

## PERSPECTIVES

# A Psychological Model of Mental Disorder

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**A coherent conceptualization of the role of psychological factors is of great importance in understanding mental disorder. Academic articles and professional reports alluding to psychological models of the etiology of mental disorder are becoming increasingly common, and there is evidence of a marked policy shift toward the provision of psychological therapies and interventions. This article 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, psychological, and social factors are coequal partners in the etiology 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 social factors, together with a person's individual experiences, lead to mental disorder through their conjoint effects on those psychological processes. Implications for research, interventions, and policy are discussed. (HARV REV PSYCHIATRY 2005;13:206–217.)**

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

In July 1989, President George Bush designated the 1990s as the “Decade of the Brain.” The European Community Council of Ministers<sup>3</sup> swiftly followed suit. More recently, advances in imaging techniques—such as computed tomography, magnetic resonance imaging, and positron emission tomography—have been hailed as offering great potential for detailed neuroanatomical investigations of many disorders.<sup>4</sup>

Nevertheless, although such research into biological aspects of mental disorder is both welcome and productive, it is important that the research be properly integrated with psychological and social accounts of the phenomena in question. Some observers fear the retrenchment of a reductionist, biomedical approach to mental disorder,<sup>5</sup> and it has been argued that diagnostic and etiological accounts stemming from a simple biomedical approach will be partial and scientifically inadequate.<sup>6</sup> Ill-informed reductionist accounts may also create the unfortunate perception that mental health care, so understood, is both dehumanizing and lacking in humanistic sensibilities.<sup>7</sup>

Psychological approaches to mental disorder offer alternative perspectives and can also be integrated with biological perspectives. This article will suggest that a comprehensive psychological model of mental disorder can offer a coherent, theoretically powerful alternative to reductionist biological accounts while also incorporating the results of biological research.

Most biomedical theories of, and biomedical interventions into, mental disorder explicitly or implicitly relate to synaptic or intracellular processes.<sup>8</sup> Such theoretical models implicate abnormalities in neurotransmitter functioning, and drug treatments (e.g., selective serotonin reuptake inhibitors) target synaptic neurotransmitters. By contrast, psychological approaches focus on associative networks based in the neural substrate but developed through learning, and rely on theories of conditioning, perception, appraisal, and belief formation, on propositional and implicational encoding,

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on mental models of the world and internalized schemas of relationships, and so forth. Psychological models of mental disorder therefore address different sorts of mechanisms than exclusively biomedical theories, 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, reports by professional bodies, strategy documents from policymakers, and proposed changes in legislation have all stressed the role of psychologists and of psychological perspectives in mental health. As an example, the first national clinical guidance issued by the United Kingdom's National Institute for Clinical Excellence concerned the frontline treatment of schizophrenia.<sup>9</sup> Among other important comments, this document recommended that "100%" of those diagnosed with schizophrenia be offered cognitive-behavioral therapy. Recent academic reviews of the role of psychosocial influences on mental illnesses and psychotic experiences,<sup>10</sup> as well as major grant-funded randomized controlled trials, have repeatedly demonstrated the effectiveness of psychological therapeutic approaches in a range of mental health problems.<sup>11–13</sup>

Psychological formulation—the summation and integration of the knowledge that is acquired through the assessment process<sup>14</sup>—represents an attempt to explain why people are experiencing difficulties. Formulations usually consist of a list of problems and possible psychological reasons for these;<sup>15</sup> they typically examine events in an individual's life and how he or she has interpreted and reacted to these; they are hypotheses about the nature and origin of problems, which are tested out over time<sup>16</sup> and therefore tend to change over the course of both assessment and therapy; and they are complex and may comprise a number of provisional hypotheses based on a large variety of psychological theories, each drawing on scientific research. Many psychologists use formulations, as do many psychiatrists—either as an adjunct to diagnosis (the iconic DSM-IV<sup>17</sup> suggests that diagnosis is only a start) or, as some psychiatrists propose, an alternative to diagnosis.<sup>18</sup> But what, then, makes for a good formulation, one that properly incorporates psychological elements? And this question raises, in turn, an even more fundamental one: what are the psychological elements of, and how does one construct, a coherent psychological model of mental disorder?

In this context it is worth mentioning the biopsychosocial model that George Engel<sup>17</sup> developed as a means of providing a scientific account of mental disorder that could challenge a reductionist biological account. The biopsychosocial model conceptualizes mental disorder as emerging from a human system that has both physical elements (a biological nervous system) and psychosocial elements (relationships, family, community, and the wider society).<sup>19,20</sup> This model was widely adopted,<sup>21–23</sup> but the inevitable microhistorical

pressures, such as the response of mainstream psychiatry to so-called antipsychiatrists,<sup>24–26</sup> has meant that some of the gloss has worn off the biopsychosocial model. Antipsychiatry remains vocal,<sup>18,27,28</sup> while, conversely, biodeterminist writings are also common,<sup>8</sup> with Wing<sup>29</sup> being an especially influential advocate.

The emphasis within the biopsychosocial model on social and psychological perspectives, and not exclusively on the biological aspects of mental disorder, may be welcome, but 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 practice, the model has been interpreted as reserving a dominant position for biomedical approaches<sup>5</sup>—with social and psychological factors being acknowledged but nevertheless considered to be mere moderators of the direct causal role of biological processes. This "primacy" of biomedical causation<sup>30</sup> has been cited, in turn, as an argument for the professional superiority of one profession (specifically, medical psychiatry) over others<sup>31</sup>—a claim that has become, needless to say, a source of some tension.<sup>32</sup>

This article 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—including a consideration of the different functions that psychological factors can play in different models of mental disorder: as causes, symptoms, and possible therapeutic factors.

Such consideration may, paradoxically, rationalize and contextualize the role of biochemical aspects in the biopsychosocial model. That model fails properly to address biological accounts of mental disorder if it cannot explain, for example, how monoamine abnormalities in depression<sup>33</sup> relate to findings of low self-esteem<sup>34</sup> and negative thinking patterns<sup>35,36</sup> and to findings of greatly increased incidence of mental disorder in disadvantaged groups.<sup>37</sup> A coherent model of the links between these findings—which would 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 conceptualization 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, one in which multiple causal

factors, different symptomatic markers, and different therapeutic elements are all appropriately addressed?

## MULTIPLE CAUSES OF MENTAL DISORDER

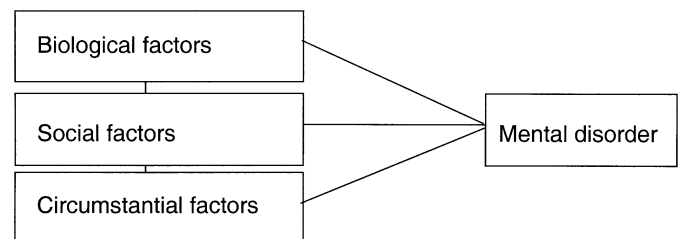
The biopsychosocial model is based on the notion of multiple simultaneous causes of mental disorder. Clinical and research evidence supports this assumption (as will be discussed below), which any proposed generic psychological model must take into account. One can imagine something along the lines of 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 0 to 100, or perfect mental health. One can further imagine three possible independent or predictor variables. The biopsychosocial model, as well as the model proposed here, includes multitude causes—multitude predictors. For the purposes of the argument presented here, however, three main classes of predictor variables (biological, social, and circumstantial) are included, as illustrated in Figure 1. It is worth noting that these classes of causal or predictor variables are slightly different from the components of the biopsychosocial model—reflecting, as will be clarified below, two important modifications of that model: the distinction between psychological processes and personally significant life events—or what I shall refer to as “circumstances”—and the specific status given to the disruption of psychological processes as a “final common pathway” to mental disorder, meaning that it is only through the disruption of psychological processes that any precursors, whether biological, social, or circumstantial, come to be expressed and experienced as mental disorder.

All of these classes of variables are causally related to mental ill health. Links between mental disorder and genetics, biochemical abnormalities, neuroanatomical abnormalities, and the structure and functioning of the brain have been studied extensively. Some authors have even argued that in contrast to biological variables, which have obvious explanatory power in cases of mental disorder, psychosocial aspects account only for human experiences that fall short of being classified as clinically significant disorders.<sup>30</sup> In any event, a variety of problems—from unipolar depression<sup>38</sup> to bipolar disorder<sup>39</sup> to schizophrenia<sup>40</sup>—has been reliably associated with genetic heritability. Consequently, in the hypothetical multiple regression equation suggested above, 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 factors contribute to mental disorder. Again, the number of studies is enormous, but there are clear links between poor housing<sup>41</sup> and poverty and unemployment,<sup>42</sup> on the one hand, and the expression of a wide range of mental

health problems, on the other; put more simply, there is an association between mental disorder and the experience of poverty and deprivation.<sup>43–45</sup> In our hypothetical example of a multiple regression equation, a general measure of disadvantageous social environment would clearly be a significant predictor of variance in mental ill health. This type of social causal variable can be seen as occupying the space of psychiatric sociology—examining and contextualizing social causes of mental disorder.<sup>46</sup>

When people refer to psychological issues, they sometimes refer to the effects of potentially life-shaping experiences, or circumstances, that affect mental health—childhood sexual abuse,<sup>47</sup> bullying,<sup>48</sup> attachment relations with parents,<sup>49</sup> assault,<sup>50</sup> and all other major and minor interpersonal experiences. Although these circumstances are of disparate kinds, they and many other life events contribute to mental disorder.<sup>51,52</sup> Because of the breadth of these circumstances, it may be difficult to capture them all on one hypothetical dimension. Nevertheless, in our hypothetical multiple regression equation, 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 variable 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. Another use of the term “psychological” is to refer to interpretation or to information processing—the process by which personal meaning is ascribed to the events. Psychologists thus routinely distinguish between life events or circumstances and the personal meaning that these events have for an individual,<sup>16</sup> whereas the original biopsychosocial model tends to obscure this distinction.



**FIGURE 1.** Contributory factors in mental disorder.

A simple statement that biological factors, social factors, and life circumstances all contribute to the development of mental disorder does not take us further than the biopsychosocial model. It is important to separate potentially salient life circumstances from both the broader social context and from the psychological interpretative processes. The relative contributions of the various hypothetical contributors to mental ill health and their relationship to one another must also be clarified, reflecting the specific psychological nature of the model

proposed here.

First, scholars and scientists from different academic and professional backgrounds may well differ as to their hypotheses about the relative importance of the putative predictor variables. Witness the current interest in genetics. As commented above, genetic aspects of mental disorder are important. The facts are frequently overstated, however, 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.<sup>53</sup> This figure is commonly taken to imply that 85% of the variance in the presentation of psychotic symptoms can be statistically attributed to the variance in the population's genome. This interpretation may be misleading, however. Rutter<sup>54</sup> cogently argues that psychosocial, circumstantial, 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 the above environmental effects do not necessarily sum to 100%), which Rutter argues may lead to subtle, 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 circumstances, and psychological factors together could explain only the "remaining" 15% of the variance in the presentation of psychotic symptoms.

In addition to their separate roles in accounting for variance, biological, social, circumstantial, and psychological variables interact. Psychological vulnerability factors such as neuroticism<sup>55</sup> may make a person more emotionally responsive to life events. Similarly, the social support buffer hypothesis<sup>56</sup> suggests that the level of a person's available social support buffers the impact of circumstantial stressors on mental health. Thus, for two individuals experiencing stressful events, the person with the greatest level of social support will experience a lower level of mental disorder. A similar set of interactions may explain why the concordance rate for monozygotic twins for no mental disorder is 100%.<sup>57</sup> Consequently, it is not inevitable that biomedical models would dominate other explanations for mental disorder. Social factors and the influence of circumstantial life events may be just as important.

## **THE PSYCHOLOGICAL CONSEQUENCES OF BIOLOGICAL ABNORMALITIES**

Giving proper weight to the role of biological factors in mental disorder in no way undermines the psychological model proposed here; what is required, however, is that biological variables be placed into a proper relationship with other factors. In the case of auditory hallucinations, for example, a fascinating story is emerging. Biologically, there is considerable evidence

of a genetic element in schizophrenia.<sup>40</sup> Schizophrenia, psychotic symptoms, and hallucinations have been linked with cerebral lateralization.<sup>58</sup> In particular, it has been suggested that people who hear voices are more likely to have poorly lateralized cerebral hemispheres, and that the brain's language areas are less lateralized in people who hear voices.<sup>59</sup> It is entirely credible, moreover, that biochemical, neuroanatomical, or genetic abnormalities lead to poor lateralization of language processing and hence to the problems associated with a diagnosis of schizophrenia.<sup>60</sup> Since one key task in neural language processing is presumably the identification and localization of perceptual experiences, this failure of lateralization seems to lead to problems in discriminating voices (heard) from other forms of cognition (thought or remembered). Psychologically, what we have in such cases is a failure or disruption of a psychological process—source monitoring or attribution, or reality monitoring.<sup>61</sup>

It would be incorrect to infer that biological factors can therefore be excluded, or even minimized, in the development of hallucinations. Nevertheless, few people would dispute that auditory hallucinations are psychological phenomena, and what the preceding analysis suggests is that hearing voices must be conceptualized as a psychological phenomenon not only in terms of result, but also in terms of process. The final, inescapable pathway to hearing disembodied voices is the misattribution of the source of percepts—a psychological process. It is influenced by biological factors, but it is equally influenced by social environmental factors and the important circumstances in a person's learning history.

The point of the above example is not to present findings concerning cerebral asymmetry in hallucinations, but to illustrate that a plausible biological pathway from genetics, through neurology, to behavior necessarily implicates psychological processes. That is, biological factors influence mental disorder through their impact on psychological processes. The case of depression is illustrative. Abnormalities in serotonin metabolism are implicated in depression;<sup>62</sup> for example, a tryptophan-reducing (and therefore serotonin-reducing) diet can induce depression.<sup>63</sup> In the psychological model of mental disorder proposed here, this association is accepted, but the causal role of biology is seen as being effected through the disruption of psychological processes. A reduction in dietary tryptophan leads to a reduction in available cerebral serotonin, which is implicated, in turn, in the neurological mechanisms supporting various important appraisal processes. It is entirely plausible to suggest that these appraisals will include the ways in which people see themselves, their world, and their future—the negative cognitive triad of the cognitive model of depression.<sup>35</sup> Thus the biological tryptophan-serotonin system could indeed lead to depression, but through psychological processes.

Likewise, insofar as social factors have a central role

in the emergence of mental disorder, it is only through the disruption of psychological processes. A psychological model of mental disorder (based on the disturbance or disruption of psychological processes) accepts that living in poverty and in conditions of social deprivation can indeed lead to problems such as depression. In particular, living in such a disadvantaged environment may lead to disillusionment, hopelessness, and learned helplessness—to a realization 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.<sup>64–66</sup> Depression is then the direct consequence of this disruption of psychological processes.

And, finally, the same applies to particular life experiences, or circumstances. Being assaulted by one's parents obviously 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 sexual, emotional, or physical abuse, the experience is likely to affect the ways in which the children (and later the adults) appraise themselves, the people in their lives, their own actions and the consequences thereof, and the ways in which relationships and social intercourse should be governed—that is, their cognitive schemas.<sup>67</sup> Indeed, there is convincing evidence<sup>68</sup> that assaults that mirror childhood events reactivate such cognitive schemas with terrible consequences.

It is important to stress that these psychological processes are not just cognitive. Cognitive processes are important (and in vogue), and the impact of writers such as Beck,<sup>35</sup> Seligman,<sup>69</sup> and Young<sup>67</sup> is undeniable. Nevertheless, other psychologists and psychiatrists have stressed disruptions or disturbances in psychological processes other than cognitive ones. The phenomenon of “learned helplessness” was mentioned earlier and is closely associated with Seligman's cognitive approach,<sup>69</sup> but learned helplessness itself was initially analyzed as a behavioral phenomenon.<sup>70</sup> Purely behavioral models of depression have a distinguished

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history<sup>71</sup> and remain highly regarded,<sup>72</sup> and behavioral elements are a key part of conventional cognitive-behavioral therapy.

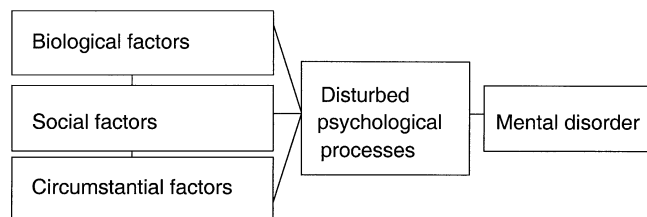
Psychodynamic approaches—which, within the model presented here, invoke the disruption or disturbance of object relations, sexual drives, or other intrapsychic events under the category of “psychological processes”—similarly have a distinguished history, have been massively influential on modern psychology, and are currently a core part of the practice and ethos of clinical psychology.<sup>73</sup> In the example of depressed mood used here, the psychodynamic notions of depression as involving, for instance, disrupted object relations<sup>74</sup> or involuted anger<sup>75</sup> remain respectable elements of psychological formulations. In the case of personality

disorders, although most recent psychological accounts of personality problems cite maladaptive cognitive schemata concerning social relationships,<sup>68,76</sup> other writers have cited either failures to integrate personality structure<sup>77</sup> or disruptions of psychodynamic processes related to fundamental object relations.<sup>78</sup> In the model presented here, disruption of psychological process is certainly not limited to the disruption of cognitive processes.

## PSYCHOLOGICAL PROCESSES

A generic psychological model of mental disorder proposes that biological, social, and circumstantial factors all operate causally by disrupting or disturbing psychological processes. This central conceptual point is illustrated in Figure 2.

Psychological approaches have 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 those events. Like the biopsychosocial model, the psychological model attempts to address the interactions between these classes of causal variables. Biological, social, and circumstantial factors are all important and all presumed to interact. Importantly, however, in this model it is the conjoint impact of these interacting factors on psychological processes that leads to mental disorder.



**FIGURE 2.** The central role of psychological processes.

This last, important point is worth elaborating. The model developed here implies not only that the common symptoms of psychiatric classifications are psychological in nature (i.e., that hallucinations, depression, anxiety, and so on 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. Consequently, as in the biopsychosocial model, multiple causal factors are implicated. In a hypothetical case of depression, for example, several distal causal factors may be salient, and several key psychological processes may be implicated. It is possible to hypothesize that social deprivation, childhood maltreatment, genetic vulnerability, and biochemical insult may all contribute to the onset of depression.

These factors may impinge on psychological

processes such as those relating to self-esteem, beliefs in self-efficacy, and expectations of reward. They may implicate neuroanatomical systems responsible for reward and reinforcement, autobiographical memory, and social cognition, and may involve neuroanatomical systems that involve serotonin (as well as other neurotransmitters). Depression, in the model presented here, is characterized as a direct consequence neither of the neuroanatomical mechanisms nor of the contributing social and circumstantial factors. Rather, these neuroanatomical mechanisms and other factors 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 these psychological processes that are seen as ultimately leading to the mental disorder—in this case, depression.

This argument can be applied to hallucinations. Abnormalities in the poorly lateralized 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 percepts 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 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 a percept's origin. 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.” Instead, they perturb the psychological processes, and it is these perturbations that lead to—that “cause”—the phenomena that are labeled mental disorder.

As another illustration, the same type of analysis can be applied to personality disorder. Innate or heritable individual differences in personality traits (e.g., increased levels of the trait of neuroticism) may be exacerbated by life circumstances (e.g., experiencing childhood abuse) to precipitate more extreme personality dysfunction later in life. Under the model presented here, however, it would be inappropriate to say that the biological traits, the life circumstances, or even the combination of the two has produced or caused a personality disorder; instead, one would consider this combination of distal factors to have profoundly affected how the persons in question perceive themselves, their world, and their relationships with others. It is this element—the perturbation of psychological processes—that leads to mental ill health.

## **WEAKNESSES AND POSSIBLE CRITICISMS OF A PSYCHOLOGICAL MODEL**

Philosophers of science consistently maintain that, even in supposedly objective science, the researcher's interests and allegiances may shape claims of causality.<sup>79</sup> This insight is particularly pertinent in the case of mental health.<sup>80</sup> If a person is “depressed” and has “low self-esteem,” it is easy to see two professionals maintaining opposing points of view: one maintains that the depression caused the lowered self-esteem, whereas the other maintains that the depression results from negative self-evaluation. Many philosophers of science would conclude that in psychiatry, “truth” is dependent on perspective.<sup>81</sup>

In the case of the model presented here, psychological processes are given center stage. It could be argued that this emphasis reflects nothing more than a perspective focused upon psychology, but even if that were true, it would not render the model invalid. Whether or not there were other perspectives, a coherent statement of the nature of psychological causation would remain 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 hypothesis is a testable one: 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<sup>79</sup> suggested, such testable hypotheses separate subjectivity from science.

### **“Truly Organic” Disorders**

While some readers may allow that a psychological model fits relatively well with disorders such as anxiety and depression, those same readers might nevertheless claim that there are “truly organic” disorders that do not fit at all. 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 questions of causation, it could be argued that the interventions are social and medical rather than predominately psychological. Psychotherapy does not ameliorate the disorders, and medical care and, particularly, social care are both important. Moreover, in the case of phenylketonuria, the consequences of the undoubted genetic cause of the condition can be (largely) avoided by the social (or social and biological) manipulation of removing all foods containing phenylalanine.

But let's take a step back. A fundamental question is why Down's syndrome and phenylketonuria are being considered at all in a discussion of mental disorder. The distinguishing feature of these two disorders—in relation to other genetic disorders—is their impact on mental processes. If phenylketonuria did not affect learning and

behavior, it would still be a genetic disorder, but it would not be a mental disorder. By contrast, cystic fibrosis is a genetic disorder with a complex therapeutic imperative, but it is not a mental disorder. In cases of Down's syndrome and phenylketonuria, care services must perform a proper formulation of the problem and address the observed functional elements. The most therapeutic intervention should be employed, but as can be inferred from an analysis of these two disorders, the identification of the best intervention does not necessarily impute causality.

In view of the above, it should be apparent that the psychological model presented here does not preclude a causal role for genetic factors with respect to particular mental disorders. By the same token, the model does not assume that all mental disorders should be amenable to psychotherapy. What the psychological model does propose is that mental disorders are characterized by the disruption of psychological processes. Down's syndrome and phenylketonuria occur because of genetic abnormalities. But it is clear that these genetic abnormalities, if not addressed through appropriate interventions, 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 article, Eric Kandel<sup>82</sup> reaffirmed the biological basis of psychiatry. He proposed a model of psychiatry that is, in essence, diametrically opposed to the model presented here. Kandel argued not only that organic or biological factors are 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 emphasizes (as does the model presented here) how biological factors can affect psychological functioning—emotions, cognitions, and behavior. But he also emphasizes the brain-based nature of psychology. He stresses how all emotions, all cognitions, and all behavior depend on brain functioning. Kandel points out that in a scientific sense, all learning involves structural changes in the brain—changes in synaptic responsivity at the very least, and possibly also ones involving neuroanatomy. He therefore logically deduces that any psychological causation of mental disorder must be ultimately organic in character, and that any therapy, even psychotherapy, must achieve its effect through biology. Not only does Kandel argue (as does the present model) that biological changes affect psychological processes, he argues that learned patterns of behavior or

self-evaluative schemata learned in childhood reflect physical changes in the neural associative networks. He argues, moreover, that any relearning that occurs during therapy represents changes in those networks.

On one level, this analysis is obviously true. Any learning must, at the molecular and synaptic level, be based on biological changes and involve the brain. But such an argument is intellectually trivial since all learning—all human behavior—is dependent on the functioning of the brain. 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—learning that 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 have a basis in 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. Consequently, a reductive attempt at explanation will fail to account for the psychological phenomena in question.

Other authors go even further than Kandel, arguing, in effect, that all psychological concepts will disappear from the psychiatric lexicon in the same way that phlogiston has disappeared from physics; that is, as we understand the neural basis of behavior, we will have no need for the notion of psychology.<sup>83</sup> There are three main criticisms of such a position. First, the available data suggest that psychological factors (e.g., 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 psychological variables and the biological substrate, but the variables' 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, honor, guilt, and so on. 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. Third, and most centrally, many commentators<sup>84–86</sup> have addressed this issue by asserting that mental disorders are irreducibly 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, though not therefore compelling, arguments in support of biological<sup>82</sup> and even social<sup>23</sup> models of mental disorder, it may be argued that we need to develop a pluralistic model of mental

disorder—and not an exclusively psychological one.

The impetus for the development of the psychological model presented here was a frustration with the biopsychosocial model. Most descriptions of that model fail to address the causal interrelatedness of the variables involved; biology, the social environment, life events, and psychological processes, while all important, are not independent factors. Consequently, a pluralistic model that regarded psychological factors as an independent class of causal agents, rather than as the final common pathway for the effects of other causal agents, would have the benefits of the biopsychosocial model but also all its flaws.

Finally, of course, the model presented here is integrative. Biological and social factors are properly acknowledged; one might even hope that this model might itself be seen as pluralistic. That said, the model presented here does place central emphasis on psychology. Although that emphasis may prevent the model from being regarded as pluralistic at present, it may well be that a pluralistic model will eventually emerge from a concurrent reading of this model with others, such as that proposed by Kandel.

## IMPLICATIONS OF A PSYCHOLOGICAL MODEL

Models such as the present, psychological one are valid if they are useful. It has been powerfully argued<sup>84,87,88</sup> that pragmatism should be the benchmark for evaluating theoretical models of mental disorder. In this context, the psychological model has obvious utility in guiding thought. It offers a conceptual framework that allows clinicians to separate and integrate multiple causes of mental disorder. Rather than proposing a simple “biological cause and psychological symptom” approach, the model separates causes both from mediating processes and from effects. The model has clear similarities to Padesky and Greenberger’s “five-factor model,”<sup>89</sup> which is widely used in cognitive-therapy training. That model proposed five elements in mental disorder— affect, behavior, cognition, environment, and physiology. It contends that a full understanding of these five factors is vital for a complete formulation. The separation of affective, behavioral, and cognitive aspects of “psychology” within this model is especially important. The weakness of the five-factor model, however, is that it fails to specify the nature of the interrelationships between these factors.

In the biopsychosocial model, although it is expected that all three elements contribute to the experience of mental disorder, it does not follow that psychological factors are central. The psychological 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 psychological model would retain the disruption of psychological processes as a logically necessary final step.

Conceptual issues notwithstanding, models are worthy of serious attention only 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, as we shall in the following sections.

## Research

In addition to placing a high priority on research into psychological factors in mental disorder, the proposed model is helpful in pointing to the importance of studying the interrelationship of biological and psychological factors. For example, research might be conducted into the biological influences on source monitoring and simultaneously into the relationships between source monitoring and the experience of hallucinations. We might also investigate other (nonbiological) 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 impinges on thought disorder, considered a psychotic phenomenon.<sup>90</sup>

Paradoxically, the psychological model of mental disorder may actually help to explicate the mode of action of biological factors. For example, although abnormalities in serotonin metabolism are implicated in depression,<sup>62</sup> the cognitive model of depression also seems to have some validity.<sup>35</sup> Rather than focusing on which of these two models is correct (to the exclusion of the other), the psychological model of mental disorder points to the need to understand the relationship between biological and cognitive elements in depression (e.g., with cognition both as a dependent and independent variable in relation to the administration of selective serotonin reuptake inhibitors). Similar exercises can be imagined in many areas—for example, the relationship between dopaminergic systems and auditory source monitoring in the context of hallucinations, or the relationship between information-processing deficits and cognitive biases in the case of delusions. Rather than using science as a form of tournament in which biological and psychological explanatory frameworks are pitted against one another, the psychological model promotes synergy.

Finally, there is the intriguing 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 factors to the measured variance in observed phenomena of mental disorder. It would be too much to expect a full mathematical model of mental disorder—or of any one category of mental disorder.



Nevertheless, there are clear, distinct predictions emanating from the different models outlined here, and they could be tested because the models make different predictions as to the relative contributions of, and the mathematical relationships between, different factors. The relative contributions of each factor within the model presented here could be tested to determine, for example, whether biological, social, or circumstantial variables contributed most to the variance in expressed mental disorder. It would be also possible to test whether psychological-process variables act (as is predicted) as complete mediators of the contribution of these variables, and this psychological model could be tested against plausible alternatives.

### Scientifically Testing Such a Model

The validity of the proposed model rests on its ability to render coherent the available evidence concerning the role of psychological factors in mental disorder and also on its catalytic validity<sup>91</sup> (which refers to the utility of the analysis in generating testable implications). As Figures 1 and 2 reveal, the psychological model invokes the existence of a mediating variable and proposes, in particular, 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 entities, the more complex structure proposed in Figure 2, with its inclusion of a mediating variable (the perturbation or disruption of psychological processes) is logically necessary, empirically justified, and, indeed, testable.

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 such variance could be validly measured) could be accounted for through a pathway as described in Figure 2 than through one conforming to Figure 1 (or indeed any other combination of the variables). But such an analysis would, in practice, be difficult. The psychological model could be falsified if it was demonstrated that a putative cause (a biological variable, social circumstance, or life event) was associated with mental disorder without being associated with a disturbance or disruption of psychological processes.

The psychological model also generates a variety of less grandiose hypotheses concerning the ways in which disruptions or dysfunctions of psychological processes affect 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 (e.g., self-concept and source-monitoring) have already proved productive and could well be extended. The ways in

which biological, social, or circumstantial factors affect psychological processes could also be investigated.

The psychological model suggests a relatively simple research strategy for would-be researchers: for any particular mental disorder, one could examine the existing literature to identify a psychological process identified with that disorder. Then, any biological, social, or circumstantial factor also associated with that mental disorder would be ideal candidates for investigation. The psychological model would predict links between those factors and the relevant psychological processes. Any revealed associations would increase our understanding of the disorder, and a failure to discover such links would undermine the model itself.

### Interventions

The psychological model would imply a key role for psychological factors in therapy, and the model itself clarifies just what the role of the health care professional should be. In order to intervene to improve mental health, one of the predictor variables needs to be influenced. In this model, interventions will need to focus not only on the three classes of causal agents, but also on the mediating psychological processes. (One can imagine a box marked "intervention" in Figure 2, 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; for example, the role of a psychiatrist may be to prescribe medication to alter biological functioning (perhaps even to "correct" some presumed "underlying abnormality"). Within this model, however, the intervention would achieve its benefit via a positive impact on the mediating psychological processes. Interventions to improve the individual's social and circumstantial environment (through the actions of many members of the care team, but principally, perhaps, social workers) would operate in much the same way. Specifically psychological interventions should, by contrast, directly target the psychological processes.

For individual clinicians, the psychological model entails that cognitive and behavioral interventions should be routinely considered for all forms of mental disorder. It does not, of course, mean that any particular interventions would be obligatory—but only that they be considered. In the UK context (with a highly socialized, state-funded health and social care system), following the psychological model would entail considerable shifts in targets and funding. In other countries, the implications may be different.

The psychological model's structure—with its psychological processes and biological, social, and circumstantial factors—may also have utility in developing an integrated treatment plan. In the hypothetical cases outlined above, one can imagine a range of biomedical interventions—from the prescription of fluoxetine, through the application of

electroconvulsive therapy, to novel and experimental approaches that could address possible organic causes. One might imagine social interventions of various kinds, including strategies focused on prevention, service provision, or even political action. In the case of circumstantial causes, the range of interventions is also broad—for example, appropriate counseling and service provision for people experiencing potentially traumatizing or health-threatening events, and preventive strategies for protecting children. It should be kept in mind, however, that all of the above interventions are different from, though complementary to, specifically psychological interventions, whose specific role is to address the disturbances or dysfunctions in psychological processes.

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? Many critics of the biopsychosocial model have noted that, in practice (and as mentioned earlier), a presumed biological “primacy” holds sway with regard to both causality and treatment of mental disorders.<sup>31</sup> Moreover, when “psychological” formulations or interventions are proposed, they often do not quite take the form proposed here. For example, although cognitive-behavioral methods and models are now widely advocated, that particular approach to treatment is based on a specific set of assumptions—namely, that core dysfunctional beliefs and consequent negative thoughts play a key role in the development of mental disorder. The psychological model does, of course, incorporate such an approach, but the range of possible psychological processes whose disruption or dysfunction could lead to mental disorder is much broader in the psychological model than with cognitive-behavioral formulations alone. This subtle shift to the incorporation of other psychological processes (for example, beyond orientations that are primarily or exclusively psychodynamic or cognitive-behavioral) may extend the range, subtlety, and power of psychologically informed, multidisciplinary care plans.

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