A psychological model of mental disorder

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Abstract

A coherent conceptualisation of the role of psychological factors is of great importance in understanding mental disorder. Academic papers and professional reports alluding to psychological models of the aetiology of mental disorder are becoming increasingly common, and there is evidence of a marked policy shift towards the provision of psychological therapies and interventions.

This paper discusses the relationship between biological, social and psychological factors in the causation and treatment of mental disorder. It argues that simple biological reductionism is not scientifically justified, and also that the specific role of psychological processes within the biopsychosocial model requires further elaboration. The biopsychosocial model is usually interpreted as implying that biological, social and psychological factors are co-equal partners in the aetiology of mental disorder. The psychological model of mental disorder presented here suggests that disruption or dysfunction in psychological processes is a final common pathway in the development of mental disorder. These processes include, but are not limited to, cognitive processes. The model proposes that biological and environmental factors, together with a person's personal experiences, lead to mental disorder through their conjoint effects on these psychological processes.

Implications for research, interventions and policy are discussed.

192 words

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Introduction

The past few years have seen a massive investment of research and development efforts in the field of mental disorder. It is estimated that in the UK, the National Health Service spends about £219 million per year on mental health research[1], and mental disorder has been estimated to cost the state £77 billion per year[2].

On July 25, 1989, President George Bush designated the 1990s the 'Decade of the Brain'. The European Community Council of Ministers[3] swiftly followed. Recent advances in imaging techniques such as computed tomography (CT), magnetic resonance imaging (MRI) and positron emission tomography (PET) have been hailed as offering great potential for detailed neuroanatomical investigations of many disorders[4]. Research into biological aspects of mental disorder is welcome. However it is important that this research is properly integrated with psychological and social accounts of mental disorder. Some observers point to a threat of a retrenchment into a reductionist, biomedical approach to mental disorder[5]. It has been argued that diagnostic and etiological accounts stemming from a simple biomedical approach will be partial and scientifically inadequate[6]. Ill-informed reductionist accounts may lead readers to an unfortunate perception that mental health care is both lacking in humanistic sensibilities and dehumanising[7].

Psychological approaches to mental disorder offer alternative perspectives and can also be integrated with biological perspectives. Indeed, this paper will suggest that a comprehensive psychological model of mental disorder can offer a coherent and

assertive alternative to purely reductionist biological accounts that nevertheless incorporates biological research.

Most biomedical theories and most biomedical interventions in mental disorder explicitly or implicitly relate to synaptic or intracellular processes[8]. Thus abnormalities in neurotransmitter functioning are implicated in theoretical models and drug treatments (selective serotonin re-uptake inhibitors etc) target synaptic neurotransmitters in the search for effective treatment. In contrast, psychological approaches focus on associative networks, based in the neural substrate, but developed through learning and relying on theories of conditioning, perception, appraisal and belief-formation, propositional and implicational encoding, mental models of the world, internalised schemas of relationships etc. Psychological models of mental disorder, therefore, address different kinds of mechanisms, but also strive to encompass more than the mere mechanics of any individual system and to look at interactions and interrelationships.

Over the past few years a number of reports by professional bodies, strategy documents from policy makers and proposed changes in legislation all stress the role of psychologists and of psychological perspectives in mental health. As an example, the first national clinical guidance issued by the UK's National Institute for Clinical Excellence concerned the frontline treatment of schizophrenia[9]. Amongst other important comments, this document recommended that "100%" of people in receipt of the diagnosis of schizophrenia should be offered cognitive behavioural therapy (p55). Recent academic reviews of the role of psychosocial influences on mental illnesses and psychotic experiences[10] and major grant-funded randomised controlled trials have repeatedly demonstrated the effectiveness of psychological therapeutic approaches in a range of mental health problems[11, 12, 13].

Psychological formulation is the summation and integration of the knowledge that is acquired through the assessment process[14]. Psychological formulations attempt to explain why people are experiencing difficulties. They usually consist of a list of problems and possible psychological reasons for these[15]. Typically, formulations examine the events of people's lives, and how the individuals have interpreted and reacted to these. Formulations are hypotheses about the nature and origin of problems, which are tested out over time[16], and therefore tend to change over the course of both assessment and therapy. Psychological case formulations are complex and may comprise a number of provisional hypotheses, based on a large variety of psychological theories, each drawing on scientific research.

The use of psychological formulations is not synonymous with being a clinical psychologist, and is certainly not incompatible with being a psychiatrist. Many individual clinical psychologists may fail to provide psychological formulations. Many medically-trained psychiatrists welcome formulation; either as an adjunct to diagnosis – the iconic DSM-IV[17] suggests that diagnosis is only a start – or, as some psychiatrists propose, as an alternative to diagnosis[18]. Psychological models of mental disorder could inform the practice of all workers in the mental health services, but they should be coherent. It is axiomatic that psychologists welcome the inclusion of specifically psychological elements in psychiatric formulations. The questions are how that should be done and what constitute coherent psychological models of mental disorder.

It is worthwhile briefly mentioning the biopsychosocial model in psychiatry. This is primarily associated with the work of George Engel[7], who attempted to provide a scientific account of mental disorder that could challenge a reductionist biological account. The biopsychosocial model therefore suggested that mental

disorder emerges from a human system that has both physical elements (a biological nervous system) and psychosocial elements (relationships, family, community and wider society)[19, 20].

The biopsychosocial model was widely adopted[21, 22, 23]. Inevitable micro-historical pressures such as a professional revolt against so-called antipsychiatrists[24, 25, 26] has meant that some of the gloss has worn off the biopsychosocial model. Antipsychiatry remains vocal[27, 18, 28] while, conversely, biodeterminist writings are also common[8]. Wing, in particular, appeared to feel the need to defend diagnosis, to assert the biomedical basis of psychiatric disorders and the utility of a 'diagnose and treat' approach[29].

The emphasis within the biopsychosocial model on social and psychological perspectives in addition to biological aspects of mental disorder may be welcome. However, consideration needs to be given to how, in each case, the elements – bio-, psycho-, and social- – relate one to another. The biopsychosocial model is, or should be, more than a simple statement that these three aspects should be included in a formulation. In particular, it has been argued that the biopsychosocial model has been interpreted in practice as reserving a dominant position for biomedical approaches[5], where indeed social and psychological factors are acknowledged and given prominence, but are considered to be moderators of the direct causal role of biological processes. This 'primacy' of biomedical causation[30] has been cited as an argument for the professional superiority of one profession over others[31]. This wider sociological debate over the implications of particular epistemological approaches has, of course, been a source of some tension[32].

This paper argues that the role of psychological and social processes in mental disorder requires further attention. The biopsychosocial model does not address the

issues of the nature of the interrelationships between elements. Importantly, it fails to address issues related to the different status and nature of the different elements – the unresolved issue of 'primacy'. The nature of psychological factors itself needs further attention. This includes a consideration of the different functions psychological factors can play in different models of mental disorders: as causes, as symptoms and as possible therapeutic factors.

Such consideration may, paradoxically, rationalise and contextualise the role of biochemical aspects. The biopsychosocial model fails properly to address biological accounts of mental disorder if it cannot relate, for example, how monoamine abnormalities in depression[33] relate to findings of low self-esteem[34] and negative thinking patterns[35, 36] and findings of greatly increased incidences of mental disorder in disadvantaged groups[37]. A coherent model of the links between these findings – which should emerge from a coherent psychological model of mental disorder – should also ensure that biological approaches to mental disorder are given proper regard.

What, then, is a psychological conceptualisation of mental disorder? Is there a coherent, simple, model of mental health that simultaneously elucidates the psychological perspective and contrasts it with that of other approaches? What would a generic psychological model of mental disorder look like? Could a coherent account of the role of psychological factors in mental disorder also help the development of an integrative model of mental disorder; in which multiple causal factors, different symptomatic markers and different therapeutic elements are all appropriately addressed?

Multiple causes of mental disorder

The biopsychosocial model is based on is the notion of multiple simultaneous causes of mental disorder. Clinical and research evidence supports this assumption, as will be discussed below. Any proposed generic psychological model must acknowledge this. One can imagine something along the lines of a theoretical multiple regression equation, with mental ill health as the dependent (predicted variable). One might even imagine measuring such disorder on a numeric scale ranging from zero to 100 or perfect mental health. One can imagine further three possible independent or predictor variables. The biopsychosocial model, and the model proposed here, includes multitude causes – multitude predictors. Nevertheless, one can imagine for the purposes of this argument three main classes of predictor variables (biological, social and circumstantial) as illustrated in Figure 1a. It is worth noting that these classes of causal or predictor variables are slightly different from the components of the biopsychosocial model. As will be clarified below, this reflects two important modifications to the biopsychosocial model: the distinction between psychological processes and personally significant life events, and the specific status given to the disruption of psychological processes as a final common pathway to mental disorder.

FIGURE 1a ABOUT HERE

All of these classes of variables are causally related to mental ill health. Clearly, biological factors are important in mental health. Links between genetics, biochemical abnormalities, neuroanatomical abnormalities or merely the structure and functioning of the physical brain and mental disorder have been studied extensively.

Some authors have argued that, while biological variables have explanatory power in cases of mental disorder, psychosocial aspects merely account for human experiences that do not amount to clinically significant disorders[30].

It is beyond the scope of this paper to discuss all possible biological factors causal in all possible forms of mental disorder. It is worth noting that these cannot be dismissed. As just one example, a variety of problems from unipolar depression[38] through bipolar disorder[39] to schizophrenia[40] have been associated reliably with genetic heritability. In the hypothetical multiple regression equation suggested above, therefore, it is likely that an imaginary predictor variable capturing the population's biomedical variance would be a significant predictor of variance in mental ill health.

Equally, we have evidence that social and environmental factors contribute to mental disorder. Again, the number of studies is enormous, but there are clear links between poor housing[41] and poverty and unemployment[42] and environmental stress and the expression of a wide range of mental health problems. In simple terms there is considerable evidence of an association between mental disorder and the experience of poverty and deprivation[43, 44, 45]. In our hypothetical example, a general and portmanteau measure of disadvantageous social environment would clearly be a significant predictor of variance in mental ill health.

An environmental, or social environmental, causal variable can be seen as occupying the space of psychiatric sociology – examining and contextualising social and environmental causes of mental disorder[46]. When people refer to psychological issues, they sometimes refer to the effects of potentially life-shaping experiences that impact on mental health – childhood sexual abuse[47], bullying[48], attachment relations with parents[49], assault[50] and all other major and minor interpersonal experiences.

Although these are disparate kinds of event, it remains true that life events in general contribute to mental disorder[51, 52].

Because of the breadth of these experiences, it may be difficult to capture them all on one hypothetical dimension. Nevertheless, in our hypothetical multiple regression equation, again, it is likely that an imaginary predictor variable capturing these potentially damaging cumulative life events would also be a significant predictor of variance in mental disorder. This represents one important meaning of the term 'psychological' as it is used in this context – as a reference to the causal role of psychosocial factors in the development of mental disorders.

A simple statement that biological factors, social factors and life events all contribute to the development of mental ill health does not take us further than the biopsychosocial model. Two further important further points are necessary, reflecting the specific psychological nature of the model proposed here.

First, people from different professional and academic backgrounds may well differ as to their hypotheses about the relative importance of the putative predictor variables.

One example of this is the current interest in genetics. As commented above, genetic aspects of mental disorder are important. However, the apparently bare facts themselves appear frequently to be overstated, and the concept of heritability itself can be misleading when applied to psychological phenomena. For example, the heritability of psychotic disorders has been quoted as being as high as .85[53]. This figure is commonly taken to imply that 85% of the variance in the presentation of the symptoms of psychosis can be statistically attributed to the variance in the population's genome. This may be misleading. Rutter[54] cogently argues that psychosocial, environmental and developmental influences on mental disorder are

significant. He points out that some of the assumptions behind molecular genetics research do not translate easily into lay language (for example, genetic effects plus environmental effects do not necessarily sum to 100%). This, Rutter argues, may lead to subtly but important misrepresentations of biological findings when discussed in contexts such as the present discussion. It is not necessarily correct, for instance, to suggest that social background, life events and psychological factors together could explain only the 'remaining' 15% of the variance in the presentation of the symptoms of psychosis.

Biological, social, circumstantial and psychological variables also interact. Personal vulnerability factors such as neuroticism[55] may make a person more emotionally responsive to life events. Similarly, the social support buffer hypothesis[56] suggests that the level of a person's available social support buffers the impact of environmental stressors on mental health. Thus, for two individuals experiencing stressful events, the person with the greatest level of social support will experience lower levels of mental disorder. A similar set of interactions may explain the often commented upon fact that the concordance rates for monozygotic twins for no mental disorder is 100%[57]. So a biomedical dominance of explanatory models of mental disorder is not inevitable. Social factors and the influence of life events may be just as important.

The psychological consequences of biological abnormalities

A discussion of the role of biological factors in mental disorder in no way undermines the psychological model proposed here. The role of biological variables are acknowledged, but placed into a proper relationship with respect to other factors. In the case of hallucinations, there is a fascinating story emerging. Biologically, there

is considerable evidence of a genetic element in schizophrenia[40]. Schizophrenia in general, psychotic symptoms more specifically and hallucinations in particular have been linked with cerebral lateralisation[58]. It has been claimed that people who hear voices are more likely to have poorly lateralised cerebral hemispheres, more specifically still that the language areas of the brain appear to be less lateralised in people who hear voices[59]. It is entirely credible that biochemical or neuroanatomical or genetic abnormalities lead to poor lateralisation of language processing and hence to the problems associated with a diagnosis of schizophrenia[60]. Since one key task in neural language processing is presumably the identification and localisation of perceptual experiences, this failure of lateralisation seems to lead to problems in the discrimination of voice (heard) from other forms of cognition (thought or remembered). Psychologically, this is a failure or a disruption of a psychological process – source monitoring or source attribution or reality monitoring[61].

Biological factors cannot therefore be excluded, or even minimised, in the development of hallucinations. Few people would dispute that auditory hallucinations are psychological phenomena. This analysis suggests that hearing voices cannot be thought of as a psychological phenomenon only in terms of effect or nature, but also in terms of process. The final, inevitable, pathway to hearing disembodied voices is the misattribution of the source of percepts. This is a psychological process. It is influenced by biological factors, but it is equally influenced by social environmental factors and the important events in a person's learning history. The point of this example is not to discuss cerebral asymmetry in hallucinations, but to illustrate that a plausible biological pathway from genetics, through neurology to behaviour necessarily implicates psychological processes. Biological factors influence mental disorder through their impact on psychological processes.

Similar augments can be advanced for the relationship between environmental or social factors and the disruption of psychological processes. Living in poor social circumstances leads to mental disorder because such social circumstances impact upon psychological processes. Life events impact on mental health through their impact upon psychological processes. These arguments also apply to a wide range of problems under the general aegis of mental ill health.

Abnormalities in serotonin metabolism are implicated in depression[62]. For example a tryptophan-reducing (serotonin-reducing) diet can induce depression[63]. In the psychological model of mental disorder proposed here, this association is accepted, but the causal role of biology is effected through the disruption of psychological processes. Thus a reduction in dietary tryptophan leads to a reduction in available cerebral serotonin. Serotonin is implicated in the neurological mechanisms supporting a number of important appraisal processes. It is entirely plausible to suggest that these appraisals will include the ways people see themselves, their world and their future – the negative cognitive triad of the cognitive model of depression[35]. Thus the biological tryptophan-serotonin system could indeed lead to depression, but through psychological processes.

Similar arguments place the disruption of psychological processes in a central role when the effects of social or experiential factors are considered. In the case of social factors, a psychological model of mental disorder based on the principle that mental disorder arises from the disturbance or disruption of psychological processes accepts that living in disadvantaged social circumstances does indeed lead to problems such as depression. In this case, however, poverty and social deprivation appears to lead to disillusionment, hopelessness and learned helplessness; a realisation that there is little or nothing that one can do to improve or change one's lot in life, and

perhaps even that one's actions have no effect or purpose[64, 65, 66]. Depression is of course a consequence, but is a direct consequence of the disruption of psychological processes.

And finally the same applies to experience. Of course being assaulted by one's parents leads to problems (frequently, again, depression). But psychologists would argue that the association between cause (assault) and the effect (mental disorder) is again mediated by the disruption or malformation of psychological processes. In the case of the impact of sexual, emotional or physical abuse, this is likely to impact on the ways in which the children (and later the adults) appraise themselves, the important people in their lives, their actions and the consequences of their actions and the ways in which relationships and social intercourse should be governed – their cognitive schemas[67]. Indeed, there is convincing evidence[68] that assaults that mirror childhood events reactivate such cognitive schemas with terrible consequences.

It is important, here, to stress that these psychological processes are not just cognitive. Cognitive processes are important (and in vogue) and the impacts of writers such as Beck[35], Seligman[69] and Young[67] are undeniable. Disruptions or disturbances in psychological processes other than cognitive processes have, however, been stressed by other psychologists and psychiatrists. The phenomenon of 'learned helplessness' has been referred to earlier, and is closely associated with Seligman's cognitive approach[69], but learned helplessness itself was initially analysed as a behavioural phenomenon[70]. Purely behavioural models of depression have a distinguished history[71] and remain highly regarded[72], and behavioural elements are a key part of conventional cognitive behavioural therapy.

Psychodynamic approaches – which, within the model presented here, invoke the disruption or disturbance of object relations, sexual drives or other intrapsychic

events under the legend 'psychological processes' – similarly have a distinguished history, have been massively influential on modern psychology and are currently a core part of the practice and ethos of clinical psychologists[73]. In the example of depressed mood used here, the psychodynamic notions of depression as involving, for instance, disrupted object relations[74] or involuted anger remain respectable elements of psychological formulations[75]. In the case of personality disorders, although most recent psychological accounts of personality problems cite maladaptive cognitive schemata concerning social relationships[76, 68], other writers have cited failures to integrate personality structure[77], and disruptions of psychodynamic processes related to fundamental object relations[78]. In the model presented here, therefore, disruption of psychological process is certainly not limited to disruption of cognitive process.

Psychological processes

A generic psychological model of mental disorder proposes that all of these three putative causes have their effects by their actions in disrupting or disturbing psychological processes. This relationship applies not only in the case of biological factors, but equally for social and experiential factors. This central conceptual point is illustrated in Figure 1b.

FIGURE 1b ABOUT HERE

A truly psychological approach has always separated events from the interpretation of events. The model proposed here neatly separates events from the

psychological processes that interpret, buffer and act consequentially upon these events. Like the biopsychosocial model, it also attempts to address the interactions between these classes of causal variables. Biological, social and circumstantial factors are all important and are all presumed to interact. Importantly, however, in this model it is the conjoint impact of these interacting factors on psychological processes that lead to mental disorder.

This, important, point is worth elaborating. The model developed here implies not only that the common symptoms of psychiatric classifications are psychological in nature (that hallucinations, depression, anxiety etc are themselves psychological phenomena), but also that psychological processes – or rather disturbances or dysfunctions in psychological processes – are necessarily responsible for shaping the nature, extent and type of the phenomena described collectively as mental disorder. This means that, as in the biopsychosocial model, multiple causal factors are implicated. Thus, in a hypothetical consideration of depression, several distal causal factors may be salient, and several key psychological processes may be implicated. It is possible to hypothesise that social deprivation and childhood maltreatment, genetic vulnerability and biochemical insult may all contribute to the onset of depression.

These factors may all impinge on psychological processes of self-esteem maintenance, self-efficacy beliefs and expectancies of reward (for example). They may implicate neuroanatomical systems responsible for reward and reinforcement, autobiographical memory and social cognition, and they may involve neuroanatomical systems that involve serotonin (as well, of course, as other neurotransmitters). Depression, in the model presented here, is characterised neither as a direct consequence of the neuroanatomical mechanisms, nor of the contributing social circumstances. Rather, these neuroanatomical mechanisms and circumstances

are viewed as being important influences on the psychological mechanisms of reward and reinforcement, social cognition and maintenance of self-esteem. And it is dysfunctions or disturbances of the se psychological processes that are seen as ultimately leading to the mental disorder – depression.

This argument can be applied to hallucinations. Abnormalities in the poorly-lateralised speech areas of the brain, probably involving dopaminergic systems, and potential abnormalities in frontal functioning could, theoretically, lead to difficulties in source-monitoring (detecting whether particular precepts are internal or external in origin). Social circumstances similar to those that are risk factors for other mental disorders may, in this model, interact with these biological variables in a number of complex ways – but most likely through the generation of emotive negative intrusive thoughts. Once again, however, in the model presented here, the experience of hallucination is seen as a product of a psychological act – the misattribution of the origin of a percept. No one would deny that the distal causative factors could (at the very least hypothetically) affect this source-monitoring process. In the model presented here, however, these distal causal factors do not 'cause hallucinations', they perturb the psychological processes, and it is the perturbations in these psychological processes that lead to – that 'cause' – the phenomena that are labelled mental disorder.

And, as a third illustration, the same principle holds true in personality disorder. Innate or heritable individual differences in personality trait (for instance increased levels of the trait of neuroticism) may be exacerbated by events (for example experiencing childhood abuse) to precipitate more extreme personality dysfunction in later life. In the model presented here, however, it would be inappropriate to say that either the biological traits, or the life events or even the

combination of these has produced or caused a personality disorder. Rather, in the present model, this combination of distal circumstances has profoundly affected how the person perceives themselves, their world and their relationship with others. It is this element, the perturbation of psychological process, which leads to mental ill health.

Weaknesses and possible criticisms of such a model

Perspective

Philosophers of science consistently maintain that, even in supposedly objective science, the interests and allegiances of the researcher may shape claims of causality[79]. This is particularly pertinent in the case of mental health[80]. If a person is 'depressed' and has 'low self-esteem', it is easy to see two professionals maintaining opposing points of view: on the one hand the depression is viewed as having caused the lowered self-esteem, on the other the depression is seen as a consequence of the negative self-evaluation. Many philosophers of science as applied to psychiatry conclude that 'truth' is dependent on perspective[81].

In the case of the model presented here, of course, psychological processes are given centre stage. It could be argued that this reflects nothing more than a perspective focussed upon psychology. That, alone, would not render the model invalid. A coherent statement of the nature of psychological causation, whether or not there were other perspectives, would be entirely appropriate. But the model presented here makes a further, more specific, claim. It states that disruption of psychological processes is a necessary final element in the origin of mental disorder. This is a testable hypothesis: if true, all mental disorder will be associated with disruption of mental processes, and such disruption will mediate the effects of biological factors on

observed symptoms. And, as Karl Popper[79] suggested, such testable hypotheses separate subjectivity from science.

'Truly organic' disorders

While some readers may allow that a model such as this could fit relatively well to disorders such as anxiety and depression, it may be claimed that there are 'truly organic' disorders that offer a more direct challenge. Disorders such as Down's syndrome and phenylketonuria are unequivocally genetic in origin. In these cases, could it genuinely be argued that psychological factors are a final, necessary, common pathway? In addition to cause, it could be argued that the interventions are social and medical rather than predominately psychological. Therapy does not ameliorate the disorders, medical care, and, particularly, social care is important.

Moreover, in the case of phenylketonuria, the consequences of the undoubted genetic cause of the condition can be (largely) ameliorated by the social (or social and biological) manipulation of removing all foods containing phenylalanine. How, then, could psychological factors intrude as a final common element?

First, the model presented here does not claim that 'therapy' is the touchstone of a psychological model. What care services must do is perform a proper formulation of the problem and address the observed functional elements. Clearly the most therapeutic intervention should be employed. But this does not necessarily impute causality. But more importantly, why might Down's syndrome and phenylketonuria be considered at all in a discussion of mental disorder? There are many genetic disorders. What characterises these two disorders is the consequence on mental processes, of the genetic problems. If phenylketonuria did not affect learning and behaviour, it would still be a genetic disorder, but it would not be a mental disorder.

Cystic fibrosis is a genetic disorder with a complex therapeutic imperative, but it is not a mental disorder.

The model presented here does not rely on the refutation of any genetic component to a given mental disorder for the psychological component to be key – quite the reverse. It does not suggest that, equally, that all mental disorders should be amenable to psychotherapy. What it suggests is that a mental disorder is characterised by the disruption of psychological process. Down's syndrome and phenylketonuria occur because of genetic abnormalities. But it is clear that these genetic abnormalities (if not addressed through diet, of course) will disrupt psychological processes, especially learning and information processing. So, in the case of Down's syndrome and phenylketonuria, biological factors are causal and social interventions are the treatments of choice. Nevertheless, these interventions are necessary precisely because the organic consequences of the genetic abnormalities disrupt the patients' psychological processes.

Biology as a final common pathway

In an influential paper, Eric Kandel[82] reaffirmed the biological basis of psychiatry. In that paper, Kandel proposed a model of psychiatry that is, in essence, diametrically opposed to the model presented here. Kandel not only argued that organic or biological factors were important in most forms of mental disorder, but more fundamentally that alterations in biological functioning (as opposed to psychological functioning) are the final common pathway for mental disorder and indeed therapy.

Kandel emphasises (as the model presented here emphasises) how biological factors can impact upon psychological functioning – emotions, cognitions and

behaviour. But he also emphasises the brain-based nature of psychology. He stresses how all emotions, all cognitions and all behaviour depends on brain functioning. Kandel points out that in a real, scientific, sense, all learning involves structural changes in the brain – changes in synaptic responsively at the very least, and possibly more gross neuroanatomical change. He therefore logically deduces that any psychological causation of mental disorder must be organic in essence, and any therapy, even psychotherapy, must achieve its effect through biology. Kandel not only argues (as the present model argues) that biological changes affect psychological processes, he argues that learned patterns of behaviour or self-evaluative schemata learned in childhood reflect physical changes in the neural associative networks. He moreover argues that any re-learning occurring during therapy represents changes in these neuroanatomical associative networks.

Clearly, this analysis must be true on one level. Any learning must, at the molecular and synaptic level, be based on biological changes (the brain is, of course, involved). But this is an intellectually trivial type of argument. Since all learning – all human behaviour – is dependent on the functioning of the brain, it is redundant to suggest that the brain must be involved. An alternative approach might suggest that biological factors underpin all forms of associative learning, and therefore each particular learned association. To associate a caress with comfort and a sense of security is learned, and that learning has a biological substantiation in the brain. Equally, perhaps for someone who has survived abuse in childhood, a caress may be associated with fear, powerlessness and self-loathing. Again this associative learning will necessarily be based on biological substrates. But the difference between learning to associate a caress with fear on the one hand and contentment on the other cannot best be explained in terms of biological factors.

Other authors may go further than Kandel. It may be argued that all psychological concepts will disappear from the psychiatric lexicon as phlogiston has disappeared from physics – as we understand the neural basis of behaviour, we will have no need for the notion of psychology[83]. These arguments have been made before, and again there are three main refutations. First, the available data suggest that psychological factors (for example the presence or absence of certain dysfunctional beliefs) predict variance in the observed symptoms of mental disorder. It is important, intellectually and practically, to understand the links between these variables and the biological substrate, but their predictive power indicates that, in any meaningful sense, they exist. Second, psychological variables implicated in mental disorder are no less dependent on neurological processes than are the psychological elements of normal life: competition, love, honour, guilt, etc. It may be the case that fundamentalist neurological theories of mental disorder would wish to explain away these concepts as mental phlogiston, but few others share this ambition.

But most centrally, many commentators[84, 85, 86] have addressed this issue by pointing out that mental disorders are, in their essence, human rather than brain phenomena. Reductive terms fail to capture the essence of human experience because it is more than the sum of its biologic parts.

A pluralistic model

In the face of coherent arguments that biology serves as a 'final common pathway' to mental disorder, and similar social model of mental disorder[23], it may be argued that a pluralistic model of mental disorder – not a psychological model – is needed. This may be true. It is not the purpose of the present paper. Kandel[82]

presented a coherent and valuable biological model of mental disorder. This is a complement.

The impetus for the development of the model presented here was a frustration with the biopsychosocial model. Most descriptions of the biopsychosocial model also fail to address the causal interrelatedness of these variables – biology, the social environment, life events and psychological processes are all important, but they are not independent factors. Consequently, a pluralistic model that emphasised a false equivalence of these elements would have the benefits of the original biopsychosocial model, but all its flaws as well.

Finally, of course, the model presented here is integrative. Biological and social factors are properly acknowledged. It would be pleasing to hope that this model might itself be seen as pluralistic. The model presented in this paper, however, does place central emphasis on psychology. If that fact were to prevent this model from being regarded as pluralistic, that would be unfortunate. Ultimately, however, pluralism may emerge from a concurrent reading of models such as that proposed by Kandel and models such as that presented here.

Implications of such a model

Conceptualisation

Models such as the one presented here are valid if they are useful. It has been powerfully argued[84, 87, 88] that pragmatism should be the benchmark of evaluating theoretical models of mental disorder. This model might be useful in guiding thought. It offers a conceptual model that allows clinicians to separate and integrate multiple of

causes of mental disorder. Rather than proposing a simple 'biological cause and psychological symptom' approach, this model separates causes from mediating processes from effects. The model presented here has clear similarities to Padesky and Greenberger's "five factor model", widely used in cognitive therapy training[89]. That model proposed five important elements in mental disorder – environment, cognition, affect, behaviour and physiology. It contends that a full understanding of these five factors is vital for a complete formulation. The separation of affective, behavioural and cognitive aspects of 'psychology' within this model is particularly important. The weakness of the five-factor model, however, is that it fails to specify the nature of the interrelationships between these aspects.

In the biopsychosocial model, although it is expected that all three elements contribute to the experience of mental disorder, it is not a necessary logical conclusion that psychological factors are central. The model proposed here is different. Even if, as some biological psychiatrists might argue, biological factors account for the majority of the variance in the occurrence of a particular form of mental disorder – for instance if one contends that neurodevelopmental abnormalities cause schizophrenia – the model presented here would retain the disruption of psychological processes as a logically necessary final step. This is not a necessary conclusion from the traditional biopsychosocial model.

But, although it may be interesting to discuss conceptual issues, models are only worthy of real attention if they serve practical functions. Indeed, one of the criticisms of the biopsychosocial model itself is that it fails to lead to practical answers. But the model presented here does have practical consequences.

Research

In terms of research effort, the model presented here might be useful. Firstly, this model is, inter alia, a suggestion that psychology is key. Research into psychological factors in mental disorder should, it would follow, be prioritised.

But it might also be wise to structure research efforts more thoughtfully. Research into specific causes of mental disorder is vital and important. Research, for instance, might be conducted into the biological influences on source monitoring and simultaneously into the relationships between source monitoring and the experience of hallucinations. At the same time, we might investigate other (non-biological) influences on source monitoring, and other psychological processes implicated in hallucinations and other phenomena of mental disorder. For instance, there is evidence that disruption of executive thought processes impinge on thought disorder, considered a psychotic phenomenon[90]. Such a strategy would not contradict existing research programmes, but might offer a rational conceptual structure.

Paradoxically, the model of mental disorder proposed here may help explicate the mode of action of biological factors. For example, as mentioned above, abnormalities in serotonin metabolism are implicated in depression[62]. At the same time, the cognitive model of depression seems to be valid[35]. It would be logical, therefore, to examine the relationship between these two elements in research. One could investigate the consequences of manipulating serotonin systems with cognition as a dependent variable (the effects on cognition of the ingestion of selective serotonin re-uptake inhibitors, for instance) or the consequences of manipulating cognition with serotonergic functioning as a dependent variable (the effects of cognitive challenge on the activity of serotonin-rich areas of the brain in functional Magnetic Resonance Imaging studies perhaps). Similar exercises can be imagined in

many areas of psychiatric research – the relationship between dopaminergic systems and auditory source monitoring in the context of hallucinations, the relationship between information-processing deficits and cognitive biases in the case of delusions. Rather than using science as a form of tournament, pitting biological and psychological explanatory frameworks against one another, the model presented here suggests synergy.

Finally, there is the beguiling prospect of examining the model directly. The notion of a regression equation was introduced earlier. With some care it may be possible directly to examine the relative contributions of different classes of factor to measurable variance in the observed phenomena of mental disorder. That is not as outlandish as it might, at first sight, appear. It is not implied that a full mathematical model of mental disorder – or any one category of mental disorder – is possible (or that such a model would achieve much). But there are clear and distinct predictions emanating from the different models outlined here that could be tested. They could be tested because these different models make different predictions as to the relative contributions, and the mathematical relationships between, different factors.

Scientifically testing such a model

The validity of the proposed model rests on its ability to render coherent the available evidence of the role of psychological factors in mental disorder and its catalytic validity. Catalytic validity[91] refers to the utility of the analysis in providing ways of understanding from which testable implications follow. As examination of Figures 1a and 1b reveals, the model presented here invokes the existence of a mediating variable. It proposes that all causes of mental disorder are mediated through their effects in disturbing or perturbing psychological processes. While Occam's razor

declares that a parsimonious (and therefore praiseworthy) scientific hypothesis does not unnecessarily multiply causes, the more complex structure proposed in Figure 1b is necessary, the inclusion of the mediating variable can be justified. This paper argues that the concept of perturbation or disruption of psychological processes is logically necessary and empirically justifiable. But, perhaps more importantly, this model could be tested.

Theoretical models, in a scientific paradigm, are useful only if they are testable. One, possibly grandiose, test would be to conduct an analysis of the statistical relationship between variables. If the model presented here is valid, more of the variance in mental disorder (assuming that could validly be measured) could be accounted for through a pathway as described in Figure 1b than through that in Figure 1a (or indeed any other combination of the variables). But such an analysis would, in practice, be difficult. It would also be true that the model presented here would be falsified if a putative cause (a biological variable, social circumstance or life event) were to be demonstrated to be associated with mental disorder without being associated with a disturbance or disruption of psychological process.

But the proposed model generates a variety of less grandiose hypotheses. These address the ways in which disruptions or dysfunctions of psychological processes impact on mental disorder. In this category, investigations of the relationship between different forms of mental disorder and disruptions of the well-established psychological phenomena discussed above have already proved profitable, and could profitably be extended. The ways in which biological factors impact on psychological processes could also be investigated. This is less common in psychological research, but it seems profitable to explore how biological factors implicated in mental disorder impacts on the kinds of psychological processes

discussed above. Similar research could address the ways in which social factors impact on these psychological processes, and of course on the ways in which life events impact on psychological processes.

One consequence of the model presented here would be a relatively simple strategy for would-be researchers: for any example of mental disorder, a cunning researcher should examine the literature to identify a psychological process identified with that phenomenon. Then, any biological factor, social factor or set of live events also associated with that mental disorder would be ideal candidates for a research investigation. The model presented here would clearly predict links between these biological, social or circumstantial factors and the psychological processes. Any revealed associations should develop our understanding of the disorder. A failure to discover such links would undermine the model presented here.

Interventions

As for research, the model presented here would imply a key role for psychological factors in therapy. As outlined above, psychological interventions are growing in popularity and are increasingly being validated in clinical trials. The model presented here explains this, and argues for more of the same. Since psychological factors are key, therapeutic manipulation of these factors are key.

In such a model the role of the health care professional is clarified. In order to intervene to improve mental health, one must influence one of the predictor variables. In this model, interventions can be thought of as potentially focussed on each of the three classes of causal agents, but also on the mediating psychological processes. One can imagine a box marked ‘intervention’ in Figure 1b, with arrows pointing to each of these four elements.

One might even see the roles of different members of the care team as addressing these different targets. Thus a medical psychiatrist may prescribe medication to alter biological functioning (perhaps even to 'correct' some presumed 'underlying abnormality'). Within this model, however, it would achieve its benefit via a positive impact on mediating psychological processes. Interventions to improve the individual's environment and social environment (through the actions of many members of the care team but principally perhaps social workers) would act similarly. Specifically psychological interventions directly target the psychological processes – or should do.

For individual clinicians, it would imply that cognitive and behavioural interventions should be routinely considered for all forms of mental disorder. It does not, of course, mean that these would be obligatory, but they should be considered. In the UK context (with a highly socialised, state-funded health and social care system) this would imply considerable shifts in targets and funding. In other countries, the implications may be different, but perhaps even more politically challenging.

This structure itself may have utility, making the role of psychological approaches coherent. In the hypothetical cases outlined above, one can imagine a range of biomedical interventions, from the prescription of Prozac, through the application of electro-convulsive therapy to novel and experimental approaches which might even address the putative organic causes. One might imagine social interventions, from social therapies though systemic changes to radically political, service provision or preventative strategies. In the case of experiential causes, the range of interventions is similarly broad, from appropriate counselling and service provision for people experiencing potentially traumatising or health-threatening events to preventative, child-protection strategies. These kinds of interventions are,

however, different from (although complementary with) specifically psychological interventions. The specific and particular role of psychological interventions is to address the disturbances or dysfunctions in psychological functioning.

Of course, in nearly all treatment settings, genuinely multidisciplinary teams operate successfully. How then does the model proposed here extend either routine good practice or the more principled application of the biopsychosocial model? First, as noted above, many critics of the biopsychosocial model have noted that, in practice a presumed biological 'primacy' in terms of both putative cause and therapeutic benefit holds sway[31]. Where 'psychological' formulations or interventions are proposed, moreover, these often do not quite take the form proposed here. As mentioned above, cognitive behavioural methods and models are now widely advocated. Many will see this as a positive move. But the cognitive approach is based on one particular set of assumptions – that core dysfunctional beliefs and consequent negative automatic thoughts play a key role in the development of mental disorder. The present proposed model, of course, incorporates such approaches. However the range of possible psychological processes whose disruption or dysfunction could lead to mental disorder is much broader than cognitive behavioural formulations alone. This subtle shift to the incorporation of other psychological processes (from psychodynamic or behavioural orientations for instance) may extend the range, subtlety and power of psychologically informed multidisciplinary care plans.

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Figure 1a: Contributory factors in mental disorder

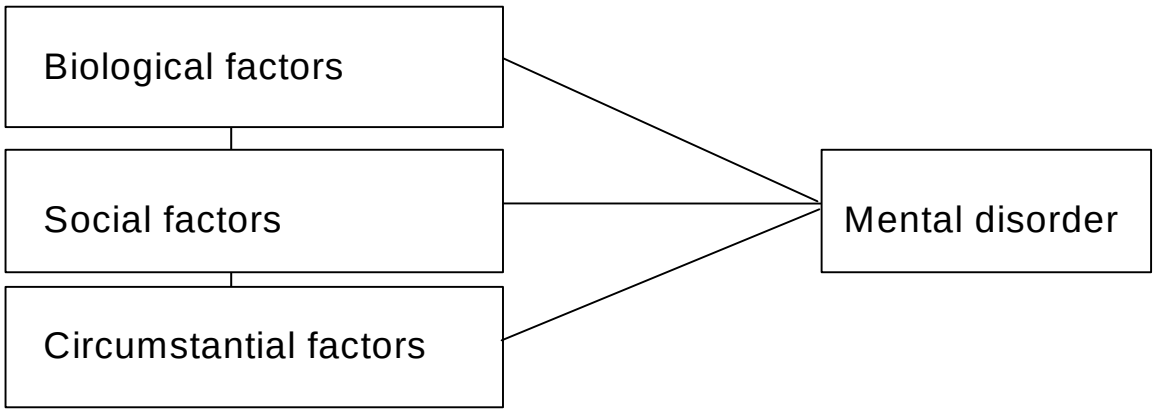

Figure 1b: The central role of psychological processes