Models of Mental Illness

Abstract

This chapter has two aims. The first aim is to compare and contrast three different conceptual-explanatory models for thinking about mental illness with an eye towards identifying the assumptions upon which each model is based, and exploring the model’s advantages and limitations in clinical contexts. Major Depressive Disorder is used as an example to illustrate these points. The second aim is to address the question of what conceptual-theoretical framework for thinking about mental illness is most likely to facilitate the discovery of causes and treatments of mental illness in research contexts. To this end, the National Institute of Mental Health’s Research Domain Criteria (RDoC) Project is briefly considered.

**Models of Mental Illness**

1. Introduction.

Each and every one of us, at some point in our lives, will be touched by mental illness—our own, or that of a parent, grandparent, aunt, uncle, sibling, spouse, child, grandchild, friend or coworker. Close to 450 million people worldwide suffer from mental or neurological disorders and the numbers continue to rise (World Health Organization 2012). In order to address the current global mental health crisis, a conceptual-explanatory framework or model adequate for investigating the causes of, diagnosing, explaining and treating mental illness is required. Yet, what kind of model will do? Should it include psychological factors like emotions, thoughts and memories, or social factors like income or living conditions? Would a model that understood mental illness as exclusively brain-based be sufficient? Will a single model prove adequate for understanding mental illness or are different models necessary? Providing some preliminary answers to these questions is the aim of this chapter.

2. Models of Mental Illness in the Clinic

Many different conceptual-explanatory frameworks of mental illness have been put forward in the scientific and philosophical literature (e.g., Freudian psychoanalytic theory, psychodynamic theory, cognitive-behavioral theory)—too many in fact to consider in a single chapter. However, there are three even more general frameworks that we may evaluate and in so doing learn lessons relevant for assessing the pros and cons of other models. These include (1) the folk psychological model, (2) the biopsychosocial model and (3) the medical model. How these three models differ from each other and their merits and failings in clinical contexts may best be illustrated by means of an example.

Rebecca, a 19-year-old woman who has just entered her sophomore year of college, begins mid-way through the semester to miss classes, mealtimes in the cafeteria and social events with her close group of friends. One of her friends, Tom, begins to worry and invites Rebecca out for a coffee to see how she is doing. A few minutes into their conversation, he asks her if everything is okay. In response, Rebecca becomes teary-eyed but does not say anything. Concerned, Tom gently asks her some additional questions: What are you sad about? Did something happen over the summer? Is everything all right at home? Rebecca gradually reveals to Tom that she has felt bad since the beginning of the semester, only wanting to sleep, and not really wanting to eat, but that she cannot point to anything specific that prompted this change in mood. Tom finds this answer strange, particularly because he has known Rebecca to be a basically happy person who has only been sad in the past for legitimate reasons like failing an exam or ending a friendship or romantic relationship. While Tom is concerned, he realizes that Rebecca may not feel comfortable telling him what is wrong. So, he gently suggests that she should talk to a school counselor, who he thinks might be able to help.

Tom’s response to Rebecca’s sadness is not uncommon. When we see a friend whose behavior is unusual and this behavior persists and is debilitating, we tend to assume that the sources of these behavioral changes are *psychological* or “in the head”and that the best approach to helping them is to try to talk to them or encourage them to see a professional. You may not have realized it, but you use a conceptual-explanatory framework on a daily basis. Specifically, you have learned from a very young age to believe that human beings as well as some non-human animals have some special quality—a mind, consciousness, awareness—that other kinds of things—such as rocks stars and trees–lack. You regularly describe yourself as having beliefs, desires, feelings and intentions. You comfortably ascribe similar internal states to other human beings and some non-human animals. You appeal to these states to explain your own behavior and to make sense of the behavior of others. When you are asked why you are sad, you often put forward reasons—beliefs, feelings, unrealized desires that you have—to explain why.

Because the vast majority of us are not professional psychologists, but rather, ordinary folk, psychologists and philosophers have come to refer to this conceptual-explanatory framework as “folk” or “commonsense” psychology (e.g., Churchland 1981). When we use this framework we are assuming what philosopher Daniel Dennett (1987) has dubbed “the intentional stance”. In other words, we posit abstract mental states rather than concrete physical states (e.g., changes in the nervous system) to explain human behavior. Sometimes the ontology that we appeal to is “mixed” in so far as we talk about both mental states and mental processes (e.g., attention, memory). Some of us may be inclined to import more advanced scientific concepts into our folk-psychological theorizing (e.g., Freud’s id, ego, and superego) or to be “folk neuroscientists” insofar as we may explain behavior by appeal to a rudimentary understanding of the brain. In trying to understand Rebecca’s sadness, Tom was being a folk psychologist and adopting the intentional stance. However, as Tom himself recognizes, this conceptual-explanatory framework does not offer any treatment options over and above talking to a person about their feelings or suggesting that they talk to someone who is a professional at understanding the mind and mental disorders.

Suppose Rebecca takes Tom’s advice and she makes an appointment with the university’s *Center for Counseling and Personal Growth*. She begins to see a licensed mental health counselor (LMHC) with a PhD in psychology (PsyD) on a weekly basis. During these sessions, the counselor asks Rebecca questions about her feelings, her recent experiences, her life growing up, her friends and family, her schoolwork, her romantic relationships, her diet and exercise habits. Over the course of these sessions, the counselor attempts to formulate hypotheses about the causes of Rebecca’s sadness and to make suggestions about strategies Rebecca might implement to improve her mood. The counselor suggests yoga, mindfulness and exercise classes, social activities such as going out with friends and joining clubs, changes in diet to include more vitamin-rich foods. He recommends that she see a medical doctor to make certain her thyroid is functioning properly and her blood-sugar levels are normal.

Implicit in such suggestions is the counselor’s appeal to an explanatory framework to explain Rebecca’s behavior that differs from that of folk psychology but is inclusive of it. This model has been referred to as the “biopsychosocial model” (Engel 1977) (See Gifford, Chapter 45) because those health care professionals who use it consider a variety of different kinds of causal factors that may result in changes in human health and behavior including: biological/physical factors (e.g., diet and exercise, neurobiological and physiological changes), psychological factors (feelings and thoughts or thought patterns) and social factors (social relationships and activities). American psychiatrist George Engel (1977) put forward this framework in the late 1970s because he believed that the successful diagnosis and treatment of patients with disease or illness required doctors to appeal to a multi-dimensional causal model. He intended it as a superior alternative to the medical or biomedical model (described below and in Chapter 45), which he regarded as interested exclusively in biological causes.

As is illustrated in Rebecca’s case, the counselor believes that psychological, biological and social factors may all be contributing to her depressed mood. Furthermore, the kinds of causal interventions that he is proposing correspond to all three of these different types of causes. For example, the counselor regards talking to him to be insufficient for improving Rebecca’s mood and he acknowledges that the causes of her sadness may be complex and that the different causal factors that he has identified may be acting independently or in concert with one another. He also appeals to this framework to identify a variety of different kinds of intervention strategies that he thinks may, either independently or in combination, make Rebecca feel better. It is clear to see how this conceptual explanatory framework differs from the folk-psychological one in terms of both the causes it posits to explain Rebecca’s sadness and the kinds of treatment strategies to which it points.

Suppose Rebecca continues to see the counselor for two months. Because she does not appear to be improving, the counselor thinks that something else might be wrong—that the causes of her sadness may include neurobiological and biochemical causes in addition to psychological and social causes. Without a license to prescribe drugs or other forms of treatment to Rebecca, he is unable to address directly these other kinds of causes. However, the university’s counseling center has a contract with a psychiatrist who can. The counselor suggests that in addition to seeing him on a regular basis, Rebecca should see this psychiatrist. He obtains Rebecca’s permission to forward his notes on her case to the psychiatrist and Rebecca schedules an appointment with her.

The psychiatrist begins by asking Rebecca how she is feeling. They talk for fifteen minutes and the psychiatrist indicates that based on the counselor’s notes, and some of the answers Rebecca has provided to her questions (e.g., how is your mood these days? your memory? your energy level?) her hypothesis is that Rebecca has *Major Depressive Disorder* (APA 2013, 160-168). She removes the 5th edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) from her bookshelf, explains to Rebecca what the symptoms of the disorder are, and identifies the reasons why she thinks Rebecca satisfies the criteria for this diagnosis. She notes that Rebecca exhibits the requisite set of symptoms insofar as she has been depressed for over 2 weeks, has lost interest in daily activities, is experiencing hypersomnia, fatigue, and an inability to think and concentrate. The psychiatrist claims that research suggests that the etiology of the disorder is complex, involving genetic, developmental and environmental factors. In other words, a complex combination of causal factors interacting throughout Rebecca’s lifetime (and even before, if we consider genetic mechanisms), likely contributed to Rebecca exhibiting the symptoms of *Major Depressive Disorder*. The psychiatrist explains that while it is impossible to intervene effectively in this complex array of historical causes, it is possible to intervene in those causes potentially and currently contributing to Rebecca’s depressed mood. One such set of causes, she claims, is neurobiological, and she identifies several drug therapies that have proved successful in intervening at the neurobiological level to treat depression. The psychiatrist tells Rebecca that counseling is also an additional effective intervention for addressing past traumas and coping with day-to-day life events.

Rebecca heeds the psychiatrist’s advice. She begins taking an anti-depressant medication. She continues to see both the counselor and the psychiatrist during the next month. After taking the drug regularly for several weeks, Rebecca’s mood begins to improve and she finds she has more energy and more interest in her studies and spending time with her friends. Her life gradually begins to return to normal.

In contrast to Tom and the counselor, the psychiatrist adopted what we will here refer to as a *sophisticated* “medical or biomedical model” of mental illness (See Chapter 45, Chapter by Kincaid). This is to be contrasted with a “strong interpretation” of the medical model (Murphy 2009) that might understand mental illness as caused primarily by brain abnormalities (e.g., imbalance in neurotransmitters, presence of lesion, abnormal neural connectivity) and treatable exclusively by intervening in the brain or nervous system (via, e.g., surgery, electroconvulsive therapy, pharmacology). Engel introduced the biopsychosocial model in the late 1970s because he was concerned that this strong interpretation of the medical model of illness had become dominant in medicine and was not only problematic for the reasons mentioned above (See Gifford, Chapter 45) but also because it led to a failure on the part of medical practitioners to treat their patients as psychological and social beings in addition to biological beings. Engel thought this dehumanized patients in ways that were antithetical to the goal of promoting their health. The biopsychosocial model was intended to encourage doctors to engage with their patients as psychological and social beings and to discover additional avenues for treatment not suggested by the strong interpretation of the medical model. The psychiatrist’s use of a “sophisticated medical model” in Rebecca’s case may be considered as a modern answer to Engel’s 1977 plea for a better conceptual-explanatory framework for clinical medicine.

For the sake of highlighting the advantages of the sophisticated medical model of mental illness over that of the strong interpretation of the medical model, let’s imagine an alternative scenario in which Rebecca goes *only* to see a psychiatrist who upholds the latter model. This psychiatrist assumes that the approach used to diagnose somatic illnesses is equally as effective for diagnosing and treating mental illness (Black 2005). He thus takes Rebecca’s medical history, but does not ask her to go into any specific details about her family history (apart from medical history), recent life experiences or interpersonal relationships. This is because the psychiatrist believes that non-biological causes (e.g., social or psychological causes) are not relevant for diagnosing and treating mental illness. Rather, he is committed to the idea that mental disorders like depression are brain-based and that “an increased understanding of the physiology of the brain will eventually improve the care of patients with mental illness” (Black 2005, 5). Apart from the drug prescription he gives to Rebecca, a brief itemization of its side effects, and the request for a follow-up visit, the psychiatrist suggests no other modes of treatment (e.g., counseling, other life-style changes, etc.).

The limitations of the strong interpretation of the medical model in the clinical context are to some extent revealed by differences in the kind of care Rebecca receives from the two psychiatrists. In the first case the psychiatrist presented Rebecca with a complex explanatory framework in which to understand her depression and how to restore her health whereas the second psychiatrist provided a single treatment option. Given that a variety of different hypotheses exist to explain the causes of Major Depressive Disorder, and given that there are different available treatments these differences in the two approaches make sense. While it is an empirical question whether any clinical psychiatrists today endorse a strong version of the medical model of mental illness**,** there are good grounds for thinking it has few proponents**.** For example, psychiatrist KevinBlack (2005, 8) claims that the medical model “regards psychotherapy” as “an important part of psychiatric practice” and that few psychiatrists today “condone uncritical acceptance of pharmacological treatments, or uncritical rejection of psychotherapy”.

Before we move on to consider conceptual-explanatory frameworks of mental illness operative in research contexts and their respective usefulness for guiding mental health research, let’s consider some additional implications of the first three conceptual-explanatory frameworks that we have considered.

Consider first the folk-psychological model of mental illness. While it is common to think of mental disorders as exclusively disorders of the mind, folk psychology leaves it unclear what the mind is and how it fits into the physical world. In fact, it is consistent with a philosophical view introduced by French philosopher René Descartes known as *mind-body dualism*. Descartes believed that the mind and body are separate substances; whereas minds are thinking things that are immaterial, bodies are material things that are extended in space and time. Mind-body dualism essentially jettisons the mind and thus, disorders of the mind, from the physical world. Yet insofar as science can only investigate phenomena that are observable, mind-body dualism and thus, folk psychology, seem to place minds and mental disorders beyond the realm of scientific understanding.

Yet, if the mind cannot be understood by science, a lot of negative consequences result. First, persons with mental illness will be considered beyond the hope of science insofar as their illnesses cannot be investigated or cured in the same way that somatic illnesses are. Second, if we think science is irrelevant for understanding and treating mental illness, we may be inclined to believe that persons with mental illness can simply get better on their own and overcome their illnesses with or without the help of their families and friends. This unfairly places the responsibility of having mental illnesses and the burden of overcoming them on those who have them. Yet, in many cases, talking to another person about one’s mental states or experiences or trying to overcome mental illness on one’s own are not viable strategies for getting better. We see this clearly in Rebecca’s case and there are many other cases. For example, persons diagnosed with *schizophrenia,* who suffer from hallucinations and delusions, cannot get rid of these experiences simply by talking about them or willing them away. Although talking about their experiences and having others understand what they are going through provides some relief, pharmacological interventions (e.g., anti-psychotics or neuroleptics) are often required to alleviate some of their symptoms.

Notice that the medical model (both the sophisticated and strong interpretations) has a positive feature insofar as it serves to eliminate some of the stigma associated with mental illness. Oftentimes people with mental illness feel that they are beyond hope because the causes of mental illness are intangible and may remain unknown. Knowing that something tangible in their brains beyond their control may be causally contributing to their illness provides them with some psychological or emotional relief. The medical model allows for the possibility that advances in science will improve our understanding of mental illness, and point the way towards viable strategies for intervention. By assuming mental illnesses have tangible physical brain-based causes, they also afford the hope of a cure.

So where does this leave the framework of folk psychology for understanding mental illness? Neurophilosopher Paul Churchland (1981) has argued that even ordinary folk/non-scientists should abandon this conceptual-explanatory framework for a mature neuroscientific theory of mental phenomena (including mental illness) in a move that he dubs “eliminative materialism”. Philosopher of psychiatry Dominic Murphy advocates for a similar position in claiming that “folk thought may be a poor guide to” individuating different kinds of mental illness (Murphy 2014, 105) and that the mind-brain sciences may serve as a better guide. One problem with abandoning folk psychology in the clinic is that even if we acknowledge that mental disorders are brain-based, research science has not provided us with complete or successful psychological or biological explanations of them**.** Consider neurobiological explanations of *Major Depressive Disorder*. Depression has historically been explained by appeal to imbalances in neurotransmitters including catecholamines like norephinephrine (“the catecholamine hypothesis”), serotonin and dopamine. Most recently imbalances in the neurotransmitter, glutamate, are also thought to be involved in depression. Such shifts in hypotheses have been common for other DSM categories as well, including *Schizophrenia* and *Substance Related and Addictive Disorders*. While scientists know that in a certain percentage of the population that suffers from depression, anti-depressant medications (e.g., selective serotonin reuptake inhibitors (SSRIs)) seem to have positive effects, and also that neuroleptics and antipsychotics may be used to control hallucinations and delusions that accompany schizophrenia, we currently do not have what might be considered adequate explanations of depression, schizophrenia and the vast majority of other phenomena identified in the DSM as mental disorders.

It is also important to recognize that folk psychology will remain (at least for the foreseeable future) the dominant framework that patients and their families and friends use to explain the ways in which they are suffering to each other and to medical practitioners. It is also will remain at least part of the conceptual-explanatory framework that patients themselves use to understand themselves and their illness. If health care professionals devalue the conceptual-theoretical framework that laypeople use to understand their health and well being, in the way that Churchland suggests we ought to, they run the risk of alienating them to the extent that they may not seek treatment. That some talk therapies are effective in treating mental illness is a compelling reason that the folk-psychological model in some form ought to continue to play a role in how medical practitioners understand, explain and talk about mental illness to patients and their loved ones.

Insofar as the biopsychosocial model of mental illness regards psychological factors as causally relevant for explaining and treating mental illness, it is inclusive of the folk psychological model. More specifically, because it views the mind and body as causally interacting it is superior to the folk psychological model. Given that application of the strong interpretation of the medical model to mental illness excludes folk psychology and the mind from both the diagnosis and treatment of mental disorders, it may be regarded as *eliminative*–because it essentially eliminates the mind from the discussion–or *reductive*—insofar as it reduces the mind to the brain—or takes the mind to be nothing over and above the brain. A sophisticated medical model, in contrast, would be closer to the biopsychosocial model.

Now that we have considered some of the basic features of different models of mental illness and some of the implications of using them for the diagnosis and treatment of persons with mental illness in clinical contexts, let’s evaluate the use of these conceptual-explanatory frameworks in research contexts.

3. Models of Mental Illness in Research Contexts

Research contexts differ from clinical contexts in many ways—too many to itemize in a single chapter. However, one important difference is that when scientists conduct research into the causes of mental illness, they cannot consider the complex causal nexus in which mental disorders are situated all at once. Rather, different areas of science decide which causes they want to investigate—sociological, environmental, psychological, neurobiological and genetic (to name only a handful)—and they use different kinds of experimental methods that carve the world up in different ways. Sociologists do field work to assess the impact of social factors on mental illness, whereas neurobiological experiments are invasive and involve the use of animal models. Within different areas of science studying the causes of mental illness, like sociology, psychology, neurobiology and genetics, different scientists investigate different kinds of causes. For example, geneticists may look at different kinds of genes implicated in a mental illness like schizophrenia (e.g., DISC1, COMT, DTNBP1, PPP1R1B). Similarly, neurobiologists may investigate the role of different neurotransmitters (e.g., dopamine, glutamate, serotonin) involved in mental illnesses. What this means is that, at best, each area of science will yield only piecemeal explanations of a given mental illness and within each area of science (e.g., neurobiology), there will be different models (e.g., the dopamine hypothesis of schizophrenia, or the glutamate hypothesis of schizophrenia) on offer to explain it.

Although we currently have a lot of piecemeal explanations for different kinds of mental illness and such explanations have shed light on avenues for therapeutic interventions—some of which have been successful—we still have no cures. Such piecemeal explanations are not considered to be ultimately satisfactory (See Kincaid 2008, Sullivan 2013, Wimsatt 2007). Yet, can the results from different areas of science that study mental illness be fit together in ways that allow us to approximate towards better causal understandings of mental illness and the development of successful treatments? As we learned in Section 2 above, there are good reasons for medical practitioners to cast their nets widely when diagnosing and treating persons with mental illness. Is it similarly necessary for researchers investigating the causes of mental illness and who work in different areas of science to situate their results within the broader causal nexus when providing explanations for mental illness?

The short answer is “yes”. In fact, there is widespread consensus that “integrative” models of mental illness are required if we want to find effective treatments for them (See for example, Albus et al. 2007; Cuthbert and Insel 2013; Insel et al. 2010; Sanislow et al. 2010). However, the same researchers that advocate for integrative explanations of mental illness have argued that current systems of psychiatric classification like the DSM and ICD have to date impeded the development of such explanations, and that before we make any real progress, we need to develop better alternative frameworks. Critics identify several reasons for thinking the DSM is an obstacle to progress in understanding the causes of mental illness. First, the group of scientists responsible for putting forward and fixing DSM diagnostic categories are not identical to the group of scientists conducting the research, and the two groups have different taxonomic aims. The DSM-5 is supposed to offer a *reliable* system for psychiatric diagnosis. Its authors think that just so long as most practicing clinical psychiatrists diagnose persons exhibiting the same sets of observable symptoms similarly (“interrater reliability”), this is sufficient for operationally defining a given mental disorder category. This is in contrast to the criterion of *validity* often operative in research contexts (e.g., Cronbach and Meehl 1955), which is intended to guarantee that scientists revise their classification systems in light of empirical discoveries. As applied to categories of mental illness, validity is supposed to ensure that diagnostic categories do not “lump together” phenomena (e.g., hallucinations, delusions, feelings of worthlessness) that do not belong in the same category or “split apart” phenomena that do (See Craver 2009).

Critics of the DSM claim that committing lumping errors is precisely what current DSM categories likely do, insofar as they “may erroneously place individuals who share superficial similarities but whose pathology springs from different sources into the same diagnostic category” (Lilienfeld 2014, 129). For example, the kinds of persons who satisfy the relevant criteria for being diagnosed as having a *Major Depressive Episode* comprise a diverse group of individuals who, while sharing a set of symptoms in common, likely do not share the causes of those symptoms in common (See Kincaid’s chapter). The causes of Rebecca’s depression, for example, may be similar or different from other individuals diagnosed with depression. This is in part why, in the clinical context, it is important to use integrative explanatory models like the biopsychosocial model, so that we might approximate towards successful therapeutic interventions. However, research scientists advocating for the development of integrative explanations of mental illness think that they can only be attained if we “release the research community from the shackles of the DSM/ICD categorical system” and start categorizing phenomena in ways that reflect what scientists already know about “fundamental circuit-based behavior dimensions” of mental illness (First 2014, 53). In other words, investigators want to use evidence about commonalities and differences in the brain circuits disrupted in mental illness to develop what they regard as valid taxonomies of mental illness. Ideally, they want people like Rebecca to be grouped together with like individuals who share the same underlying pathology, and whose illness may be treatable using the same methods.

Research scientists are not alone in criticizing the DSM. Debates about psychiatric classification in philosophy of psychiatry have centered on the question of whether or not mental disorders are natural kinds. To put it another way, philosophers have been concerned with the question of whether current systems of psychiatric classification pick out true divisions in kinds of phenomena in nature (See for example Kincaid and Sullivan 2013). Some critics have argued that DSM categories fail to detect natural kinds in part because they have been historically shaped by different theoretical considerations that have impeded scientific progress. Murphy (2013), for example, claims that the concept of “delusion” which remains part of the DSM-5 definition of schizophrenia, is a *folk-psychological* concept. The problem with this, he claims, is that (2006, 62), “we want to explain, taxonomize, and conceptualize mental illnesses without being inhibited by folk categories if they impede the search for power, generality, and progress” in understanding mental disorders.

To overcome the limitations of current diagnostic taxonomies, a subset of investigators at the US National Institute for Mental Health, who have advocated for the development of integrative explanations of mental illness have put forward a new framework for thinking about mental illness known as the *Research Domain Criteria* (RDoC) Project. In development since 2010, RDoC constitutes a research reorientation at NIIMH to direct funding away from DSM and ICD “consensus-based clusters of clinical symptoms” and towards research that conceives of current mental disorder categories as “complex combination[s] of disturbances in more fundamental processes, or dimensions of function, that do not necessarily align with currently identified categories of disorder” (Carter, Kerns, Cohen 2001, 181). The RDoC Matrix (See for example Lilienfeld 2014, 131), is essentially a table for organizing and inputting findings from current and future psychopathological research. The basic assumption upon which the matrix is based is that mental illness is caused by disruptions in discrete domains of psychological functioning. There are five such domains identified in the rows of the matrix: (1) positive and (2) negative valence systems and (3) cognitive, (4) social processing and (5) arousal/modulatory systems. The columns of the matrix are intended to reflect the fact that research on domains of psychological functioning spans multiple “levels of organization” from genes to cells to networks to behavior to self-reports and that different areas of science investigate different “units” that each may causally contribute to psychological function and dysfunction. The different areas of science represented in the matrix, however, may be understood to share a taxonomy of functions/functional domains in common. According to Charles Sanislow and colleagues, RDoC “encourages integration of clinical and experimental findings from multiple approaches, including, for example, behavioral, neurophysiological, and genetic discoveries” (2010, 3). Its advocates thus regard integrative explanatory models as fundamental for explaining mental illness.

So, in answer to the question of what kind of conceptual-explanatory framework is optimal for advancing our understanding of mental illness in research contexts, the resounding answer seems to be a framework that integrates information emanating from a wide variety of different areas of science that have historically studied the causes of mental illness. Whether RDoC is the correct approach for reconceptualizing mental illness in ways that facilitate causal discovery and the development of successful therapeutic interventions is something that perhaps only time will tell. It may be that the RDoC is too restrictive in terms of the types of causes of mental illness its proponents are willing to consider. It seems that the categories of psychological functions put forward by RDoC proponents are equally as consensus-based as the DSM categories. It is also important to recognize that for all of its purported faults, the DSM diagnostic categories have been “serviceable for clinicians of varying [theoretical] perspectives” (Horwitz and Wakefield 2007, 97) and have allowed for researchers coming from a wide variety of different scientific backgrounds to share targets of empirical inquiry in common. It is not clear that RDoC categories will be similarly serviceable and whether the implications of adopting it will be positive or negative. Again, perhaps only time will tell. For our purposes it is simply important to recognize that discovering the causes of mental illness will continue to require collaborative efforts.

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**Further Reading**

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**Related Topics**

Gifford’s Chapter 45

Kincaid’s Chapter on Psychiatric Classification

Biographical Note

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