

MENTAL HEALTH IN NEW ZEALAND FROM A PUBLIC HEALTH PERSPECTIVE

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MANATU HAUORA

CHAPTER 20: PERSONALITY DISORDERS

ROGER MULDER

The legitimate place for personality disorders (PDs) in the field of mental health is controversial. Some argue that it has no place and that personality disorders consist of little more than a list of judgemental and pejorative adjectives given to patients who do not fit into standard diagnostic categories or who are unresponsive to treatment. Such critics claim that the classification of PDs is based on clinical impression and anecdotal evidence rather than any systematic research. The label of a personality disorder may stigmatise patients and make them less likely to receive adequate treatment, or be used as a reason to refuse treatment. It is thus little more than a moral judgement masquerading as a clinical diagnosis (Blackburn 1988).

While there is considerable truth to the above statements, it has also long been apparent that there are patients who have pervasive and sustained maladaptive behaviours that result in serious disability. They often present, or are brought by others, to medical and social services (Mulder, Wells et al 1994). Many have other psychiatric illnesses whose management is made more difficult by the presence of their inflexible and maladaptive behaviours. This large and severely impaired group of patients have a significant impact on the resources of mental health services, and cannot be ignored.

NORMAL AND ABNORMAL PERSONALITY

As it is commonly understood, personality is what makes one individual different from another. Although the concept is well established, it is extremely difficult to define. Most definitions include characteristic behaviours, attitudes, relationships with others and self-concepts. These definitions share some features in that the characteristics are considered to be stable, manifest in different environments, recognisable to others and present since adolescence or early adulthood.

Defining *abnormal* personality presents similar difficulties. Such a definition should encompass attitudes, behaviours and impact on others as well as subjective distress. The definition also needs to take account of the individual's cultural and social expectations. *DSM-IV* defines a personality disorder as an enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual's culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment (APA 1994). The definition implies a judgement that certain behaviours are deviant. At this point there are no reliable objective criteria on which to make this judgement, so that what constitutes 'marked deviation' is based on clinical deduction.

CLASSIFICATION

Attempts to classify disorders of personality have a long history. Most share the concept of a person without overt psychiatric symptoms, but who differs fundamentally from other people because of an innate inability to exhibit normal social adjustment and to maintain normal social relationships, and who also exhibits defects in personality development. In other words, abnormal behaviour in the presence of otherwise normal reasoning processes (Tyrer et al 1993).

Until this century, most classifications focused on the ‘psychopath’: a specific category of people committed to antisocial and immoral behaviour as a consequence of personal deficiencies. Since the 1920s, however, there has been an increasing interest in a wider range of persistent maladaptive behaviours including behaviour that is fearful, odd, suspicious and self-destructive.

The advent of *DSM-III* in 1980 brought about two fundamental changes in personality disorder classification. The first was the introduction of operational criteria, while the second was giving personality disorders their own axis (Axis II). The criteria have the merit of clear definition and more reliable diagnosis but also give a spurious impression of accuracy (Tyrer et al 1993). In fact there was very little empirical data on which to base the criteria, which like the categories themselves, are a mixture of theory, opinion and historical precedent. Having personality disorders on a separate axis has the advantage of allowing individuals to be classified by mental state and personality state, rather than having to decide which is predominant, but it also blurs the fact that personality disorders can strongly influence mental state and vice versa. Nevertheless, the *DSM-III* (and now *DSM-IV*) classification system has resulted in a major increase in research and interest in the personality disorders (Gorton and Akhtar 1990).

DSM-IV (APA 1994) has standardised all personality disorder subtypes as polythetic categories (ie, a certain number of variable criteria from a given set are needed for the diagnosis to be applied). Each category has seven to nine diagnostic criteria of which three to five are needed to make the diagnosis of personality disorder. The number of categories and the criteria have changed significantly in each edition of *DSM*. There are currently 10 personality disorder categories, each of which is briefly defined in Table 20.1. *DSM-IV* also groups the personality disorders into three clusters based on descriptive similarities. Cluster A incorporates paranoid, schizoid and schizotypal PDs. Individuals with these disorders often appear odd or eccentric with limited interpersonal skills. Cluster B consist of antisocial, borderline, histrionic and narcissistic PDs. Individuals with these disorders often seem to be dramatic, emotional and erratic. Cluster C consists of avoidant, dependent and obsessive-compulsive PDs. Individuals with these disorders tend to be introverted and appear anxious and fearful. These clusters have some value clinically but have not been consistently validated.

Table 20.1: DSM-IV personality disorder categories

<i>Disorder</i>	<i>Definition</i>
Paranoid personality disorder	A pattern of distrust and suspiciousness such that the motives of other people are interpreted as malevolent
Schizoid personality disorder	A pattern of detachment from social relationships and a restricted range of emotional expression
Schizotypal personality disorder	A pattern of acute discomfort in close relationships, cognitive or perceptual distortions, and eccentricities of behaviour
Antisocial personality disorder	A pattern of disregard for, and violation of, the rights of others
Borderline personality disorder	A pattern of instability in interpersonal relationships, self-image, and affects, and marked impulsivity
Histrionic personality disorder	A pattern of excessive emotionality and attention seeking
Narcissistic personality disorder	A pattern of grandiosity, need for admiration, and lack of empathy
Avoidant personality disorder	A pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation
Dependent personality disorder	A pattern of submissive and clinging behaviour related to an excessive need to be taken care of
Obsessive-compulsive personality disorder	A pattern of preoccupation with orderliness, perfectionism, and control
Personality disorder not otherwise specified	

EPIDEMIOLOGY

Accurate information about the prevalence of personality disorders is sparse, except for antisocial personality disorder (ASPD). Several major epidemiological studies have employed standardised diagnostic criteria for ASPD. The Epidemiologic Catchment Area (ECA) study in three United States sites showed lifetime prevalence rates for antisocial personality disorder of 2.6 percent, the prevalence varying in each site from 2.1 to 3.4 percent (Robins and Regier 1991). Lifetime prevalence for males was significantly higher (4.5 percent) than for females (0.8 percent). The Christchurch, New Zealand, study employed similar methodology and reported a lifetime rate of 3.1 percent (males 4.2 percent, females 0.5 percent) (Wells et al 1989). A lifetime prevalence of 3.7 percent was found for ASPD in Edmonton, Canada (Bland et al 1988). Rates were much lower in Taiwan, at around 0.14 percent (Yeh and Hwu 1992).

Rates for other personality disorders vary depending on how restrictive the diagnostic criteria are. The Midtown Manhattan Study (Srole et al 1962) estimated the prevalence of all personality disorders as 10 percent. Casey and Tyrer (1986) found personality disorders in 11 percent of a random sample of

200 subjects as adjudged by the Personality Assessment Scale (PAS). Leighton and colleagues (1963) found a prevalence of nearly 20 percent in men, including the category of 'sociopaths'. The best current estimate is from a Baltimore sample of 810 adults examined using a semi-structured interview (Samuels et al 1994). The prevalence of personality disorders in these adults was 5.9 percent (9.3 percent when provisional cases were included). There are no prevalence data for non-ASPD personality disorders using a New Zealand sample. There is no evidence of different rates among Māori or Pacific populations.

A significantly higher prevalence of personality disorders has been found in urban communities and a decreasing rate with increasing age (Amer and Molinari 1994). The prevalence is much higher in any environment in which the socially impaired are concentrated, such as slums, boarding-houses and prisons. Unpublished data (Brinded et al 1995) suggest rates of personality disorders in New Zealand prisons are very high: overall rates of ASPD were 70 percent for males, 39 percent for females. This study also reported ASPD rates among Māori prisoners: they were lower for males (56 percent) but higher for females (60 percent), compared with non-Māori prisoners. A significant proportion of the prisoners also had features of schizoid PD (18 percent), and avoidant/dependent PD (24 percent). Rates of personality disorders among psychiatric patients are also high. Mulder, Joyce and colleagues (1994), using a structured interview in a Christchurch sample of patients with major depression, reported a rate of personality disorders of 50 percent; the most common were borderline, avoidant, and paranoid PDs.

Little is known about epidemiological trends in personality disorders. Rates of admission for individuals with personality disorders in New Zealand appear to be dropping (Mulder 1991), but this probably reflects admission policy rather than prevalence trends. There is convincing evidence that delinquent behaviour among youth, however it is measured, has increased markedly since the time of the Second World War in all Western countries (Smith 1995). Little is known about trends in other behaviours.

COURSE AND PROGNOSIS

Since by definition a PD is a persistent and enduring maladaptive pattern of behaviour, its course should be reasonably stable. The adult behaviours are usually present, in some form, in childhood and adolescence. There is a particularly impressive body of evidence from longitudinal studies, including the Christchurch and Dunedin cohort studies, demonstrating the continuity between antisocial behaviour in childhood, adolescence and adulthood (Smith 1995). There is also considerable evidence that anxious and shy behaviours begin in early childhood and display high degrees of stability into adulthood (Kagan 1994). This evidence strongly suggests that any preventive strategies would need to be initiated early in an individual's life.

The course of PDs in adulthood is poorly studied. These patients are often difficult to follow up and the nature of their symptoms makes clinicians reluctant to follow them. There is emerging evidence that some behaviours – particularly those labelled antisocial and borderline – improve significantly over time. Measured criminal behaviour reduces after the age of 35 (Smith 1995) and psychosocial measures of outcome in patients with borderline PD improve in as little as 5–10 years (Stone et al 1987). Other PDs – notably obsessive-compulsive, paranoid and schizoid – tend to persist into old age.

RISK FACTORS

Little is known about the cause of specific personality disorders. Most recent work supports the common-sense conclusion that both biology and environment make significant contributions.

BIOLOGICAL

The evidence that biological variables contribute to personality-disordered behaviours comes from three areas of research. The first is genetic studies. Measurable aspects of normal personality are at least moderately heritable, with most studies suggesting that genes account for approximately half the variation found in normally distributed behavioural traits (Holden 1987). Because of problems in classification, it is more difficult to be certain about personality disorders, but a recent survey of the literature reported that there is consistent evidence of a genetic contribution to several categories of abnormal personality (McGuffin and Thapar 1993). The pattern of inheritance is probably polygenic.

The second area of research is studies on temperament. Temperament may be defined as simple, non-motivational, non-cognitive behavioural characteristics. Temperamental variables (eg, sociability, activity, impulsivity) can be reliably identified by the time a child is two. There is now overwhelming evidence that children differ markedly from one another in a host of ways, that these differences are reasonably stable and persistent and that they matter in terms of implications for later development, including abnormal personality traits (Rutter 1987). There is also considerable evidence that these manifest differences are linked with neurobiological variables including autonomic reactivity, cortisol levels and neurotransmitters (Mulder 1992).

The third area is neurochemistry. Certain behaviours appear to be related to abnormal neurotransmitter states. The most consistent finding is that low levels of central nervous system (CNS) serotonin are linked with impulsive, violent and disinhibited behaviour in both man and animals (Dolan 1994). Other research links disturbances in CNS dopamine with conduct disorder in boys (Galvin et al 1991).

ENVIRONMENTAL

Although clinical experience and personality theories indicate that environment plays a major role in the development of personality disorders, there is remarkably little evidence to support such a view. Vaillant, for example, found very little correlation between a bleak early childhood and the adult use of immature defence mechanisms commonly associated with personality disorders (Vaillant 1977).

Much of the reason for this apparent disparity reflects methods of investigation. There has been a long-standing search for specific environmental factors, with the underlying assumption that such factors would impact significantly on most, if not all, individuals. There is now growing evidence that the strongest environmental effects on personality development are not common events but those that have significant meaning for the individual involved. In other words, the same environmental factor may seem trivial to one individual, but highly significant to another – depending on temperament and past experiences. In fact, environmental factors that are shared by children in the same family have little impact on personality development; the strongest environmental effects are those that impinge differently on different children within the family (Plomin and Daniels 1987). Aligned with this has been increasing recognition of the importance of vulnerability and resistance: so-called resilient children may be brought up in adverse environmental circumstances with apparent minimal effect, while other children in a similar (or even less adverse) environment may develop maladaptive behaviour traits.

To confuse matters further, there is also increasing recognition of the role that temperament and other factors have in creating environmental effects. Children, to some degree, create their own environmental events. For example, a temperamentally stable, sociable child will elicit more positive behaviour from parents, siblings and teachers than an anxious or irritable child. These interactions may amplify small differences in genetic endowment.

PREVENTION

The scale of the problems caused by individuals with PDs suggests that if effective prevention strategies were available their use would be justified. As we have seen, PDs are common and usually chronic. They significantly disable the person concerned and frequently affect those around them. Although the research is limited, it suggests that people with PDs suffer increased rates of separation, divorce, child custody proceedings, crime, parasuicide and suicide and have frequent contact with health and social services. The most extensive outcome study of individuals with PDs was reported by Valliant (1977), who showed that those judged to have a PD in late adolescence remained severely impaired with chronically unsuccessful work histories and impaired interpersonal relationships throughout the 33-year follow-up.

Although prevention is justified, there are a number of problems that limit its application to people with PDs. The first is the nature of the PD categories themselves. As noted, with the exception of ASPD and possibly borderline PD and schizotypal PD, the diagnostic categories are not validated and have almost no accurate epidemiological or natural history data. The problem of whether categorical or dimensional models are more valid is still not resolved. The behaviours described by PDs appear to be distributed dimensionally, yet much research is based on the rather arbitrary categories defined in *DSM-IV* or *ICD-10*. Using a dimensional construct might be more helpful in studying broad preventive strategies that could extend to 'subthreshold' cases of PDs. Unfortunately, there is currently no consensus over which dimensional model to use. The second problem is that the cause of PDs is unknown. It is likely that there are multiple factors that contribute to personality-disordered behaviour. The third problem is that, with the exception of delinquent behaviour, there are few studies that have looked at the efficacy of preventive interventions in children or adolescents. There are even fewer studies that have followed up such interventions to see whether they have an effect on adult behaviours.

The following discussion is therefore largely a speculation on possible intervention strategies, and a chronicle of the lack of accurate and useful information in the area of PDs. Whether the possible interventions are even feasible within the framework of public health strategies is doubtful.

BIOLOGICAL FACTORS

The first point is obvious but worth stating. While genetic factors are clearly important risk factors for at least some PDs, there is little we can do to change the gene pool. We do not understand enough about the hereditary influences underlying personality to suggest any possible strategies (Stone 1995). Even if there were possible strategies, there are enormous ethical issues to be considered.

Physical health does not appear to be closely linked with PDs. Improvements in physical health have not led to any apparent reduction in personality-disordered behaviour: criminal behaviour, for example, has significantly increased since the Second World War – a period of improving physical health (Smith 1995). It is possible that PDs may be more common in persons whose mothers, while pregnant, abused alcohol and other substances that are dangerous to a foetus (Stone 1995). Efforts to improve methods of educating expectant mothers and caring for their nutritional and emotional needs might therefore be useful.

COMORBID PSYCHIATRIC DISORDERS

One possible strategy to reduce PDs is early, prompt and effective treatment of childhood psychological symptoms and disorders that affect personality development. Children with unstable mood often present with symptoms reminiscent of PDs – aggressive behaviour alternating with social withdrawal and avoidance. Effective treatment of their mood might reduce the development of comorbid PDs to which they appear to be prone (Goodwin and Jamison 1990). Attention-deficit/hyperactivity disorder can often be ameliorated by appropriate treatment during childhood and adolescence (Shaywitz and Shaywitz 1990), and this may reduce irritable and antisocial behaviour. Similarly, dyslexia and specific learning difficulties may be misinterpreted by parents as laziness or oppositional behaviour, resulting in the children receiving severe criticism. This, in turn, may undermine the child's self-confidence and so lead to various personality disturbances, particularly those of Cluster C (Stone 1995). Untreated childhood anxiety disorders such as separation anxiety and social phobia may lead to later avoidant and dependent PDs. Alcohol and drug abuse is strongly associated with the Cluster B PDs. The association is complex and linked to the social setting, cultural beliefs and expectations about the effects of the substance (Fagan 1990). However, there is good evidence that changes in total consumption of alcohol are associated with changes in the level of recorded violent behaviour (Smith 1995).

ENVIRONMENTAL FACTORS

The role of environmental factors in producing PDs is important but their association is complex and poorly understood. There is some agreement that environmental influences have their influence during personality development (ie, childhood and adolescence). Any intervention would thus need to be initiated early in the individual's life and, given the nature of personality development, would probably need to be comprehensive and protracted.

There is no evidence that any specific intervention targeted at PDs has been effective. The interventions suggested are generally universal prevention measures (eg, good parenting, preventing childhood abuse, allowing children to develop skills), which may be regarded as desirable for everyone. Such interventions range from macrosocial changes to those targeted at individuals. They are targeted at improving psychosocial health in childhood and adolescence with the hope that these measures will result in better adjustment and less personality-disordered behaviour in adulthood.

There is an excellent review of risk factors and possible interventions in childhood and adolescent disorders in Chapter 5 of this report by Fergusson and colleagues. The following discussion summarises possible applications of such interventions to PD prevention.

Increased social equity and social opportunity are by themselves unlikely to lead to large changes in the vulnerability of young people to psychosocial disorders, and by implication PDs (Rutter and Smith 1995; Fergusson et al 1997). The only well-studied behavioural disorder, criminal activity, has increased in frequency while living conditions were improving throughout the Western world (Smith 1995). Similarly, unemployment rates were low in the 1950s and 1960s when behavioural disorders appeared to be becoming more frequent. Although unemployment is associated with juvenile offending internationally (eg, West 1967), and the Christchurch cohort study showed that unemployment was associated with conduct disorder, the elevated risk appears to be caused by personal and social factors that were present prior to the onset of unemployment rather than the unemployment itself (Fergusson et al 1997). Changes in employment levels will probably only result in minor improvements in personality-disordered behaviour.

Community-based studies aimed at reducing antisocial behaviour and delinquency have produced contradictory results. Those using behaviour modification components and focusing on the immediate social environment (as opposed to the broader community) may have marginally more success (Fergusson et al 1997).

Because parenting factors and childhood environment consistently emerge as risk factors for PDs, family-based interventions have been advocated. There is little doubt that some patterns of parenting are obviously deleterious. Any significant and prolonged abuse (whether physical, sexual or emotional) will always be harmful and may lead to PDs in adulthood. In one study, extreme family violence was found to increase the chances of becoming delinquent by a factor of four (Lewis et al 1989). Studies of individuals with borderline PD have consistently reported high rates of childhood sexual abuse. Individuals with PDs report poorer maternal and paternal care (Mulder, Joyce et al 1994). Similarly, cruelty or gross neglect will result in high levels of psychopathology – including, but certainly not confined to, PDs. Any way of preventing such patterns of parenting would clearly be worthwhile and would result in the reduction of a great deal of suffering and distress. However it would not be a specific preventive intervention for PDs but rather a universal prevention measure. Evidence for the efficacy of intervening at a family level is sparse, but in general the results have been moderately encouraging (see Chapters 5 and 19). Such interventions need to be targeted at high-risk families, to be initiated early, and to be intensive and prolonged (Yoshikawa 1994).

There is much more controversy about targeting less obviously inappropriate parental behaviours. For example, it is possible (although unproven) that overindulgent parents with grandiose ambitions for their children might result in higher rates of narcissistic PD in these children. It has been suggested that controlling and rigid parents are associated with the development of obsessive-compulsive PD in their children (Stone 1995), and that clinging, dependent parents' children may have more dependent behaviours as an adult. Should mental health measures extend to advising on parental style, particularly when the link between such styles and PDs is completely unproven and there is no evidence of any interventions that substantially change parental style?

SECONDARY PREVENTION

Since personality disorders by definition have an early onset and are an enduring disorder, they are a logical target for secondary prevention. The aim of secondary prevention programmes is to direct early and prompt treatment of a disorder with the goal of shortening its duration and hence its prevalence. Unfortunately, in the case of personality disorders, there are no proven effective treatments for any of the individual disorders.

The preliminary findings of some studies suggest that it may be possible to reduce the morbidity associated with personality disorders. However, methodological flaws and weaknesses interfere with interpreting data and with comparing data across studies (Koaner and Lineham 1996). Lineham and colleagues (1994), in a randomised controlled outcome trial, have reported that patients who received one year of dialectical behaviour therapy (a particular type of behaviour therapy) had better overall social adjustment than those receiving standard treatment. Stein's (1992) review of drug treatment of personality disorders suggested that while drugs will not cure personality disorder, drug treatment may reduce some of the maladaptive behaviour associated with such disorders. It is possible that selective serotonin reuptake inhibitors and mood stabilisers may, in selected cases, have a role to play (Mulder 1996). There are no New Zealand studies specifically looking at treatment of personality disorders.

CONCLUSIONS

Personality disorders refer to a disparate group of disorders that are currently poorly validated and difficult to diagnose reliably. The relationship of personality disorder to normal personality is not clear and how to measure the maladaptive traits that distinguish personality disorders from normal personality functioning is unknown. The relationship between Axis I and Axis II disorders is disputed. Except for ASPD, epidemiological data and accurate knowledge about the natural history of these disorders is inadequate. The specific aetiology of personality disorders is unknown although it is probable that genetic factors and parenting factors are important. Most major issues concerning assessment and treatment remain unresolved. Models for treatment and strategies for treatment development are only just beginning to be discussed. All of these problems suggest that it is premature to seriously advocate preventive strategies for personality disorders. A number of intervening steps are needed.

The first step is to create reliable and valid diagnostic categories and to study their epidemiology and natural history. The second is to attempt to determine the key aetiological factors in some of the personality-disorder categories. The third is to test if it is possible to develop effective treatment and perform outcome research to see if the treatment results in persistent long-term changes. The fourth is to develop a body of methodology for evaluation of prevention activities. This should include experimental research designs involving long-term follow-up, state-of-the-art analytic techniques, methods of evaluation of cost-effectiveness and the logic of population-attributable risk (Eaton and Harrison 1996).

At present, efforts are best directed at universal preventive measures. The most important would seem to be reducing abuse of children, and early assessment and effective treatment of childhood and adolescent psychiatric and learning disorders. The limited data suggest that such interventions are best targeted at at-risk families, peers and individuals rather than the general community. Careful consideration and, if possible, randomised controlled outcome studies should accompany the preventive measures. Without them we will possibly waste our resources or, even worse, harm those we seek to help.

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