

Western models for mental health: A cautionary note

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Severe mental illness (psychosis; conditions in which the individual is said to in some sense lose touch with reality, and in which hallucinations and delusions – abnormal beliefs – are prominent symptoms) has a major impact on the well-being of individuals and the prosperity of nations. Individuals with severe mental illness are typically diagnosed as suffering from ‘schizophrenia’, ‘bipolar disorder’ or related conditions. These conditions are associated with a high risk of suicide, prolonged disability, loss of economic productivity, and very high costs to carers and governments (see Bentall, 2009).

Not surprisingly, in the developed world these conditions are the focus of well-resourced psychiatric services. In the second half of the 20th century, many new treatments were developed for patients with psychosis, including antipsychotic and other drug therapies (Healy, 2004), and also psychological treatments such as behavioural family therapy and cognitive behaviour therapy (Pilling et al., 2002). Over this period, an increasing number of people in the developed world have received psychiatric care. For example, more than ten per cent of adult US citizens are prescribed antidepressants annually (Olfson and Marcus, 2009) and, between 1997 and 2004, the number of people in that country receiving more powerful antipsychotic drugs rose from 2.2 million to 3.4 million (Moncrieff, 2008). Not surprisingly, in recent years health agencies in the developing world have increasingly embraced the Western psychiatric model on the assumption that it is an effective approach for addressing the burden of severe mental illness, sometimes making only limited attempts to adapt the model to local cultural conditions (Watters, 2010).

However, despite the introduction of modern drug and psychological treatments, there is very little evidence that outcomes for patients with severe mental illness have improved (Warner, 1985). Indeed, in the industrialised world, the number of people disabled by severe psychiatric disorders has climbed steadily since the end of World War II (Whitaker, 2005). There is even evidence that patients attending well-resourced psychiatric services in the industrialised nations have poorer outcomes than patients receiving minimal treatment in the developing world (Jablensky et al., 1992). These apparently paradoxical findings are in marked contrast to findings for physical conditions such as heart disease and cancer, for which outcomes have improved over time and are better in wealthy, industrialised nations (Bentall, 2009).

Of course there may be many reasons for the failure of modern psychiatry to deliver improvements in public mental health in the developed world. Perhaps psychotic disorders are just more difficult

to treat than other kinds of mental illness. Perhaps there are special kinds of stresses in the developed world, which make recovery more difficult despite the existence of effective treatments. An alternative possibility is that, despite widespread support, there is something fundamentally wrong with the Western biomedical approach. Here I briefly address two false assumptions made by the Western biomedical approach that go some way to explaining its failure.

Assumption 1

There are many different kinds of psychiatric disorder, which are qualitatively distinct from healthy functioning

Any rational scientific system requires a way of classifying the phenomena of interest. Modern psychiatry uses diagnostic concepts similar to those employed in physical medicine to differentiate between different kinds of psychiatric disorders. This approach was first developed by German researchers in the late 19th century, most notably by Emil Kraepelin (1856–1926). Their assumptions about the classification of mental illnesses have been carried forwards by modern psychiatrists, especially the authors of the third and subsequent editions of the American Psychiatric Association's *Diagnostic and Statistical Manual* (American Psychiatric Association, 1980, 1994), some of whom styled themselves as ‘neo Kraepelinians’ (Klerman, 1986). These assumptions included: (i) there is a clear dividing line between mental illness and healthy functioning; (ii) there are a number of discrete mental illnesses, for example ‘schizophrenia’ and ‘bipolar disorder’; and (iii) these disorders reflect dysfunctions of the brain that are largely inherited (Bentall, 2003).

It is hard to exaggerate the impact that this framework has had on psychiatric research and clinical practice. For example, with few exceptions researchers continue to compare individuals with one diagnosis (for example, schizophrenia defined according to the Diagnostic and Statistical Manual of Mental Disorders (DSM) or the similar International Classification of Diseases) with healthy individuals, on the assumption that the patients with the diagnosis have something pathological in common that separates them from the comparison group. Even a cursory inspection of the world's leading psychiatric journals will reveal that, in research of this kind, most effort has been directed towards identifying biological factors that are thought to be aetiologically significant (studies using brain scans and molecular genetics predominate) in patients.

And yet, this method of classifying psychiatric disorders is not supported by scientific evidence and has little value clinically.

Statistical studies of psychiatric symptoms show that they do not co-occur in the way that psychiatric manuals presume. Indeed, rather than breaking down into the two major categories of schizophrenia and bipolar disorder, the psychoses defy any kind of simple categorical structure and are probably best represented in terms of five independent symptom dimensions of positive symptoms (hallucinations and delusions), negative symptoms (social withdrawal and the inability to experience pleasure), cognitive disorganisation, depression and mania (a state of high excitability; Demjaha et al., 2009), perhaps within a superordinate single psychosis syndrome that encompasses both schizophrenia and bipolar patients (Reininghaus, Priebe and Bentall, 2012).

There are many further reasons for doubting the validity of traditional Kraepelinian diagnoses. For example, if schizophrenia and bipolar disorder really were separate conditions, we would expect them to run true in families (that is, the first-degree relatives of people with schizophrenia would be at elevated risk of suffering from schizophrenia and not bipolar disorder; the reverse would be true for the first degree relatives of bipolar patients), but this is not the case (Lichtenstein et al., 2009). We would also expect the two conditions to respond to different treatments but, again, this turns out not to be the case in practice (Johnstone, Crow, Frith and Owens, 1988; Tamminga and Davis, 2007). Indeed, these days patients with psychosis tend to be treated with antipsychotic drugs, whatever their diagnosis.

As we have seen, the conventional biomedical approach also assumes that there is a clear dividing line between severe mental illness and healthy functioning. Admissions to psychiatric services suggest that, in those countries for which adequate data exists, the proportion of the population that at some point suffers from schizophrenia is less than one per cent (Jablensky, 2000) with about three per cent of people experiencing a psychosis of any kind (Perala et al., 2007). However, these figures almost certainly severely underestimate the extent to which psychotic symptoms, such as hallucinations and delusions, are experienced within the world's population, with estimates from epidemiological surveys suggesting that nearer ten per cent are affected (Poulton et al., 2000; Shevlin, Dorahy and Adamson, 2007; van Os, Hanssen, Bijl and Ravelli, 2000). Hence, there are many people who experience psychosis who do not seek, or fail to find, psychiatric treatment, most often because their psychotic experiences are not disturbing and they are coping well with life. These findings are consistent with dimensional models, which assume that psychotic symptoms lie at the extreme ends of continua of human experience (Claridge and Davis, 2003; Linscott and van Os, 2010). Hence, it seems that there is no clear dividing line between mental illness and normal functioning.

Assumption 2

The causes of severe mental illness are largely biological

Biomedical psychiatry assumes that the causes of psychiatric disorder are largely endogenous (internal) – perhaps some kind of biochemical fault in the central nervous system which is usually believed to be genetic in origin. Hence, the lion's share of research into the aetiology of 'schizophrenia' or 'bipolar disorder' focuses either on neural processes or on genetics. It is easy to be impressed by some of this research, which often employs dazzling

technologies such as functional magnetic resonance imaging or molecular techniques for sequencing genes. It is often forgotten that these technologies are prone to error and, indeed, are often less reliable than less dazzling and more mundane methods of research. For example, statistical analyses have shown that neuroimaging studies are particularly prone to false positive reports ('type-1 errors', in the jargon of statistics), making replication of research findings problematic (Button et al., 2013). The same point has been made about the molecular genetics of psychiatric disorders, where failed replications have been commonplace (Crow, 2008).

Psychiatric geneticists have justified the hunt for 'schizophrenia genes' and 'bipolar genes' on the basis that these disorders are highly heritable (figures of 70–80 per cent heritable are typically quoted). Researchers have typically assumed that these estimates can be treated as a gene/environment ratio, showing that psychosis is largely caused by genes and little influenced by environmental risk factors. However, this is a profoundly mistaken understanding of what these estimates mean (see Bentall, 2009, for a detailed explanation of why this is so).

Heritability estimates are, essentially, correlations between the entire genome and diagnoses, and refer to *populations* and not individuals. They are usually estimated from studies of monozygotic (MZ; identical) and dizygotic (DZ; fraternal) twins. MZ twins have identical DNA whereas DZ twins do not (they are simply siblings who happen to be born at the same time). Hence, if the rate of concordance for a disorder (twins are said to be concordant if they are both ill) is greater in MZ than DZ twins, this is taken as evidence that genes play a role in the disorder.

The assumptions and methods employed by researchers conducting twin studies have been much criticised for reasons which cannot be discussed here because of limited space (see Joseph, 2003). However, even if the data from these studies can be taken at face value, high heritability estimates do not preclude strong environmental effects. For example, if variance in the environment is low (imagine a world in which everyone smokes exactly 20 cigarettes a day) heritability must be high (in such a world, only genetic factors can determine who gets lung cancer and who does not). Hence, to take a real-life example, in the USA intelligence is highly heritable in middle-class families (in which there is very little environmental variation – all children are encouraged to study) but has very low heritability in working-class families (which are more variable; Turkheimer, Haley, Waldron, D'Onofrio, and Gottesman, 2003). Heritability estimates are also undermined by gene x environment interactions (which occur, for example, when people with particular genes are exposed to particular environments which, in turn, cause illness). In these circumstances, environmental influences are always under-estimated (Dickins and Flynn, 2001). The bottom line is that environmental effects cannot be estimated by subtracting genetic effects (that is, just because heritability is 70 per cent, it does not mean that only 30 per cent of the cause is environmental) and have to be studied in their own right.

In fact, there is substantial research evidence that environmental factors, especially during childhood, play an important causal role in severe mental illness. For example, children are much more likely to become psychotic in adult life if they are raised in conditions of social deprivation – e.g. in urban environments (Pedersen and Mortensen, 2001; Wicks, Hjern and Daman, 2010) or by parents

who communicate with them in a dysfunctional way (Wahlberg et al., 1997). In my own work, I have recently conducted a meta-analysis (statistical synthesis of data from all relevant research studies) of the effects of childhood trauma (sexual and physical abuse, bullying by peers and separation from parents) on risk of psychosis in adulthood (Varese et al., 2012), finding that a child who has experienced trauma is approximately three times more likely to become psychotic than one who has not, with a dose–response relationship (so that a child who is exposed to multiple traumas has an especially high risk).

Conclusions

The Western biomedical model attributes severe mental illness to endogenous disease processes in the central nervous system. This model leads researchers to look for abnormal genes and brain function in patients in the hope that discovery of these processes will lead to effective treatments. In fact, the drug treatments currently used to treat psychosis were mostly discovered adventitiously (Healy, 2004) and recent research has shown that these treatments are nowhere near as effective as was once thought (Kirsch et al., 2008; Lepping, Sambhi, Whittington, Lane and Poole, 2011). Hence, the whole edifice of biomedical psychiatry seems poorly founded.

What are we to do? Elsewhere, I have argued that we are more likely to make progress in understanding severe mental illness if we abandon conventional diagnoses and instead try to understand the mechanisms underlying specific symptoms such as hallucinations and delusions (Bentall, 2003). Recently, some pharmaceutical researchers have reluctantly acknowledged that current approaches to psychiatric diagnosis have impeded the development of effective treatments and have suggested a similar approach (Fibiger, 2012). This approach is often taken by psychologists developing novel psychological treatments for psychosis and, as I have already noted, there is emerging evidence that these can be effective, although they are certainly not a panacea (Wykes, Steel, Everitt and Tarrier, 2008).

In the meantime, an important implication of the above analysis is that, in the long term, we may have more success in preventing severe mental illness than we have had in its treatment. After all, environmental factors can be manipulated whereas genes cannot. We need to find ways of making our cities less psychologically toxic and ways of ensuring that all children have benign childhoods. Just as the physical health of nations has been improved by eliminating or at least ameliorating environmental risk factors such as pollution and smoking, so too, governments need to consider how they can help foster psycho-civilised societies in which mental health is guaranteed for all citizens. This is a realistic ambition, but we need the vision and the will to work towards it.

Endnotes

1 World Health Organization, 1992.

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