislow, et al (2001). However, despite several attempts to refine criteria, Sanislow et al (2001) report high rates of co-occurring axis i and axis ii disorders in those with BPD and cite this as illustration of the heterogeneity of the disorder. Axis i disorders represent clinical disorders and include schizophrenia, depression, substance use disorder, anxiety and panic as well as many others. Co-morbidity has also been extensively studied by Skodol and colleagues (1995, 1999 a& b, 2002a) who have found patterns of co-morbidity between the axis i and axis ii disorders, specifically BPD and mood, anxiety and substance use disorders.

Diagnostic efficiency is the extent to which diagnostic criteria are able to discriminate between individuals with BPD from those without the disorder. As noted above many theorists have found that the distinction between axis i and axis ii disorders is unclear. However, several further studies have been conducted specifically exploring discrimination within axis ii i.e. just looking at the overlap between BPD and other personality disorders and found varying degrees of clarity. Grilo, McGlashan, Morey, Gunderson, Skodol, Shea et al (2001) revealed “substantial variability” in the efficiency of the nine criteria for BPD. Also, Grilo, Becker, Anez and McGlashan (2004) found that some criteria appear more frequently in patients than others. For example, affective instability appeared most frequently and identity and suicidality or self injury the least frequently. This suggests that some criteria have a stronger predictive power than others and thus supports Sanislow’s (2001) assertion (noted above) that BPD encompasses a heterogeneous client group. Also, in

the DSM-iv BPD criteria, it is implied that each criteria is equivalent in their predictive power, however this research suggests otherwise. Grilo Becker Anez and McGlashan (2004) thus question whether or not the BPD criteria should be weighted beyond their rank order. They also found that although the affective instability criterion was the most frequently occurring symptom it nonetheless lacked specificity. Lastly, factor analysis undertaken by Fossati, Maffeei, Bagnato, Donati, Caterina, and Novella, (1999) found that BPD reflects a “uni-dimensional or singular construct”, thus supporting the DSM-iv diagnosis. However, they stressed that they couldn’t “rule out the possibility of natural subgroups”. The difference between natural subgroups and a heterogeneous concept is open to debate.

There is a vast amount of literature which can be summarised as exploring the “states versus traits” debate. The critical question is, does an entity such as personality exist, or do we possess a series of behavioural dispositions that are activated according to different stimuli? Personality traits refer to prominent features of a person’s behavioural repertoire. There is potentially a very large number of traits, some may include fearfulness, conscientiousness, impulsiveness, meticulousness and selfishness. The point is that traits are considered fairly static and therefore the argument follows that there is something to treat with medication. In contrast, states are dependent upon situations and are thus very changeable and are thus less easily treated with medication. Interactionists argue that behaviour depends on an interaction between the person (their traits) and their environment (the situation in which they find themselves).

In relation to this debate, an analysis of DSM-iii-R criteria undertaken by Sanislow, Grilo and McGlashan (2000) suggests there *may* be homogeneous domains of BPD but some of these domains reflect traits, some reflect symptoms and still others reflect symptomatic behaviours (state dependent). They have thus suggested a three factor model based on their findings. They argue that treatment plans may be used to specify medication to target affective dysregulation (to treat the traits), cognitive behaviour therapy could be used to target behavioural dysregulation (states) and long term psychotherapy may be used to treat the disturbed relatedness factor (states). This reveals some similarities to work conducted by Reich (1987) who noted the confounding influence of state, role (trait) and situational factors. Also on this issue, Oldham (1992) writes there is considerable controversy regarding the use of a categorical diagnostic system such as DSM-iv for the personality disorders because dimensional models of personality are more commonly used in personality studies and by using dimensional models it is possible to accommodate style, traits and pathology. However, he concludes that “it has been difficult to achieve consensus on a single dimensional model”. The same argument being waged at the categorical model. Perhaps his concluding remark reflects the more likely reason why a dimensional model has not been implemented because “the process is not easily applicable to medical systems of classification” (p.42, 1992).

Herkov and Blashfield (1995) argue that serious criticisms still exist regarding the reliability and validity of the axis ii or personality disorders. Aside from the

problems of a categorical taxonomic system and the low predictive value of some criteria as outlined above, they cite several further researchers evidence emphasising unclear boundaries between normal and abnormal personality (Widiger, Frances & Spitzer, 1988). They also cite the findings of Morey and Ochoa's research (1989) where the correspondence between clinical diagnoses, that is those made by clinicians, and criterion diagnoses, those made purely based on the diagnostic criteria, was poor.

In replicating Morey and Ochoa's findings, Herkov and Blashfield (1995) found that although diagnoses fit more than one diagnostic criteria, clinicians typically only used one diagnosis. They argued that multiple diagnoses weren't given because a) clinicians were inexperienced in the use of DSM-iii-R; b) clinicians believed that multiple diagnoses were unhelpful in terms of treatments, and c) some disorders dominate over others and therefore in such circumstances they would only use the dominant label. Herkov and Blashfield (1995) found that BPD was the most frequently chosen personality disorder. They thus conclude that a hierarchy exists among the personality disorders in the way in which clinicians assign diagnoses.

One explanation they use for this finding is that of parsimony. They write "suppose that a patient goes to the internist complaining of headache, high fever and stomach pains. Rather than search for an illness for each of these separate symptoms, the physician will likely attempt to identify a diagnosis which will account for all of these symptoms. In the same manner, when psychiatric patients present with symptoms of several personality disorders, clinicians may choose a personality disorder that will account for most of these symptoms" (p.320, 1995).

To summarise, probably the most unanimous conclusion drawn from research exploring the DSM criteria used to diagnose BPD is that it is a heterogeneous concept. Conclusions drawn from the above strongly suggest that a BPD diagnosis a.) is frequently co-mingled with axis i disorders; b.) is frequently co-mingled with other personality disorders; c.) fails to take account of the reality that "symptoms" may be transient (state versus trait argument); d.) fails to make allowances for the existence of multiple causes which may not be biological in origin (state versus trait argument); and e.) is quite likely to be used by clinicians as a heuristic rule of thumb guiding treatments in a fairly haphazard manner. These problems likely partly reflect the historical routes of the label which seemed to be a dumping ground to catch anyone that didn't fit one of the other diagnostic labels in vogue at the time. The result seems to be like free T shirts given in promotional launches, "one size fits nobody".

However, it seems a lot easier to blame the client for being difficult rather than the diagnostic criteria for being poor. Proctor and Shaw (2004) strongly argue that clients with a BPD diagnosis have frequently been labelled "difficult or challenging". They also note diagnosis has "generally been given to people who are seen as untreatable and (clients) are often told that services can do little to help them" (p.7). Meares and Stevenson (1999) write that patients with a BPD diagnosis "are confronted with the rejection, abuse and neglect which was characteristic of their early lives". They write "one of our patients, for

example, was asked by a psychiatric nurse following a suicide attempt “why didn’t you do it properly?””. Clients with a BPD diagnosis have also been told they are “attention seekers”, “self harmers” “manipulators” and “liars” (see Jo’s story, Asylum, 2004 for example).

In stark contrast to the categorical system of DSM-iv which pathologises clients and assumes there is a physical, biological cause for mental illness, further researchers argue that the “symptoms” of BPD are normal expressions of feeling and ways of coping and surviving following trauma (Shaw and Proctor, 2004). Proctor and Shaw (2004) also argue that the criteria used to diagnose BPD force the clinician to make judgements about when an emotional response becomes “pathological”. Proctor and Shaw (2004) use the example “inappropriate anger” and state that the use of these terms “involves a professional making a judgement about what degree of anger is appropriate from which person and in what circumstances” (p. 7, 2004).

There is an equally vast amount of literature demonstrating the link between childhood trauma, typically abuse but also disorganisation of early attachments and BPD (For examples, see Liotti, Pasquini & the Italian Group, 2000; Joyce, McKenzie, Luty, Mulder, Carter, Sullivan & Cloninger, 2003). Liotti et al (2000) summarise the findings from several studies and note the “similarity between BPD and the path of personality growth laid open by disorganised attachment... both conditions are characterised by un-integrated representations of self with other, by poor control of impulses and emotions, by proneness towards dissociative experiences and by meta-cognitive deficits” (Hesse & Main, 1999; Meins, 1997; Lyons, 1996).

Liotti et al (2000) found that serious losses in the life of the attachment figure and patient’s early traumatic experiences were predictive factors for the development of BPD. Specifically, they noted that loss of a significant care giver, emotional and physical neglect; physical, emotional and sexual abuse; and the witnessing of violence, were all predictive factors for BPD. They found that the probability of developing BPD in the future for a child whose mother had a perinatal serious loss is two and a half times greater compared to a child without that exposure, independently of the risk associated with traumatic experiences.

Joyce, McKenzie, Luty, Mulder, Carter, Sullivan and Cloninger (2003) found that whilst childhood abuse was an important risk factor it is “neither necessary nor sufficient”. Eight per cent of clients with a diagnosis of BPD reported no abuse and 67% with severe abuse did not develop BPD. They found that combining childhood abuse with parental neglect was a more powerful risk factor than abuse alone. They also argue that the child’s temperament (specifically high novelty seeking and high harm avoidance) was a significant factor.

Further theorists advocate a stress-vulnerability model where predisposing genetic risk factors in combination with stressful life experiences (e.g. abuse) are considered (Oldham, 1992). Oldham (1992) writes that endo-phenotypes are being studied, such as impulsive aggression that are thought to reflect

underlying genetic vulnerabilities (Siever et al, 2002). Hansenne et al (2002) also found that reduction in central nervous system serotonin levels have been correlated with impulsive aggression in patients with BPD. And affective instability which has been reported to characterise patients with BPD may be related to cholinergic irregularities (New & Siever, 2002).

In addition, patients with a history of sustained childhood abuse have been shown to demonstrate hyper-responsiveness of the hypothalamic-pituitary-adrenal system and this has been argued to be evident in patients with BPD (Rinne, de Kloet, Wouters, Goekoop-Jaap, DeRijk and Van Den Brink, 2002). Similarly, based on knowledge of previous research on anatomical structures in the brain, it would follow that key structures involved in the perception of emotion, the amygdala and the hippocampus, would show shrinkage in clients with BPD if they had experienced sustained abuse.

Probably the most comprehensive model looking at the aetiology of personality disorders is that produced by Magnavita (2004). Magnavita (2004) writes there is no simple answer, the aetiology is multi-factorial and complex. Any attempts to reduce the cause to one level of abstraction such as biology, trauma, social or interpersonal interactions are likely to be fruitless. He vehemently argues that major models that are blended have by far the best theoretical coherence and explanatory power. His multi-factorial model draws from all the literature hitherto discussed and adds further dimensions. The model includes; genetic predisposition, attachment experience, traumatic events, family constellation and dysfunction, socio-cultural and political forces.

Researchers are increasingly moving away from explaining personality disorders and mental ill health generally, in terms of nature or nurture, genes or environment, biology or culture. These are not mutually exclusive influences on development but moreover there is a complex interplay between them running in multiple directions. A baby's temperament will depend to some degree on his biology. If he has poor temperature control he may easily be cold and therefore fretful. His mother's first experience with her baby may be that he is thus fractious. This will influence how she relates to him and the attachment process. Further environmental influences from other family members and the outside world will shape the baby's personality and this will continue throughout childhood. He will learn very early on what happens when he makes a demand on the environment. Based on his early responses he will learn whether or not the environment is safe and whether or not he is a worthy person.

The criticisms waged at the use of the BPD diagnostic label in particular from clinicians, researchers and clients appear to far outweigh those waged at any other diagnostic label. This is because of the very negative associations which have been made between this label and the patients given it. As aforementioned many have said it is synonymous with "difficult to treat". Hodges writes (2003) "Why are you having trouble with Mr X?". "Because he's borderline". "Why do you consider him borderline?" "Because he is difficult to treat." Kroll (1993) uses the term "garbage can" to describe clients categorised as having post traumatic stress disorder or BPD.

Given that the “symptoms” of BPD may be adaptive responses to very troubling environments how useful is this label? Accepting that there are multiple problems with the DSM-iv diagnosis for BPD and following the current move away from singular explanations for the “aetiology of disorders”, where does this lead us in terms of supporting clients who suffer from these difficulties? I neither advocate pharmacological nor psychological treatments over each other. I would advocate what the client feels works. I believe that in an ideal world clients would choose what they feel they want in the way of support to help them overcome their difficulties and that this support would be available to them. However, the reality is that clients are unlikely to be given much of a choice in respect to their treatment, if there is treatment available at all. Perhaps the best we can hope for is that clients given the unfortunate diagnosis of BPD may be given the same respect as any other member of society who belongs to a minority “out” group and not have to bare the brunt of prejudicial attitudes.

I conclude with an extract taken from Herman (1992) where Judith Herman, a psychiatrist, describes a diagnosis of personality disorder as “little more than a sophisticated insult”.

“They say that I am personality disordered, yet another label telling me what I am. Even worse they say its not amenable to treatment, as if its not me or if it is me then I am not a person. I walk out of the office, I was in there for seven and a half minutes: amazing that a whole life can be pronounced on in seven and a half minutes. I feel like a worthless waste of space and time, why would anyone waste their time with me”.

Tyler, (Herman 1992, p.123)

References

Asylum the magazine for democratic psychiatry, 14,3, 3-27.

Fossati, A., Maffeei, C., Bagnato, M., Donati, D., Caterina, N., & Novella, L. (1999). Latent structure analysis of DSM-IV borderline personality disorder criteria. *Comprehensive Psychiatry* 40, 72-79.

Grilo, C.M., Becker, D.F., Anez, L.M. & McGlashan, T.H. (2004) Diagnostic efficiency of DSM-iv criteria for borderline personality disorder: An evaluation of Hispanic men and women with substance use disorders. *Journal of Consulting and Clinical Psychology*, 72,1,126-131.

Grilo, C.M., McGlashan, T.H. Morey, L.C., Gunderson, J.G., Skodol, A.E., Shea, M.T. et al (2001) Internal consistency, intercriterion overlap, and diagnostic efficiency of criteria sets for DSM-IV personality disorders. *Acta Psychiatrica Scandinavica*, 104, 264-272.

Hansenne, M., Pitchot, W., Pinto, E., et al (2002) 5-HT1A dysfunction in borderline personality disorder. *Psychological Medicine*, 32, (5) 935-941. In Oldham, J.M. *Borderline personality disorder: An overview*. (2004) *Psychiatric Times*, 42-46.

Herkov, M.,J. & Blashfield, R.K. (1995) Clinician diagnoses of personality disorders: Evidence of a hierarchical structure. *Journal of Personality Assessment*, 65(2), 313-321.

Herman (1992) *Trauma and recovery*, Harper Collins: London, p.123)

Hesse, E. & Main, M. (1999) Second generation effects of unresolved trauma in non-maltreating parents: Dissociated frightened and threatening parental behaviour. *Psychoanal Enquiry*, 19, 481-450. In Liotti, G., Pasquini, P. & The Italian Group for the study of dissociation. (2000) *Predictive factors for borderline personality disorder: Patients' early traumatic experiences and losses suffered by the attachment figure*. *Acta Psychiatrica Scandinavia*, 102, 282-289.

Hodges, S. (1993) Borderline personality disorder and post-traumatic stress disorder: time for integration? *Journal of Counselling and development: JCD Alexandria*, 81,4, 409-417.

Joyce, P.R., McKenzie, J.M., Luty, S.E., Mulder, R.t., Carter, J.D., Sullivan, P.F. & Cloninger, C.R. (2003) Temperament, childhood environment and psychopathology as risk factors for avoidant and borderline personality disorder. *Australia and New Zealand Journal of Psychiatry*, 37, 756-764.

Kroll, J. (2003). *PTSD A/borderline in therapy: Finding the balance*. New York: Norton.

Lyons, R. K. (1996) Attachment relationships among children with aggressive behaviour problems: The role of disorganised early attachment patterns. *Journal of Consulting and Clinical Psychology*, 64,64-73. In Liotti, G., Pasquini, P. & The Italian Group for the study of dissociation. (2000) *Predictive factors for borderline personality disorder: Patients' early traumatic experiences and losses suffered by the attachment figure. Acta Psychiatrica Scandinavia*, 102, 282-289.

Liotti, G., Pasquini, P. & The Italian Group for the study of dissociation. (2000) *Predictive factors for borderline personality disorder: Patients' early traumatic experiences and losses suffered by the attachment figure. Acta Psychiatrica Scandinavia*, 102, 282-289.

Magnavita, J. (Ed) (2004) *Handbook of personality disorder*. John Wiley: New York.

Meares, R., Stevenson, J. & Comerford, A. (1999) Psychotherapy with Borderline patients: A comparison between treated and untreated cohorts. *Australian and New Zealand Journal of Psychiatry*, 33, 467-472.

Meins, E. (1997) Security of attachment and the social development of cognition. *Hove: Psychology Press*. In Liotti, G., Pasquini, P. & The Italian Group for the study of dissociation. (2000) *Predictive factors for borderline personality disorder: Patients' early traumatic experiences and losses suffered by the attachment figure. Acta Psychiatrica Scandinavia*, 102, 282-289.

Montgomery, S.A. (1987) The psychopharmacology of borderline personality disorders. *Acta Psychiatrica Belgique*, 87, 260-266.

Morey, L.C. & Ochoa, E.S. (1989) An investigation of adherence to diagnostic criteria: Clinical diagnosis of the DSM-iii personality disorders. *Journal of Personality Disorders*, 3, 180-192.

New, A.S. & Siever, L.J. (2002) Neurobiology and genetics of borderline personality disorder. *Psychiatric Annals*, 32 (6) 329-336. In Oldham, J.M. *Borderline personality disorder: An overview*. (2004) *Psychiatric Times*, 42-46.

Oldham, J.M. *Borderline personality disorder: An overview*. (2004) *Psychiatric Times*, 42-46.

Proctor, G. & Shaw, C. (2004) Borderline personality disorder under the microscope. *Asylum the magazine for democratic psychiatry*, 14,3, 6-8.

Reich, J.H. (1987) Instruments measuring DSM-iii and DSM-iii-R personality disorders. *Journal of Personality Disorders*, 1, 220,240.

Rinne, T., de Kloet, R.E., Wouters, L., Goekoop-Jaap, G., DeRijk, R.H. & Van Den Brink, W. (2002) Hyperresponsiveness of the hypothalamic-pituitary-adrenal axis to combined dexamethasone/corticotropin-releasing hormone

challenge in female borderline personality disorder subjects with a history of sustained abuse. *Biological Psychiatry*, 52, (11), 1102-1112.

Shaw, C. & Proctor (2004) Women at the margins: A feminist critique of borderline personality disorder. *Asylum the magazine for democratic psychiatry*, 14,3, 8-10.

Sanislow, C.A., Grilo, C.M. McGlashan, T.H. (2000) Factor analysis of the DSM-iii-R Borderline personality disorder criteria in psychiatric in-patients. *Comprehensive Psychiatry*, 40, 72-79.

Sanislow, C.A., Grilo, C.M. Morey, L.C., Bender, D.S., Skodol, A.E., Gunderson, J.G., Shea, M.T., Stout, R.L., Zanarini, M.C. & McGlashan, T.H. (2001) Confirmatory factor analysis of the DSM-iv criteria for borderline personality disorder: findings from the collaborative longitudinal personality disorder study.

Siever, L.J., Torgensen, S. & Gunderson, J.G. et al (2002) The borderline diagnosis III: Identifying endophenotypes for genetic studies. *Biological Psychiatry*, 51(12) 964-968.

Skodol, A., Oldham, J.M. & Hyler, S.E. et al (1995) Patterns of anxiety and personality disorder co-morbidity. *Journal of Psychiatry Research*, 29 (5) 361-374. In Oldham, J.M. *Borderline personality disorder: An overview*. (2004) *Psychiatric Times*, 42-46.

Skodol, A., Oldham, J.M., & Gallaher, P.E. (1999a) Axis II Co-morbidity of substance use disorders among patients referred for treatment for personality disorders. *American Journal of Psychiatry*, 156, 733-738.

Skodol, A., Gunderson, J., Pfohl, B., Widiger, T., Livesey, W., & Siever, L. (2002a) The borderline diagnosis: Psychopathology, co-morbidity and personality structure. *Biological Psychiatry*, 1, 936-950.

Skodol, A., Stout, R.L. & McGlashan, T.H. et al (1999b) Co-occurrence of mood and personality disorders: A report from the collaborative longitudinal personality disorders study (CLRS). *Depression Anxiety*, 10,(4) 175-182. In Oldham, J.M. *Borderline personality disorder: An overview*. (2004) *Psychiatric Times*, 42-46.

Widiger, T., Frances, A. & Spitzer, R. (1988). The DSM-iii-R personality disorders: An Overview. *American Journal of Psychiatry*, 145, 786-795.