

## Section 2

---

# The nature of mental illness



## Chapter 4

---

# Mental illness is indeed a myth

Hanna Pickard

### Abstract

This chapter offers a novel defence of Szasz's claim that mental illness is a myth by bringing to bear a standard type of thought experiment used in philosophical discussions of the meaning of natural kind concepts. This makes it possible to accept Szasz's conclusion that mental illness involves problems of living, some of which may be moral in nature, while bypassing the debate about the meaning of the concept of illness. The chapter then considers the nature of schizophrenia and the personality disorders (PDs) within this framework. It argues that neither is likely to constitute a scientifically valid category, but that nonetheless their symptoms can be scientifically explained. It concludes with a discussion of the way in which Cluster B or 'bad' PDs involve failures of virtue or character, and argues that this does not preclude them from being appropriately treated within contemporary, multidisciplinary, mental health services.

### 4.1 Introduction

Thomas Szasz is famous for his slogan that mental illness is a myth (Szasz, 1960, 1974). Its pithiness makes it ambiguous. Its shock content makes it politically serviceable. But nonetheless it summarizes an interesting and substantial philosophical position. Szasz's position is that the problems which psychiatry treats are not medical but moral. That is because mental illness is not actually an illness – properly understood.

The aim of this chapter is to explore what is right and what is wrong in this view. The chapter has three parts. In the first part, I outline Szasz's argument that mental illness is not actually an illness, together with some of the more common objections to it. I resolve this debate by bringing to bear a type of thought experiment which is standardly used in philosophical accounts of the meaning of natural kind concepts. This makes it possible to get beyond semantic disputes over the meaning of the concept of illness. Szasz's claim that mental illness is a myth can then be reconciled with the potential scientific validity of particular kinds of mental illnesses. In the second part,

I briefly examine the evidence against schizophrenia having this status. Although there is reason to be sceptical that schizophrenia constitutes a scientifically valid category, I suggest that this should not make us sceptical that its symptoms are open to scientific explanation, broadly conceived. In the third part, I turn to the Cluster B or 'bad' PDs. PDs are generally regarded as poorly defined and unlikely to be real illnesses or diseases. Louis Charland has argued that the Cluster B categories – narcissistic, histrionic, borderline, and antisocial – are by definition moral as opposed to clinical conditions (Charland, 2004, 2006). Hence the Cluster B PDs seem poised to vindicate Szasz's position. I argue that Charland's account of Cluster B PDs is overly simplistic. The Cluster B PDs do involve, among other traits, failures of virtue and character. However, it is possible to construct scientific explanations of the development of virtue and character – of how it progresses or fails. And this in turn helps us understand why one of the most effective treatments for PDs, namely, group psychotherapy, works. For these and other reasons, Cluster B PDs are appropriately treated in contemporary, multidisciplinary, mental health clinics. Taken together, these considerations make the question of whether or not the PDs are medical conditions idle. The chapter concludes by highlighting the relevance of these findings to Szasz's overall view.

## 4.2 Bodily illness, mental illness, and natural kinds

The aim of this section is to show that Szasz's positive conclusions about the nature of mental illness can be preserved even if his argument purporting to establish these conclusions is rejected. In outline, Szasz's argument for the claim that mental illness is a myth is as follows. Mental illness would appear from its name to be a kind of illness. But Szasz believes that our understanding of illness is fundamentally bodily: bodily illness is our basic paradigm or model of illness. We do not have two equal species of a single conceptual kind. Hence, only if mental illness meets the criteria for illness, as established by reflection on the paradigm or model of bodily illness, can it count as real. What then are the criteria for illness? Szasz holds that illness involves a deviation from the normal anatomical or physiological structure and functioning of the human body. Clearly, such deviations can be of great concern to us: illness has negative connotations, just as health has positive ones. But Szasz thinks we should aim to extract the scientific core of the concept of illness from its more evaluative connotations. The deviations are to be biologically defined as, for instance, lesions can be. Given this account of our concept of illness, we can now ask whether or not mental illness is indeed a real illness. The answer will depend on whether or not mental illness involves a deviation from the normal anatomical or physiological structure and functioning of the human body. And Szasz claims that the scientific evidence suggests that it does not. It involves instead a deviation from 'psychosocial, ethical, and legal' norms (Szasz, 1960, p.114).

Hence, according to Szasz, the term 'mental illness' is a misnomer. Mental illness is not illness. It is rather a form of cultural deviation – a failure, whether voluntary or not, to conform to normal physical and psychological behavioural expectations. The concept is used to refer to 'problems of living' so severe, wayward, or disturbing, for the individual or society, that they are not treated as falling within the more ordinary,

acceptable miseries and difficulties of human life. These problems are of course perfectly real. Something is very much wrong. But they do not constitute an illness, according to Szasz, once that term has been properly understood.

Szasz has always claimed that, if science ultimately provides evidence for an organic, biological basis for mental illness, then it would count as a real illness. The most likely basis would of course involve a deviation from the normal structure and functioning of the human brain. Hence he concedes that if, for instance, a brain lesion was discovered which reliably correlated with schizophrenia, then schizophrenia would count as a real illness. Mental illness would not be a myth.

Critics often seize on this concession. On the one hand, neuroscience has progressed since Szasz first put forward this argument. Perhaps, there is now sufficient evidence for a correlation between abnormal structure and functioning of the brain and schizophrenia for it to count as a real illness. I shall discuss some of this evidence in the following section. But, even if the jury is still out on the current strength of the evidence, one might think that it is a good bet that strong evidence for a neural basis for mental illness will in the end be forthcoming. Scientific optimism in this respect may not seem irrational.

On the other hand, critics often seize on Szasz's account of bodily illness, for it is clearly open to counterexample. Athletic prowess, for instance, is a deviation from normal anatomical or physiological structure and functioning. But it is not an illness. Moreover, the account provides no measure for determining how much deviation is required for illness. When, for instance, does raised blood sugar truly become diabetes?<sup>1</sup> These gaps in Szasz's account have contributed to a growing literature attempting to analyse our concepts of illness and disease, and correspondingly, well-being and health. One powerful alternative account links illness or disease to evolutionary disadvantage: illness or disease is a dysfunction which is likely to reduce life or reproductive expectancy (Boorse, 1975; Kendell, 1975). Scientific evidence can then be mustered to argue that particular kinds of mental illnesses do or do not constitute this sort of dysfunction. There is evidence, for instance, that schizophrenia is correlated with reduced life expectancy and increased risk of suicide (Radomsky *et al.*, 1999; Hannerz *et al.*, 2001). Another alternative account links illness or disease to failures of action or 'ordinary doing' (Fulford, 1989). Many symptoms of mental illness are failures of action. For instance, arguably most of the negative symptoms of schizophrenia, such as *athymia* (flattening of emotional expressions and action), *alogia* (reduction in speech and poverty of speech content), and *abulia* (lack of personal grooming and general low energy), as well as some of the positive symptoms, such as delusions of control, involve disorders of action. Hence according to both these alternative accounts, mental illness again would not be a myth.

But Szasz should not have made his initial concession. Or, more precisely, it is possible to hold both of the following claims: first, that particular kinds of mental illnesses may prove to be valid scientific kinds, and second, that our concept of mental

<sup>1</sup> See Haslam, 2002 for an attempt to answer this kind of question through practical considerations.

illness, as an overarching or generic category, involves a deviation from ‘psychosocial, ethical, and legal’ norms and, in this sense, may be unlike our concept of bodily illness – however this is ultimately correctly understood. To see why this conjunction is possible, I want to bring to bear on this debate the standard type of thought experiment used in philosophy to establish whether or not a concept is a natural kind concept.

This type of philosophical thought experiment involves prying apart the superficial properties of a kind of thing from its underlying, scientific properties. This allows us to test our intuitions about the meaning of the concept of that kind: to determine which sorts of properties the concept tracks. Consider, for instance, a famous example from Hilary Putnam’s early work on externalism about meaning (Putnam, 1973).<sup>2</sup> Water is a colourless, clear liquid which we drink and with which we clean. It is found in oceans, lakes, and rain clouds, as well as baths and bottles. It freezes and evaporates in certain circumstances. These are some of its superficial properties. We learn about these properties of water simply by living in the ordinary world, observing, and interacting with it. But water also has the chemical composition  $H_2O$ . We learn about this only by doing science. Now imagine that there is a planet called Twin Earth. On Twin Earth, there is a liquid which looks and behaves and is used just like water. It is even called ‘water’ by the natives. But it has a different chemical composition. We can call this XYZ. Is the liquid found on Twin Earth actually water? Putnam thinks that our intuitions are clear: it is not. Water is  $H_2O$ . This liquid is XYZ.

What we learn from this thought experiment is that our concept of water is not determined by superficial properties. It is rather determined by underlying, scientific properties, such as chemical composition. That is the essence of what water is. Only something that possesses the scientific property of having the chemical composition of  $H_2O$  can be water. That is why XYZ is not water despite having all the same superficial properties. Hence, our concept of water is a natural kind concept because its meaning is linked to underlying, scientific properties, as opposed to superficial properties. The concept tracks a kind of thing which exists in the natural world independently of us and our interactions with or conceptions of that thing.

Now imagine the following thought experiment about schizophrenia. Schizophrenia is currently defined by its symptoms. These are superficial or personal-level properties pertaining to psychological and physical functioning and behaviour, which are identified by psychiatrists through interview and observation. But now suppose that, as Szasz concedes is possible, we discover a brain lesion that correlates with schizophrenia. Suppose that this correlation is extremely reliable. It is so reliable, in fact, that the diagnostic procedure for schizophrenia changes. When psychiatrists see a new patient who, at first sight, has a clinical presentation which might indicate schizophrenia, rather than using interview and observation, they perform a brain scan in aid of diagnosis: they test in the first instance for the underlying scientific property, not for the superficial symptoms.

<sup>2</sup> See also Kripke, 1972 for a seminal discussion of these topics.

Now suppose that, in the course of routine brain scans, say, we discover a person who has this lesion, but none of the symptoms currently definitional of schizophrenia.<sup>3</sup> Her problems of living are perfectly ordinary. Nothing is terribly wrong. That is a conceptual possibility we seem to be able to coherently imagine once we have embarked on this thought experiment. It may also be a metaphysical possibility. Compare, for instance, prostate cancer. A man may have prostate cancer (because the cells in his prostate have mutated into cancer cells) without having any superficial symptoms which would have led to a diagnosis in absence of advance screening (such as a blood-test for increased prostate-specific antigen). But, for our needs, conceptual possibility is sufficient: our interest lies in probing the meaning of our concepts.

Does this woman have schizophrenia? We may be unsure, but, it seems at least possible that, given the conditions imagined in this thought experiment, our intuitions incline us to think that she does. For instance, we can easily imagine that she might be advised that the lesion should be operated on for preventative reason, lest it develop from 'latent' into 'full-blown' schizophrenia. But instead, suppose we ask: is this woman mentally ill? It seems our intuitions about this are entirely clear. She is not. We may in the end judge that she has schizophrenia, given the hypothesized discovery of its underlying, scientific property and its place in diagnostic procedures. But she is not mentally ill – any more than she is mentally disturbed, or mentally distressed, or mad, or crazy, or insane. She has no superficial or personal-level symptoms. She does not deviate from our 'psychosocial, ethical, and legal' norms.

Hence Szasz's claim that mental illness, considered as an overarching or generic category, is a myth is compatible with the claim that particular kinds of mental illnesses, like schizophrenia, are valid scientific kinds. If we pry apart the superficial and the underlying scientific properties, our concept of mental illness tracks the former, even if our concepts of particular kinds of mental illnesses track or come to track the latter.

The importance of this point is twofold. First, it is important because it means that Szasz need not concede that science may in the end prove him wrong. Practically, that helps safeguard the various positive clinical and political changes that have their origins partly in Szasz's ideas, such as the new focus on mental health service-user involvement and responsibility. Theoretically, that allows us to begin to set the potential interest and truth of Szasz's views about the moral nature of psychiatric problems within a developing psychiatric science. I shall discuss what this means in more detail in the following sections of this chapter.

Second, it is important because it allows us to bypass the ongoing debate about the meaning of our concepts of illness, disease, well-being, and health. For it is reasonable to be sceptical that this debate will prove fruitful if conducted as an analytic enterprise. On the one hand, just as there were counterexamples to Szasz's account of bodily illness, so too there are counterexamples to evolutionary and action accounts. For instance, evolutionary accounts typically count homosexuality as an illness or disease, whilst failing to count conditions like non-erythrodermic psoriasis. Action accounts

<sup>3</sup> To aid the imagination, we can hypothesize if we wish that her cognitive reserve is extraordinarily high.

have difficulty accommodating illnesses whose symptoms involve not dysfunctions of movement, but abnormal subjective experiences and pain, for instance, mild tooth decay, or the common cold. On the other hand, all these accounts face the problem of how much these concepts involve phenomenological as opposed to scientific criteria; and, relatedly, how much they involve the idea that illness and disease are bad, health and well-being good.<sup>4</sup>

We can, of course, choose to stipulate what we shall take these concepts to mean. It might be helpful, for instance, both for political and theoretical purposes, to reserve the concept of disease for evolutionary dysfunction, and to use the concept of illness to cover more subjective, experiential properties of poor health.<sup>5</sup> But that would constitute a decision, not an analysis. Our concepts of illness, disease, well-being, and health – whether mental or physical – lie at the interface of science and common sense, of fact and value, as well as exhibiting a large degree of cultural and historical development and variation.<sup>6</sup> It is thus highly unlikely that they are the right kind of concepts to admit of the kind of analytical definition which is needed to ground philosophical arguments of the sort Szasz and his critics alike envisage. Of course, that is not to say that their exploration is not of considerable interest. But it should be undertaken firmly as a social and historical explanatory endeavour, not as a question of semantic analysis.

Hence, no matter how the concept of illness is properly understood, and whether or not mental illness conforms to it, we can yet draw two conclusions. First, Szasz is correct that our concept of mental illness and its cognates involve ‘deviation from psychosocial, ethical, and legal norms’. These concepts refer to problems of living which are certainly personal, and possibly moral. Second, particular kinds of mental illnesses may yet constitute valid scientific kinds. The next section looks at the status of schizophrenia in this regard.

### 4.3 The scientific status of schizophrenia

The paradigm example of a kind of mental illness which might count as a real illness is schizophrenia. But scepticism about the prospects of analyzing illness and related concepts should make us wary of focusing too narrowly on the question of whether or not schizophrenia is an illness. Instead, we can ask a related though distinct question, namely, whether or not it constitutes a valid scientific kind or category. This is because, a positive answer to that question might be sufficient – given the clinical features of schizophrenia, which are not in dispute – for schizophrenia to then count as a real illness, whatever that ultimately does or should mean.

Is schizophrenia a valid scientific kind or category? Sometimes philosophers intend something very modest by this idea. They mean only that a category is in fact scientifically studied and supports explanations and inductive inferences (Dupre, 1993; Cooper, 2007).

<sup>4</sup> See Carel, 2008 for an account that emphasizes the phenomenological aspects of health and illness.

<sup>5</sup> Arguably this is how Boorse, 1975 should be read.

<sup>6</sup> See, for instance, Foucault, 1971 and 1976; Kleinman, 1980; Thagard, 1997; Porter, 2002.



But if that is all that is meant, then PDs are as scientifically valid as schizophrenia. For there is a wealth of scientific research on, for instance, borderline PD, and it is clear that knowing that a person is borderline allows one to offer explanations and make inductive inferences which are potentially as reliable as any psychological inferences can be (e.g. if someone is borderline, then perceptions of abandonment will cause immoderate fear and anger responses). In the next section, I shall explore the extent to which the Cluster B PDs may indeed be open to scientific explanation. But it is clear that those who appeal to schizophrenia as a scientifically valid category of mental illness would wish to distinguish it from the more amorphous PDs. So this modest understanding cannot be sufficient to capture the scientific hope for schizophrenia.

The potential scientific validity of schizophrenia instead typically involves two ideas. The first is that schizophrenia is a category which carves the world at its joints: it accurately picks out a real and independently existing kind of thing, objectively distinct from other, perhaps superficially comparable, kinds of things. Indeed, this is explicitly recognized in DSM-IV which states that psychiatric categories succeed when 'there are clear boundaries between classes, and when the different classes are mutually exclusive' (APA, 1994, p. xxii). The second is that an underlying, scientific basis for schizophrenia will be found which is correlated with and potentially explanatory of the development and nature of its superficial symptoms.

It is now widely accepted within schizophrenia research that there is strong evidence for questioning whether schizophrenia is a scientifically valid category thus understood. The evidence involves the difficulty distinguishing it from other mental illnesses and, in particular, the other major Axis I psychosis, bipolar disorder, together with the presence of minor psychotic symptoms in the general and prodrome population. Broadly speaking, there are at least six major considerations.<sup>7</sup>

*Genetics and brain structure.* The most likely underlying, scientific correlate for schizophrenia is genes or brain structure. With respect to genetics, it is well known that mental illness runs in families. There is some evidence that certain specific psychotic symptoms, like thought disorder, may do as well (Wahlberg *et al.*, 1997). However, other symptoms, like paranoid tendencies and delusions, probably do not (Coolidge *et al.*, 2001). But it is clear that genetic factors are only one element in a causal explanation of schizophrenia. Analysis of twin and adoptive studies, for instance, seems to point to the importance of non-genetic factors in the development of schizophrenia (Bentall, 2003). Prenatal development, substance abuse, stress, anxiety, and mood disorders, and social and environmental factors such as urban life, migration, poverty, family dysfunction, and isolation, are all thought to be contributors (Broome *et al.*, 2005). Many of these factors, of course, are known to contribute to the development of other kinds of mental illnesses too. Finally, the genes that seem to be correlated with schizophrenia are non-specific: they are also correlated with bipolar disorder (Craddock *et al.*, 2006).

With respect to brain structure, there is ample evidence for structural and functional brain abnormalities in schizophrenia (Shenton *et al.*, 2001). However, there is

<sup>7</sup> See Bentall, 2003 for a good survey of this and related evidence.

some evidence that some of these abnormalities may be caused by medication (Molina *et al.*, 2005). And there are relatively few studies of brain abnormalities in other mental illnesses, in particular, bipolar disorder, so comparative studies are not readily available. Finally, until we have a fuller and more precise understanding of how the brain abnormalities in schizophrenia cause superficial symptoms, we cannot rule out the possibility that they are caused by them.

*Reliability of diagnoses.* The reliability of diagnoses for mental illness is not high. A reliability study of diagnosis of schizophrenia and bipolar disorder for DSM-III where conditions were idealized, places the reliability of diagnoses at 0.6 (Williams *et al.*, 1992). A more recent study in non-idealized conditions places it at 0.65 (McGorry *et al.*, 1995). Meanwhile, the further criteria and complications introduced in DSM-IV make the categories of mental illness themselves appear increasingly gerrymandered (Kutchins and Kirk, 1997). Of course, lack of reliability is not conclusive evidence. Clinical training and procedures vary. Humans are fallible. However, reliability is nonetheless a general indication or guide to stable and objective categories. When a category is scientifically valid and a procedure for identifying the category standard, it is reasonable to expect reliability to be good.

*Exclusion rules and co-morbidity.* A salient form of gerrymandering is the use of exclusion rules to ensure the uniqueness of diagnosis in DSM-IV. For instance, a person cannot be diagnosed with schizophrenia if he or she meets the criteria for schizoaffective disorder, major depression, or mania. Similarly, a patient cannot be diagnosed with bipolar disorder if his or her symptoms fit a different diagnosis better. In a large-scale study funded by the American National Institute for Mental Health involving over 18 000 patients, it was found that 60% of people who met the criteria for one disorder equally met the criteria for another disorder if the exclusion rules were suspended (Robins *et al.*, 1991). This level of co-morbidity is higher than chance. It is also higher than we might expect even taking into account the potential causal interaction between different categories of mental illness. It seems likely that it reflects a failure of the DSM-IV categories to capture objectively real and distinct scientific kinds.

*Discriminant function analysis.* Studies using discriminant function analysis seem to indicate a continuum between schizophrenia and bipolar disorder. The basic idea behind this sort of analysis is simple. Patients are not diagnosed but are instead assigned scores according to the extent to which their individual symptoms are schizophrenic, and the extent to which their individual symptoms are bipolar. If schizophrenia and bipolar disorder are real and distinct kinds, the majority of the patients' scores should cluster at one or either pole. Instead, the opposite appears to be the case: most patients' scores are intermediate (Kendell and Gurlay, 1970; Brockington *et al.*, 1991).

*Psychotic symptoms in the general and prodrome population.* Isolated and transient psychotic phenomena are present in the general population. One study suggests that 25% of the general population report hallucinations or delusions (Poulton *et al.*, 2000). Some of the risk factors associated with schizophrenia are also associated with such minor psychotic phenomena (van Os *et al.*, 2000). Hence there is evidence

that minor symptoms in the general population are related to the psychotic symptoms of schizophrenia. There are also prodromal patients who seem to develop along a trajectory: from cognitive dysfunction, to decreased motivation and socialization, to positive psychotic symptoms but without sufficient intensity and longevity to meet the criteria for schizophrenia (Broome *et al.*, 2005). Some of these patients go on to develop symptoms which do meet the criteria, but others do not. This combined evidence suggests that schizophrenic-type symptoms are not unique to schizophrenia, but rather exist along a continuum.

*The history and sociology of psychiatry.* This topic is too large to be adequately addressed within the scope of this chapter. However, it is worth emphasizing that psychiatry has had a complicated history, and has been more subject, than many disciplines, to political, social, and legal pressures. Decisions about how to demarcate psychiatric categories have clear practical human import, in a way that decisions about how to demarcate sub-atomic particles do not. It is also worth remembering that mental illnesses befall people who then come to think of themselves as a particular kind of person, with a particular sort of problem. Ways of conceptualizing symptoms can then become less tractable, and can also spread through cultural transmission. Although this may be less applicable to schizophrenia than to identity, eating, and personality disorders, an awareness of the historical and social context of psychiatry is still essential.<sup>8</sup>

Taken together, this evidence suggests that schizophrenia is not a category that carves the world at its joints. As yet, scientists have not discovered an underlying, scientific property, whether genetic or neurological, with which it is distinctively correlated. Meanwhile, evidence stemming from studies of reliability, co-morbidity, discriminant function analysis, psychotic symptoms in the general and prodrome population, and the history and sociology of psychiatry, suggests that the category as currently defined, by superficial properties, is unlikely to be tracking a unified and distinct kind of thing. Hence the possibility that schizophrenia picks out real symptoms without tracking a scientifically valid kind is genuine.<sup>9</sup> Importantly, this does not mean that the symptoms of schizophrenia cannot be scientifically explained.

Suppose that, rather than conceive of our current categories of mental illnesses as falling into real and distinct kinds, we conceive of them as positioned along a continuum or spectrum: there are various dimensions along which all patients vary. It may nonetheless be possible to carve up the continuum or spectrum in a scientifically objective way. Factor analyses attempt to do just this. They attempt scientifically to establish how symptoms cluster together. There is growing evidence, for instance, that schizophrenia divides into three clusters: positive symptoms (e.g. hallucinations and delusions), negative

<sup>8</sup> For discussion of some of these themes, see, for instance, Hacking, 1995, 1998, and 1999; Bentall, 2003; Fulford *et al.*, 2006; Radden, 2006.

<sup>9</sup> It is possible to accept this point while yet holding that schizophrenia is nonetheless a kind and not just a collection of symptoms. Haslam, 2002, suggests that we need to allow for many 'kinds of kinds': psychiatric illnesses may not be natural kinds, but they may nonetheless be practical, fuzzy, or discrete kinds.

symptoms (e.g. apathy, athymia, and asociality), and cognitive disorganization (e.g. disturbed speech and problems of attention) (Liddle, 1987; Andreasen *et al.*, 1995). There is also some evidence that these clusters may be valid not only for schizophrenia, but also for major depression and bipolar disorder (Toomey *et al.*, 1998). There are substantive questions about the validity of factor analysis, involving selection of symptoms, patients, and methods of calculation. But importantly, if the division of symptoms into clusters can be validated, the process of discovering an underlying, scientific property, which is not only correlated with the cluster, but potentially explanatory of it, can begin. Cognitive models for clusters of symptoms can be constructed. Bridges to neuroscience can then be built. And causal and development conditions can be explored. A focus on clusters of symptoms opens the door to scientific explanation, in a way that a focus on categories of mental illness currently does not.<sup>10</sup>

If schizophrenia is not a scientifically valid kind, then the prospects of any of our current categories of mental illness proving to be so are dim. But nonetheless, we have a model of how the symptoms of schizophrenia – Szasz’s ‘problems of living’ – can potentially be scientifically explained. In the next and final section of this paper, I consider the Cluster B PDs, and whether or not this is equally true of them.

#### 4.4 The ‘bad’ personality disorders: moral or medical conditions?

PDs are not generally regarded as mental illnesses, although few would deny that people with PD experience high levels of mental distress and disturbance. They are defined as ‘an enduring pattern of inner experience and behaviour that departs markedly from the individual’s culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress and impairment’ (APA, 1994, p. 629). Classifications of PDs have shifted over time. DSM-IV groups them into three clusters. Cluster A includes paranoid, schizoid, and schizotypal; Cluster B includes antisocial, borderline, histrionic, and narcissistic; Cluster C includes avoidant, dependent, and obsessive–compulsive. These are colloquially called the ‘mad’, ‘bad’, and ‘sad’ clusters respectively. Although precise percentages are disputed, there appear to be high levels of co-morbidity between PDs, and between PDs and Axis I disorders, as well as high levels of PD in the population at large. Patients with PDs have a reputation of being difficult to treat, and being disliked by clinicians.

Louis Charland has argued that Cluster B PDs are empirically valid categories, in that they represent genuine behavioural syndromes and capture regularities in human behaviour, but that they are not clinically valid categories (Charland, 2004, 2006). Charland does not define what a clinically valid category is. But the reason he gives is that, in contrast with the other types of PDs, he takes Cluster B PDs to be moral conditions: they are defined by moral failings. He points out that the criteria for antisocial PD include lying and conning. Narcissistic PD is partly defined by a lack of empathy. And he takes the

<sup>10</sup> For a paradigm example of this approach, see Frith, 1992. Frith explains schizophrenic disorders of thought and action as stemming from a failure of self-monitoring. Although I am sympathetic to the spirit of this kind of account, I question the adequacy of some of its details in my ‘Schizophrenia and the epistemology of self-knowledge’ (under review).

inappropriate sexual behaviour characteristic of histrionic PD, and the intense anger, impulsivity, and instability of personal relationships characteristic of borderline PD, to imply 'moral deficits in empathy and regard for others' (Charland, 2006, p. 122). Hence, he concludes that Cluster B PDs are not clinical but rather moral conditions. For this reason, recovery from a Cluster B PD involves something more akin to 'moral conversion' than medical treatment or therapy (*ibid.*). And there is no reason to think that psychiatrists, psychologists, or psychotherapists of virtually any bent are well placed to effect this.

Charland's argument is problematic in at least two regards. On the one hand, the supposed moral dimension of narcissistic, histrionic, and borderline PD is only one aspect of these categories. The criteria for diagnosis are polythetic: they are various, and a patient need only meet a required number for diagnosis, not all. So it is possible, for instance, to meet the threshold for diagnosis with narcissistic PD without lacking empathy. A person could instead suffer from grandiosity and a need for admiration on a sufficiently large scale. Similarly, severe and persistent self-harming behaviour is as central to borderline PD as intense anger towards others. Antisocial PD is perhaps different: here a pervasive disregard for and violation of the rights of others is more central to the syndrome. But, in general, the fact that certain categories of PD have a supposedly moral dimension does not on its own make them moral as opposed to clinical conditions. That would be to ignore all the other, standard clinical, dimensions of the category.

On the other hand, it is not clear that the dimensions of histrionic and borderline PD that concern Charland are properly described as moral deficits. Bernard Williams has suggested that morality proper is fundamentally to do with rights and obligations (Williams, 1985; 1993). Even if this seems too narrow, it does seem important to distinguish our concept of the moral from a broader, ethical perspective, which is concerned more generally with human virtue and goodness. Engaging in inappropriate sexual behaviour, as the histrionic person may, or failing to control intense anger and to lash out at others, as the borderline person may, certainly count as failings, but they are not obviously failures of morality. They are failures of character. These are not the kinds of things that the virtuous do. I work in a NHS Therapeutic Community for people with PD. Most of the patients meet the threshold for diagnosis with one or more Cluster B PD. Compared with people whose personalities are not diagnosable, these patients often seem to lack the virtues of, for instance, temperance and moderation, fairness and generosity to others, humility, trust, patience, and love and respect for self and others. Hence Charland has put his finger on something important, even if he has oversimplified his case. The Cluster B PDs do seem to represent an instance where Szasz's position is correct to this extent: psychiatry here treats problems which clearly pertain to questions, if not exactly of morality, then certainly of virtue and character. This raises two questions. First, can the 'bad' PDs nonetheless be scientifically explained? Second, are they medical conditions? In particular, should they be treated by psychiatrists within contemporary, multidisciplinary mental health clinics?

Consider first the question of scientific explanation. Charland claims that the Cluster B PDs are empirically valid categories in the modest sense of capturing regularities in behaviour and so supporting explanations and inductive inferences. This is clearly correct. But the more demanding question is whether it is possible to construct, at least in principle, a scientific explanation of how they develop. Part of the difficulty

in answering this question is that, on the whole, there is little to go by. On the one hand, psychoanalytic theory has offered developmental explanations for many of the features associated with PDs.<sup>11</sup> This body of theory can be clinically helpful. And some of its ideas are, once clarified, potentially important and testable. But as it stands as a theoretical corpus, its scientific status is dim. Academic psychology, on the other hand, has not tended to address itself explicitly to questions of the development of the virtues and the formation of character. For instance, Lawrence Kohlberg has developed a justly famous, clear, rigorous, and empirically validated theory of the stages of moral development (Kohlberg, 1981). But Kohlberg's theory – and the literature surrounding it – is concerned with moral reasoning, with a particular emphasis on justice. It does not address questions of virtue and character.

Indeed, arguably our best account of the development of the virtues and the formation of character is still Aristotle's.<sup>12</sup> Unlike many moral philosophers, Aristotle emphasizes the development of the good person over time. The acquisition of virtue and character is a long process, each phase consequent on what has come before. Simplifying somewhat, we can see Aristotle as dividing this process into three stages.

The first stage involves a good upbringing and a habituation to virtue. Desires and feelings start to shape patterns of motivation and response – start to shape character traits – well before children become reasonable and reflective beings. Children need to be brought up in an environment which allows virtuous ways of being to become their second nature. The virtues need to become habits, flowing more or less readily and easily, as opposed to involving deliberation or ambivalence. For this to happen, children need guidance. But the nature of this guidance is very distinctive. It is not only that the child must be guided so that he or she acquires the right habits. The child must also be guided so that, in acquiring these habits, he or she is able to come to see that they are good.

This is the second stage of development. What Aristotle means by the claim that the child must see that the virtues are good is not simple to spell out, but, roughly speaking, it involves knowing that behaving virtuously is good not simply because that is what one has been told, but for oneself. And Aristotle holds that one comes to this knowledge through pleasure and enjoyment of virtuous ways of being: one comes to see that virtue is good because one is guided in one's doing of virtuous actions so that one finds pleasure or enjoyment in the doing. This pleasure, and the knowledge that it produces, serves to transform childhood habits into stable states of character.

The third and final stage, which can only occur once the child has grown up and become a fully mature human being, is the acquisition, through reason and reflection, of the knowledge of why virtue is good. For Aristotle, such knowledge is only available to the person who already knows that it is.

<sup>11</sup> See, for instance, McWilliams, 1994, for a survey of the contribution of psychoanalysis to understanding personality and character.

<sup>12</sup> I owe my understanding of Aristotle's position to Myles Burnyeat's seminal paper 'Aristotle on learning to be good' (Burnyeat, 1980).

To make this more concrete, let us take as an example of the development of the capacity to share. Children need to be told that they must share. They need to be made to do it, so that it becomes a natural expectation that they have – a habit. But this guidance must not proceed by brute force. It must allow them to come to enjoy sharing, to find the pleasure in giving, and so to learn for themselves that sharing is good. Now it is a serious question what such guidance in fact involves. It is natural to conjecture that it should be, for instance, firm but not punishing; conscious of and open about the genuine struggle of sharing for the child, as well as praising of his or her successes; lastly, it should ensure that the child is also the recipient of sharing – that he or she gets a chance to experience how nice it is to be shared with. But whatever the precise nature of the guidance must be, the point is that it must allow the child to feel the pleasure in sharing. This pleasure then serves to cement the capacity to share in children – to make it a stable part of their character. Later, once they are grown, they can then reflect on why sharing is good. For instance, perhaps it is a requirement of kindness, or alternatively, of the principles of equality and justice.

The importance of Aristotle's account is that it provides us with a general and clear theory about how character traits form and develop over time – an abstract schema. And that schema is something that can be scientifically explored, both quite generally, and in the case of particular character traits.

Consider, for instance, the intense anger and impulsivity which is characteristic of borderline PD. This trait is essentially a failure of moderation and control of anger. No doubt one component of the explanation of possession of this trait is genetic predisposition. But most borderline patients have suffered terrible childhoods, with high levels of emotional, sexual, or physical abuse or neglect. They have not had the sort of upbringing and guidance Aristotle suggests the virtues require. Meanwhile, we are beginning to understand more about the development of the brain structure and functioning underpinning the control and moderation of anger. There is evidence that the orbitofrontal cortex is the part of the brain which, among other functions, is responsible for managing one's own emotions and responding to the emotions of others (O'Doherty *et al.*, 2003; Schore, 2003). It can hold back strong and basic fear and anger responses, for instance, which originate in the evolutionarily more primitive amygdala and hypothalamus. But the proper development of the orbitofrontal cortex is experience-dependent. On the one hand, there is evidence that severe neglect in infancy is correlated with a lack of orbitofrontal development in later years (Chugani *et al.*, 2001). On the other hand, there is evidence that positive attention – smiles, warmth, and praise – from carers produces a biochemical response with two effects: it is pleasurable and it helps the neurons in the orbitofrontal cortex grow (Schore, 1994). The evidence about possible orbitofrontal abnormality among patients with borderline PD is limited, although there is some indication of hypometabolism and reduced volume (De La Fuente *et al.*, 1997; Lyoo *et al.*, 1998). However, an initial study comparing borderline PD patients with patients with damage to their orbitofrontal cortex concludes that both categories of patients, in contrast with normal controls, were similar in the extent of their heightened anger and impulsivity, whilst differing in most other clinical respects (Berlin *et al.*, 2005). Synthesizing this research suggests a simple hypothesis: the anger and impulsivity characteristic of borderline PD can be

explained by abnormal development of the orbitofrontal cortex because of early childhood neglect and abuse, perhaps in conjunction with genetic predisposition.

Of course, this hypothesis is only a beginning. It may prove wrong. And even if it proves true, there are many details left to flesh out. Can we say more about how and why the right sort of guidance facilitates orbitofrontal growth and the wrong sort of guidance impedes it? Can we say more about what the right or wrong sort of guidance is? How exactly does the orbitofrontal cortex control or fail to control these emotions? The point is that this hypothesis represents a kind of scientific explanation which should, in principle, be available for character traits quite generally – traits which we consider virtues, and traits which we consider vices. Aristotle has provided us with a general schema for thinking about the development of character. And the integration of research from genetics, psychology, psychiatry, neuroscience, and the social sciences can provide a way of explaining the genesis of particular traits within this schema.

It is possible that pursuing this sort of research will validate the categories of PD by revealing how each behavioural syndrome, with its polythetic criteria, has a coherent, unified scientific underlying and developmental explanation. More likely, perhaps, it will pry apart the different character traits within each category, explaining each individually, and suggesting how they naturally, although not inevitably, cluster together. The point is that the failings of virtue and character typical of PDs can be scientifically explained. Just as we can potentially explain the symptoms of schizophrenia and how they cluster, so too we can potentially explain the character traits typical of PDs and how they cluster. These explanations do not depend on either schizophrenia or the PDs being scientifically valid kinds.

Consider now the second question. Are Cluster B PDs medical conditions despite the fact that they involve failures of virtue and character? The fact that Cluster B PDs can potentially be scientifically explained does not entail that they should be properly treated through, broadly speaking, medical means. Indeed, according to an austere understanding of our concept of medicine, which links it strongly to our concept of illness, they should not. Nonetheless, there is good reason to hold that PDs are properly treated in contemporary, multidisciplinary, mental health clinics, involving psychiatrists, psychologists, and psychotherapists of various bents. At least in this respect, they are medical conditions.

There are three reasons for this. Two are practical. First, there are high levels of co-morbidity between PDs, and between PDs and Axis I disorders. Patients who possess traits which count as failings of virtue or character are likely also to possess traits and symptoms which do not. Many of these patients will have been placed on medication, like antipsychotics, antidepressants, and mood stabilizers which require medical monitoring. They may also need to be treated in conjunction with other services, for instance, those specializing in substance abuse or eating disorders. Hence, the bulk of their treatment is unlikely to be a form of ‘moral conversion’ – to use Charland’s phrase – even if a part of their treatment is.

Second, the most effective treatment specifically targeting PDs themselves is psychotherapy (Fonagy *et al.*, 2006). Indeed, it is now widely held that recovery is possible. Of course, there is a long-standing question about whether psychotherapy itself should be conceived of as a medical treatment, and, relatedly, whether only



medically trained clinicians should practice it. What seems clear, however, is that, again, treatment within contemporary, multidisciplinary, mental health clinics is appropriate.

The third reason is theoretical. Suppose we accept that psychotherapy counts as a medical treatment if this is modestly construed as meaning that treatment by mental health services is appropriate. We still face the question: why does this treatment work? Charland likens recovery from Cluster B PDs to 'moral conversion'. He is therefore inclined to believe that psychotherapy works because it harbours specifically moral imperatives and interventions. For instance, he suggests that the therapeutic aim with borderline patients is 'to convince the client to try and be more honest, more truthful, less manipulative, and less resentful and vindictive. These are deeply human matters, where success probably hinges largely, if not entirely, on the therapist's ability as a moral being rather than a professional clinician' (Charland, 2006, p. 124). There is ample evidence suggesting that a major factor in therapeutic success is the human relationship between patient and therapist (Yalom, 1970). But this characterization makes psychotherapy look as if it presents the patient with a series of moral demands with which he or she can then choose to comply – and hopefully will, if only the psychotherapist argues convincingly enough. That would certainly count as a form of 'moral conversion'. Indeed, this conception of the nature of psychotherapy is presumably part of why Charland is sympathetic to the idea that PDs do not excuse moral failings – that judgements of responsibility must be distinguished from questions of character (Charland, 2006, p. 116).

But if Aristotle's schema is correct, then this cannot be the right view of why psychotherapy works as a treatment for PDs quite generally, let alone when we consider Cluster B categories. For, the basic insight of his account is that virtue and character develop over time, becoming more and more cemented and stable within a person. A borderline patient does not become convinced through therapy that it is right to moderate and control their anger. Indeed, it is likely that borderline patients will only embark on therapy in the first place if they hold this belief. It is rather that therapy provides borderline patients with the skills needed to moderate and control their anger: they learn to be less impulsive, more considered. Similarly, narcissistic patients may develop the capacity to be empathetic: through therapy, they come to acquire a new skill. More generally, the success of psychotherapy as a treatment for PDs – of whichever cluster – would seem not to depend on 'moral conversion' but rather on its potential for changing a person's character.

The Therapeutic Community where I work requires patients to commit to an 18-month, full-time programme of various kinds of group therapy, including, for instance, cognitive behavioural therapy, analytic group work, medication and self-diagnostic groups, psychodrama, and art therapy. The patients are also required to cook, clean, and generally participate in the running of the programme and the life of the community. The programme offers many different routes to change. But broadly speaking, these fall into two major kinds. The first is the chance to form new habits. This involves at least four components. First, therapists and more senior patients in the community both model and actively guide the behaviour of new patients. Second, therapy groups offer patients the opportunity to try new kinds of social behaviour in

a safe environment. Third, the community actively supports new behaviour, allowing patients the chance to experience its rewards – to find pleasure in it. Fourth, patients are given the opportunity to practice this behaviour – to try to solidify newly acquired traits. These routes to change can be seen as mirroring the processes Aristotle identifies as the first and second stages of a good upbringing.

The second kind of route to change is the acquisition of knowledge of the self. Through group therapy, patients come to know what they are like as a person, and how they developed that particular combination of traits. This knowledge is acquired through reflection on their behaviour within the group, and the exploration of its link to past experience (including the chance to re-live some of those experiences). It serves greatly to expand a patient's freedom and sense of self-esteem. Knowledge increases freedom because it allows mastery of and choice in behaviour that was previously automatic.<sup>13</sup> Knowledge increases self-esteem because an understanding of the nature and development of one's character offers the possibility of more realistic self-assessment. These routes to change do not explicitly mirror Aristotle's third stage of moral development, because they do not overtly address the question of why the virtues are good. They rather address the question of who one is and how one got there. However, they are likely to involve reflection on who one would like to become. An answer to this question usually requires exploring the nature of human virtue and goodness.

Cluster B PDs involve failures of virtue and character. There is potential for a scientific explanation of these disorders through a multilevel account of the development and formation of virtue and character. Meanwhile, psychotherapy works as a treatment because it can effect a change in character. Hence, there is a coherent and unified account of the nature, explanation, and treatment of Cluster B PDs. Finally, the treatment is most practically delivered within multidisciplinary, mental health clinics. So are Cluster B PDs medical conditions? At this point, it is difficult to see what knowledge can be gained by pressing this question: it is idle. For, it seems only to express a demand for an analysis of the meaning of our concept of medicine which goes beyond a modest construal that links it to contemporary health services. But, it is difficult to see how to pursue this demand without drawing on an analysis of our concepts of illness and disease, health and well-being. For instance, it is natural to suggest that medicine involves the diagnosis and treatment of illnesses or diseases, with the aim of restoring health or improving well-being. But now, we shall need an analysis of these concepts. As we saw earlier, this is not a need we should aim to meet. These concepts, and, relatedly, the concept of medicine, lie at the interface of science and common sense, of fact and value, as well as exhibiting cultural and historical development and variation. We can undertake a social and historical explanation, or we can make a decision about what we want them to mean. But we should not pretend that analysis can lead us to discover what a medical condition is, and, correspondingly, whether or not Cluster B PDs count as such.

---

<sup>13</sup> For a discussion of how this is possible, see Hampshire, 1974.

## 4.5 Conclusion

Szasz got mired in questions of the meaning of terms. But his conclusions were nonetheless in many ways correct. He believed that psychiatry could be a science, and that psychotherapy could be an effective means of change. What we must do is ‘recast and redefine the problem of “mental illness” so that it may be encompassed in a morally explicit science of man’ (Szasz, 1974, p. 263). Cluster B PDs involve failures of virtue and character. From a Szaszian perspective, they are especially striking because of how clearly they are defined, at least in part, by deviations from ‘psychosocial, ethical, and legal’ norms. But they can nonetheless be scientifically explained and effectively treated within contemporary health services. There need be no antithesis between science and morality within psychiatry. Mental illness is a myth that science can explain.

## Acknowledgements

I thank my colleagues at the Complex Needs Service as well as Lisa Bortolotti, Matthew Broome, Neil Levy, Ian Phillips, Maja Spener, and Nick Shea for extremely helpful comments on this chapter and discussion of its ideas.

## References

- American Psychiatric Association (APA) (1994). *Diagnostic and Statistical Manual of Mental Disorders*, 4th edn. Washington DC, American Psychiatric Association.
- Andreasen, N. C., Roy, M. A., and Flaum, M. (1995). Positive and negative symptoms. In *Schizophrenia* (eds. S. R. Hirsch, and D. R. Weinberger). Oxford, Blackwell
- Bentall, R. (2003). *Madness Explained*. London, Penguin Books.
- Berlin, H. A., Rolls, E. T., and Iversen, S. D. (2005). Borderline personality disorder, impulsivity, and the orbitofrontal cortex. *American Journal of Psychiatry*, **162**, 2360–2373.
- Boorse, C. (1975). On the distinction between disease and illness. *Philosophy and Public Affairs*, **5**, 49–68.
- Brockington, I., Roper, A., and Buckley, M. *et al.*, (1991). Bipolar disorder, cycloid psychosis and schizophrenia: a study using ‘lifetime’ psychopathology ratings, factor analysis and canonical variate analysis. *European Journal of Psychiatry*, **6**, 223–236.
- Broome, M. R., Woolley, J. B., and Tabraham, P. *et al.*, (2005). What causes the onset of psychosis? *Schizophrenia Research*, **79**, 23–24.
- Burnyeat, M. (1980). Aristotle on learning to be good. In *Essays on Aristotle’s Ethics* (ed. A. E. Rorty). Berkeley, CA, University of California Press.
- Carel, H. (2008). *Illness*. London, Acumen Publishing Ltd.
- Charland, L. (2004). Moral treatment and the personality disorders. In *The Philosophy of Psychiatry: A Companion* (ed. J. Radden). Oxford, Oxford University Press.
- Charland, L. (2006). Moral nature of the DSM-IV Cluster B personality disorders. *Journal of Personality Disorders*, **20**, 116–125.
- Chugani, H., Behen, M., Muzik, O., Juhász, C., Nagy, F., and Chugani, D. C. (2001). Local brain functional activity following early deprivation: a study of post-institutionalised Romanian orphans. *Neuroimage*, **14**, 1290–1301.
- Coolidge, F. L., Thede, L. L., and Jang, K. L. (2001). Heritability of personality disorders in childhood: a preliminary investigation. *Journal of Personality Disorders*, **15**, 33–40.

- Cooper, R. (2007). *Psychiatry and Philosophy of Science*. London, Acumen Publishing Ltd.
- Craddock, N., O'Donovan, M. C., and Owen, M. J. (2006). Genes for schizophrenia and bipolar disorder? Implications for psychiatric nosology. *Schizophrenia Bulletin*, **32**, 9–16.
- De La Fuente, J. M., Goldman, S., and Stanus, E. *et al.*, (1997). Brain glucose metabolism in borderline personality disorder. *Journal of Psychiatric Research*, **31**, 531–541.
- Dupre, J. (1993). *The Disorder of Things*. Cambridge, MA, Harvard University Press.
- Fonagy, P. and Bateman, A.W. (2006). Progress in the treatment of borderline personality disorder. *British Journal of Psychiatry*, **188**, 1–3.
- Foucault, M. (1971). *Madness and Civilisation*. London, Tavistock.
- Foucault, M. (1976). *The Birth of the Clinic*. London, Tavistock.
- Frith, C. D. (1992). *The Cognitive Neuropsychology of Schizophrenia*. Hove, UK, Erlbaum UK Taylor & Francis.
- Fulford, K. W. M. (1989). *Moral Theory and Medical Practice*. Cambridge, Cambridge University Press.
- Fulford, K. W. M., Thornton, T., and Graham, G. (2006). *Oxford Textbook of Philosophy and Psychiatry*. Oxford, Oxford University Press.
- Hacking, I. (1995). *Rewriting the Soul*. Cambridge MA, Harvard University Press.
- Hacking, I. (1998). *Mad Travellers*. Charlottesville VA, University of Virginia Press.
- Hacking, I. (1999). *The Social Construction of What?* Cambridge MA, Harvard University Press.
- Hampshire, S. (1974). Disposition and memory. In *Freud* (ed. R. Wollheim). New York, Anchor Books.
- Hannerz, H., Borga, P., and Borritz, M. (2001). Life expectations for individuals with psychiatric diagnoses. *Public Health*, **115**, 328–337.
- Haslam, N. (2002). Kinds of kinds: a conceptual taxonomy of psychiatric categories. *Philosophy, Psychiatry, and Psychology*, **9**, 203–217.
- Kendell, R. E. (1975). The concept of disease and its implications for psychiatry. *British Journal of Psychiatry*, **127**, 305–15.
- Kendell, R. E. and Gourlay, J. A. (1970). The clinical distinction between the affective psychoses and schizophrenia. *British Journal of Psychiatry*, **117**, 261–266.
- Kleinman, A. (1980). *Patients and Healers in the Context of Culture: An Exploration of the Borderland between Anthropology, Medicine, and Psychiatry*. Berkeley, CA, University of California Press.
- Kohlberg, L. (1981). *Essays on Moral Development, Vol. I: The Philosophy of Moral Development*. New York, Harper & Row.
- Kripke, S. (1972). *Naming and Necessity*. Oxford, Blackwell.
- Kutchins, H. and Kirk, S. A. (1997). *Making us Crazy: DSM – The Psychiatric Bible and the Creation of Mental Disorders*. New York, Free Press.
- Liddle, P. F. (1987). The symptoms of chronic schizophrenia: a reexamination of the positive–negative dichotomy. *British Journal of Psychiatry*, **151**, 145–151.
- Lyoo, I. K., Han, M. H., and Cho, D. Y. (1998). A brain MRI study in subjects with borderline personality disorder. *Journal of Affective Disorders*, **50**, 235–243.
- McGorry, P. D., Mihalopoulos, C., Henry, L., *et al.* (1995). Spurious precision: procedural validity of diagnostic assessment in psychotic disorders. *American Journal of Psychiatry*, **152**, 220–223.

- McWilliams, N. (1994). *Psychoanalytic Diagnosis: Understanding Personality Structure in the Clinical Process*. New York, The Guildford Press.
- Molina, V., Reig, S., Sanz, J., et al. (2005). Increase in gray matter and decrease in white matter volumes in the cortex during treatment with atypical neuroleptics in schizophrenia. *Schizophrenia Research*, **80**, 61–71.
- O'Doherty, J., Critchley, H., Deichmann, R., and Dolan, R. J. (2003). Dissociating valence of outcome from behavioural control in human orbital and ventral prefrontal cortices. *Journal of Neuroscience*, **23**, 7931–7939.
- Pickard, H. (under review). Schizophrenia and the Epistemology of Self-knowledge.
- Poulton, R., Caspi, A., Moffitt, T. E., Cannon, M., Murray, R., and Harrington, H. (2000). Children's self-reported psychotic symptoms and adult schizophreniform disorder: a 15-year longitudinal study. *Archives of General Psychiatry*, **57**, 1053–1058.
- Porter, R. (2002). *Madness: A Brief History*. Oxford, Oxford University Press.
- Putnam, H. (1973). Meaning and reference. *Journal of Philosophy*, **70**, 699–711.
- Radden, J. (ed.) (2004). *The Philosophy of Psychiatry: A Companion*. Oxford, Oxford University Press.
- Radomsky, E. D., Haas G. L., Mann, J. J., and Sweeny, J. A. (1999). Suicide behaviour in patients with schizophrenia and other psychiatric disorders. *American Journal of Psychiatry*, **156**, 1590–1595.
- Robins, L. N., Locke, B. Z., and Reiger, D. A. (1991). An overview of psychiatric disorders in America. In *Psychiatric Disorders in America* (eds. L. N. Robins, and B. Z. Locke). New York, Free Press.
- Schore, A. (1994). *Affect Regulation and the Origin of the Self*. Hillsdale NJ, Lawrence Erlbaum Associates Inc.
- Schore, A. (2003). *Affect Dysregulation and Disorders of the Self*. New York, Norton.
- Shenton, M. E., Chandler, C. D., Frumin, M., McCarley, R. W. (2001). A review of MRI findings in schizophrenia. *Schizophrenia Research*, **49**, 1–52.
- Szasz, T. (1960). The myth of mental illness. *American Psychologist*, **15**, 113–118.
- Szasz, T. (1974). *The Myth of Mental Illness*. London, Palladin.
- Thagard, P. (1996). The concept of disease: structure and change. *Communication and Cognition*, **29**, 445–478.
- Toomey, R., Faraone, S. V., Simpson, J. C., Tsung, M. T. (1998). Negative, positive and disorganized symptom dimension in schizophrenia, major depression, and bipolar disorder. *Journal of Nervous and Mental Disease*, **186**, 470–476.
- Van Os, J., Hanssen, M., Bijl, R. V., and Ravelli, A. (2000). Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophrenia Research*, **45**, 11–20.
- Walhberg, K. E., Wynne, L. C., and Oja, H. et al., (1997). Gene–environment interaction in vulnerability to schizophrenia: findings from the Finnish Adoptive Family Study of Schizophrenia. *American Journal of Psychiatry*, **154**, 355–362.
- Williams, B. A. O. (1985). *Ethics and the Limits of Philosophy*. London, HarperCollins.
- Williams, B. A. O. (1993). *Morality*. Cambridge, Cambridge University Press.
- Williams, J. B., Gibbon, M., and First, M. B. et al., (1992). The structured clinical interview for DSM-III-R (SCID): II. Multi-site test-retest reliability. *Archives of General Psychiatry*, **49**, 630–636.
- Yalom, I. D. (1970). *The Theory and Practice of Group Psychotherapy*. New York, HarperCollins.

