Toward a Philosophical Structure for Psychiatry

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This article, which seeks to sketch a coherent conceptual and philosophical framework for psychiatry, confronts two major questions: how do mind and brain interrelate, and how can we integrate the multiple explanatory perspectives of psychiatric illness? Eight propositions are proposed and defended: 1) psychiatry is irrevocably grounded in mental, first-person experiences; 2) Cartesian substance dualism is false; 3) epiphenomenalism is false; 4) both brain→mind and mind→brain causality are real; 5) psychiatric disorders are etiologically complex, and no more "spirochete-like" discoveries will be made that explain their origins in simple terms; 6) explanatory pluralism is preferable to monistic explanatory approaches, especially biological reductionism; 7) psychiatry must move beyond a prescientific "battle of paradigms" to embrace complexity and support empirically rigorous and pluralistic explanatory models; 8) psychiatry should strive for "patchy reductionism" with the goal of "piecemeal integration" in trying to explain complex etiological pathways to illness bit by bit.

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> Many a psychiatrist has said that he did not want to burden himself with a philosophy…but the exclusion of philosophy would…be disastrous for psychiatry.
>
> —K. Jaspers (1, p. 769)

Whether we know it or not, to practice or to do research in the field of mental health requires us to assume certain positions on several philosophical issues, two of which are particularly central. The first such issue is the nature of the interrelationship of the brain and the mind. The second is to understand how the various explanatory approaches that can be taken toward psychiatric disorders can best be interrelated.

Because our field deals with fundamental questions of what it means to be human, psychiatry is particularly susceptible to preconceptions that can strongly color the value we assign to differing methodological perspectives. With the growth of neuroscience and molecular biology, psychiatry is set to inherit rich insights into the basic workings of the human brain. To maximally use this new information, however, will require that we have our conceptual house in order.

This article seeks to sketch a coherent conceptual and philosophical framework for psychiatry that consists of eight major propositions:

1. Psychiatry is irrevocably grounded in mental, first-person experiences.
2. Cartesian substance dualism is false.
3. Epiphenomenalism is false.
4. Both brain→mind and mind→brain causality are real.
5. Psychiatric disorders are etiologically complex, and we can expect no more "spirochete-like" discoveries that will explain their origins in simple terms.
6. Explanatory pluralism is preferable to monistic explanatory approaches, especially biological reductionism.
7. Psychiatry needs to move from a prescientific "battle of paradigms" toward a more mature approach that embraces complexity along with empirically rigorous and pluralistic explanatory models.
8. Finally, we need to accept "patchy reductionism" with the goal of piecemeal integration in trying to explain the complex etiological pathways to psychiatric illness a little bit at a time.

Grounding in the Mental World

Foundational to this framework is the view that the field of psychiatry is deeply and irreversibly wedded to the mental world. The questions that have played such a prominent role in the history of psychology—whether mental processes can or ought to be studied (2)—are simply not relevant for psychiatry. Our central goal as a medical discipline is the alleviation of the human suffering that results from dysfunctional alterations in certain domains of first-person, subjective experience, such as mood, perception, and cognition. Our nosological constructs are largely composed of descriptions of first-person experiences (e.g., sad mood, hallucinations, and irrational fears). The clinical work of psychiatry constantly requires us to assess and interpret the first-person reports of our patients. Many of the target symptoms that we treat can only be evaluated by asking our patients about their subjective experiences. While we want to take advantage of the many

advances in the neurosciences and molecular biology, this cannot be done at the expense of abandoning our grounding in the world of human mental suffering.

Shedding the Chains of Descartes

An initial task is to confront one large piece of historical baggage. No philosophical concept has been as widely influential in our field or as potentially pernicious in its effects as that of Cartesian dualism. While individual psychiatrists may, for their own personal or religious reasons, continue to advocate mind-body dualism, it is time for the field of psychiatry to declare that Cartesian substance dualism is false. We need to reject definitively the belief that mind and brain reflect two fundamentally different and ultimately incommensurable kinds of "stuff." Rather, in accord with an overwhelming degree of clinical and scientific evidence, we should conclude that the human first-person world of subjective experience emerges from and is entirely dependent upon brain functioning. The mental world does not exist independently of its physical instantiation in the brain. To reject Cartesian dualism (and accept monism, the view that mental and physical processes are both reflections of the same fundamental stuff) means to no longer consider the mental (or functional) to be a fundamentally different thing from the biological (or organic). Rather, the mental and the biological become different ways of viewing and/or different levels of analysis of the mind-brain system.

This rejection of Cartesian dualism requires a significant shift in our way of thinking. Although American psychiatry officially abandoned the functional-organic dichotomy—one of the many echoes of Cartesian dualism—with DSM-IV (3), and the abandonment of dualism has been recently called for by Kandel (4), dualistic thinking and vocabulary remain deeply entrenched in our approach to clinical and research problems. From the ways we organize our clinical presentations to our categorizations of risk factors, we remain deeply imbedded in the Cartesian framework of seeing the mind and brain as reflecting fundamentally different spheres of reality.

One immediate beneficial consequence of a rejection of Cartesian dualism is our confrontation with the misunderstandings that can arise from the claims of what *might* be called weak biological explanation. The rejection of Cartesian dualism logically leads to the conclusion that all psychiatric disorders are biological. Although we should not belittle this claim (that, for example, would eliminate primary spiritual causes of mental illness), the greater danger now is a tendency to exaggerate its significance. By rejecting dualism, we accept that all psychiatric disorders are biological. But so then are all mental processes, pathological or otherwise. The very ubiquity of this claim of weak biology robs it of much of its gravitas. Indeed, if the rejection of Cartesian dualism is correct, then the declaration that a particular psychiatric disorder is biological is a tautology and is as informative as saying, "This circle is round." Nothing new is learned by this claim that was not already evident by the acceptance of a monistic view of mind-brain functioning.

Facing Down Epiphenomenalism

Having rejected Cartesian dualism, we are not yet home free philosophically. Another major viewpoint on the mind-body problem would, if true, also have a profound impact on the field of psychiatry. The core assertion of epiphenomenalism is that the mental world is without causal efficacy, our mental life being simply froth on the wave or steam from the engine. Thoughts, feelings, and impulses occur within our subjective experience, but they do nothing. All the causal action occurs at the level of brain function. Whether and how this assertion can be formally disproven is a subject beyond the bounds of this essay. For the present purposes, I wish to simply assert its falsity and argue that thoughts, feelings, and impulses matter not only because they are responsible for huge amounts of human suffering but because they do things.

Acceptance of Bidirectional Mind→Brain and Brain→Mind Causality

Given our rejection of Cartesian dualism and our acceptance of an integrated mind-brain system, it becomes necessary to accept the concept of brain-to-mind causality. That is, changes in the brain can directly affect mental functioning. In our rejection of epiphenomenalism, we commit ourselves to the concept of mind-to-brain causality. In ways we can observe but not yet fully understand, subjective, first-person mental phenomena have causal efficacy in the world. They affect our brains and our bodies and through them the outside world. (In asserting the causal efficacy of mental phenomena, I am not reintroducing dualism "through the back door." Rather, consistent with several philosophical positions—in particular, nonreductive materialism [5, 6]—I argue that mental processes carry critical causal information about human behavior. For two recent thoughtful treatments of this problem, see references 7 and 8.)

Stop Searching for Big, Simple Explanations

Our strongly held desires to find *the* explanation for individual psychiatric disorders are misplaced and counterproductive. Psychiatry has historically seen a few big explanations, most notably the discovery of the spirochete for general paresis. It is highly unlikely that spirochete-like big explanations remain to be discovered for major psychiatric disorders. We have hunted for big, simple neuropathological explanations for psychiatric disorders and

have not found them. We have hunted for big, simple neurochemical explanations for psychiatric disorders and have not found them. We have hunted for big, simple genetic explanations for psychiatric disorders and have not found them.

Our current knowledge, although incomplete, strongly suggests that all major psychiatric disorders are complex and multifactoral. What we can best hope for is lots of small explanations, from a variety of explanatory perspectives, each addressing part of the complex etiological processes leading to disorders. It will be particularly challenging to understand how these many different small explanations all fit together.

In grieving for our loss of big explanations, we similarly have to give up our hope for simple, linear explanatory models. It will not be "A→B→C→D." Etiological pathways will be complex and interacting, more like networks than individual linear pathways.

Acceptance of Explanatory Pluralism

Introduction to Levels of Explanation

Multiple explanatory perspectives can be adopted in our attempts to understand most natural phenomena. Furthermore, for any given phenomenon, these perspectives will differ in their informativeness and efficiency. It is possible to study scientific questions from perspectives that are both too basic and too abstract. However, currently, the former is a greater concern and so will be the focus of this discussion. The concept of "levels of explanation" is so central to this argument that I will illustrate it with three scenarios.

Scenario 1

Jackie is a physiologist studying hormonal regulation. She accepts that the large biological molecules she is examining are constituted of atoms that are made up of particles that are in turn made up of subatomic particles. However, in seeking to alter certain aspects of a hormonal system that she is studying, she might consult with a biochemist or pharmacologist but not with a particle physicist. Why? Because the kind of effects she wants to produce—the stimulation of particular hormonal receptors—results from the actions of large biological molecules. Knowing what quarks are doing in these molecules will not help her achieve her desired goal.

Scenario 2

Bill is performing a statistical analysis on his computer and is getting the wrong result because he has made a mistake in his statistical program. Being a down-to-earth kind of guy, Bill decides to take off the back of his computer, pull out the motherboard, and reach for his soldering iron, hoping to find a loose connection to solder, thereby solving his programming problem. Why is this the wrong approach? After all, a computer is really just a bunch of circuits and electrons. Using a soldering iron is a highly inefficient approach because it is an intervention directed at the wrong explanatory level in the complex system. The cause of the dysfunction is at the level of high-order computer code and could not be easily perceived or repaired at the level of circuits on a motherboard.

Scenario 3

Kathy, a young psychiatrist, is asked by a distressed parent to consult with her about her son, Brian, who has decided to leave a career in science to enter the priesthood. The upset parent insists that Kathy order a brain scan to find a way to change his decision. "There must be something the matter with his brain, doctor. How could he throw away such a promising scientific career?" Kathy sees the young man, who appears thoughtful and mature, and he describes the deep satisfaction and inspiration he feels in the Catholic religion. He understands the possible hardships ahead of him but feels he is making the right decision. Kathy tells the parent that she is not going to order a magnetic resonance imaging scan. There is no evidence, she states, that there is anything the matter with his brain, and no interventions that would act directly on his brain are indicated in this situation. She feels that he has reached his decision in a reasonable way, but the mother should feel free, if she wants, to try to argue her son out of his decision.

What is going on in these three scenarios? In each case, we have a higher-order system that is completely constituted from lower-order elements. That is, Jackie's macromolecules are made up of subatomic particles. Bill's computer is made up of circuits and electrons. Brian's mental processes are expressed in the biology of his brain. However, in each of these scenarios, an intervention at the level of the lower-order elements is likely to be, at best, inefficient and, at worst, ineffective and possibly harmful.

The Limits of Biological Reductionism

> There is no such thing as a psychiatry that is too biological.
>
> —S.B. Guze (9)

The last several decades have seen a rise to prominence within psychiatry of a biological reductionist perspective. Advocates of this point of view argue that the only valid approach to understanding psychiatric disorders or, more broadly, psychological functioning is in terms of basic neurobiological processes (10). Multilevel models, especially those including mental and social explanatory perspectives, are typically rejected (sometimes with the epithet of being nonscientific or "soft-headed") or accepted only with the caveat that all the "real" causal effects occur at the level of basic biology.

This position might be seen as a logical consequence of the rejection of Cartesian dualism. After all, if we agree that there are no mental processes that are independent of brain function, then should not all the causes of psychiatric disorders be reduced to brain processes? Although this reductionist perspective is understandable in sociological terms as a reaction to prior radical mentalistic programs within psychiatry (e.g., some forms of dynamic psychia-

try) and is appealing because of the ease with which it fits into a medical model, this approach is too narrow to encompass the range of causal processes that are operative in psychiatric disorders.

The limits of biological reductionism are well illustrated by the three scenarios just outlined. Contrary to Guze's assertion, psychiatry can be too biological in the same sense that it would be an error for Jackie to focus on subatomic particles in her physiological research, for Bill to try to fix his problem with statistical analysis by using a soldering iron, or for Kathy to employ psychopharmacology to reverse Brian's career decision. Note that I do not contest that ultimately (in the sense of "weak biology") all psychiatric illness is biological. What is at issue here is the optimal level in the causal processes underlying psychiatric illness at which intervention can be best focused and understanding most easily achieved.

Explanatory Pluralism

In the tradition of other thoughtful commentators (especially Engel [11] and McHugh and Slavney [12]), in place of biological reductionism, I advocate *explanatory pluralism* (13–17) as the approach best suited to understanding the nature of psychiatric illness. Explanatory pluralism hypothesizes multiple mutually informative perspectives with which to approach natural phenomena. Typically, these perspectives differ in their levels of abstraction, use divergent scientific tools, and provide different and complementary kinds of understanding. Explanatory pluralism is especially appropriate for psychiatry because psychiatric disorders are typically influenced by causal processes operating at several levels of abstraction.

A clear example of explanatory pluralism comes from biology, where it is useful to distinguish between "how" questions and "why" questions (18). For example, in examining the large and colorful tail of the male peacock, we could study its developmental biology to clarify physiologically how such a tail develops. Alternatively, we could seek, in the evolutionary history of the peacock, an answer about why the tail develops, presumably through mechanisms of sexual selection. Neither the how/physiological nor the why/evolutionary explanatory perspective can easily replace or invalidate the other. It is simply in the nature of the phenomenon that it can be usefully approached scientifically from two different perspectives.

(The pluralistic explanatory approach outlined in this essay assumes the natural science perspective that Jaspers termed "explanation" [1]. I do not here address another highly relevant question—how does the information acquired from this perspective relate to knowledge obtained, through empathy, from human relationships, through the process termed "understanding" by Jaspers [1]?)

Arguments for Explanatory Pluralism and Against Biological Reductionism

I will now review eight arguments in favor of explanatory pluralism and against biological reductionism or other unimodal perspectives on psychiatric illness (including radical mentalistic accounts). These arguments assume the conclusive demonstration that specific biological processes that are manifest, for example, at the level of genetic risk factors or neurochemical alterations play a significant causal role in all psychiatric disorders.

First, a long clinical tradition and much empirical evidence of increasing methodological rigor point to the importance of first-person mental processes in the etiology of psychiatric disorders. Of the many possible studies, one recent investigation will illustrate this point (19). In a large epidemiological sample of twins, severely stressful life events and onsets of major depression and generalized anxiety were studied. Descriptions of the severely stressful life events were blindly reviewed by trained raters and scored for their level of loss, humiliation, entrapment, and danger. Even though only highly threatening life events were studied, these ratings further predicted the risk of depression and anxiety.

Humiliation and loss are classical, subjective, first-person experiences that humans can recognize in themselves and in others. Although humiliation is ultimately expressed in the brain, this does not mean that the basic neurobiological level is necessarily the most efficient level at which to observe humiliation. Trying to understand humiliation by looking at basic brain biology may be like Bill trying to fix his statistical analyses with his soldering iron. It may be the wrong explanatory level.

Second, a large body of descriptive literature shows convincingly that cultural processes affect psychiatric illness. For example, a recent meta-analysis (20) concluded that rates of bulimia have meaningfully increased in Western countries in recent years. Furthermore, in non-Western countries, the prevalence of bulimia is strongly related to the degree of contact with Western culture (20). One study in Fiji (21) has shown a substantial rise in eating disorder pathology in adolescent girls after the introduction of television and the associated intense exposure to Western ideals about body image. These results suggest that the risk for bulimia is related to cultural models of ideal body size. While culture ultimately exists as belief systems in the brains of individual members of a cultural group, it is unlikely that cultural forces that shape psychopathology can be efficiently understood at the level of basic brain biology.

Third, our first two examples illustrate that, in addition to neurobiological and genetic risk factors, a full etiological understanding of at least some psychiatric disorders will require consideration of psychological and cultural factors. We have, however, been naively assuming a model in which biological, psychological, and cultural factors each independently affect risk. However, the reality is more complex, thereby posing further difficulties for the

reductionist biological model. The impact of genetic factors on the risk for psychiatric disorders or drug use can be modified by the rearing environment (22, 23), stressful life experiences (24, 25), and exposure to cultural forces (26). Recent work in bulimia suggests that this disorder arises given a combination of a biological/genetic predisposition and cultural factors encouraging slim body ideals. The actions of basic biological risk factors for psychiatric illness are modified by forces acting at higher levels of abstraction.

Furthermore, gene expression is extensively modified by both simple (e.g., light-dark cycle) and complex (e.g., learning tasks, maternal separation) environmental stimuli (27), and even relatively gross aspects of neuronal and brain anatomy can be modified by experience (28). A bottom-up hard reductionist approach to psychiatric illness will be futile if basic neurobiological risk factors are frequently modified by higher-order processes, including environmental, psychological, and cultural experiences.

Fourth, biological reductionists assume that neurobiological risk factors for psychiatric disorders operate through physiological "inside-the-skin" pathways. However, an emerging body of research suggests that this assumption is false. Part of the way in which genetic risk factors influence the liability to psychiatric disorders is through "outside-the-skin" pathways that alter the probability of exposure to high-risk environments. For example, genetic risk factors for major depression increase the probability of interpersonal and marital difficulties, which are known risk factors for depression (29). This is not a theoretical issue. If the impact of genetic risk factors is mediated through environmental processes, this opens up new possible modes of prevention.

Fifth, hard reductive models in science strive for clear "one-to-one" relationships between basic processes and outcome variables. Such simple relationships are not plausible for psychiatric illnesses. For example, individual genetic risk factors probably predispose to a range of different psychiatric disorders, depending on other genetic, developmental, and environmental factors (30), and many different DNA variants probably predispose to one disorder (31). This pattern of many-to-many causal links between basic etiological processes and outcomes is more compatible with pluralistic than with monistic reductive etiological models.

Sixth, a series of important questions in psychiatry are historical in nature and not plausibly subject to reductive biological explanations. Why are humans prone to develop depression when exposed to social adversity? Why do genetic risk factors for schizophrenia persist in human populations? Like the puzzle of the peacock's tail, these questions are best answered at historical/evolutionary and not physiological levels.

Seventh, how, using a hard reductive biological approach toward psychiatry, can we define dysfunction (14)? While certain psychiatric symptoms may be pathological at a basic biological level (e.g., hallucinations), many symptoms are dysfunctional only in certain contexts. At a physiological level, a panic attack during a near-fatal climbing accident in a psychiatrically healthy individual or in a crowded shopping mall in a patient with agoraphobia are probably the same. Since many psychiatric disorders include, by definition, some degree of psychosocial dysfunction (32), explanation at the level of biology alone is unlikely to be sufficient.

Eighth, biological systems generally and mind-body systems more specifically have goals and generate processes to address these goals, such as the maintenance of blood pressure or self-esteem and the acquisition of food, sexual partners, or status. As argued persuasively by Bolton and Hill (7), these information-based systems cannot be reduced to their molecular constituents without a loss of explanatory power. After all, the biology of a neural impulse—the influx and efflux of sodium, potassium, and calcium ions—is essentially the same all over the brain. These impulses have specific causal efficacy only through the particular neuronal system in which they are imbedded. Critical causal processes in the mind-brain system can only be captured though an understanding of the higher organizational levels of these goal-directed systems.

What Kind of Explanatory Pluralism Do We Need?

As outlined in an illuminating chapter by Mitchell et al. (33), explanatory pluralism can come in several "flavors," two of which interest us here. *Compatible pluralism* recognizes the existence of distinct and independently meaningful levels of analysis. However, for scientific and/or sociological reasons, research in these distinct levels occurs largely in isolation. In *integrative pluralism*, by contrast, active efforts are made to incorporate divergent levels of analysis. This approach assumes that, for most problems, single-level analyses will lead to only partial answers. However, rather than building large theoretical structures, integrative pluralism establishes small "local" integrations across levels of analysis.

Our field may be in particular need of integrative pluralism, where scientists, without abandoning conceptual rigor, cross borders between different etiological frameworks or levels of explanation. Such efforts may be unusually scientifically fruitful and work bit by bit toward broader integrative paradigms. Recent examples of integrative pluralism in psychiatric research would include the incorporation by Gutman and Nemeroff (34) of early traumatic events into neurobiological models for depression and the efforts by Caspi and colleagues to include specific genotypes in an epidemiological study examining the development of antisocial behavior (35) and depression (25) after exposure to environmental adversity.

Problems With Implementation of Explanatory Pluralism

In mental health research, explanatory orientations are too often adopted for ideological rather than empirical reasons. At its worst, our field consists of mutually antagonistic, noninteracting theoretical camps. One approach to this cacophony of divergent explanatory orientations would be to impose rigidly one methodological perspective, such as biological reductionism. However, this is unfeasible and would be unlikely to succeed even if it could be accomplished.

Rather, our task, the difficulty of which is hard to overestimate, is to establish a methodologically rigorous but conceptually open-minded scientific playing field. Advocating explanatory pluralism for psychiatry should not be construed as a vacuous invitation to treat all methodologies as of equal value. As divergent perspectives compete for resources and students, the deciding factors should not be the orientation of the methods but rather the power of the designs, the replicability of the results, and their relevance to understanding the causal pathways to psychiatric disorders.

Thomas Kuhn (36), the famous philosopher of science who stressed the degree to which science was intrinsically a social activity, would suggest that this agenda may be a fool's quest. He might argue that the competing scientific paradigms within psychiatry are "incommensurable," that their advocates have such widely divergent viewpoints that they effectively inhabit different professional worlds. Furthermore, he would assert that data in our field are heavily theory-laden and deeply intertwined with theoretical assumptions. In such circumstances, effective communication across paradigms and finding a common ground on which the various paradigms might fairly compete would be difficult.

These arguments have force. I recall too many sterile arguments between psychoanalysts, social psychiatrists, and biological psychiatrists in the late 1970s to lightly dismiss Kuhn's contention of the incommensurability of different theoretical perspectives. Furthermore, I remember with surprise the growing realization that in earlier generations, researchers from divergent perspectives had taken the same set of data—evidence that schizophrenia ran in families—and assumed that it proved biological (37) or family-dynamic (38) etiological theories of schizophrenia.

However, Kuhn's perspective may be too pessimistic. Many philosophers of science now disagree with the more radical versions of his claims (39). Getting researchers from different perspectives to agree on broadly similar interpretations of data is not impossible. Within the field of mental health research, we have seen increasing "cross-paradigm" discussions and collaborations. The ideological rancor that characterized earlier debates may be lessening, and the optimists among us might ascribe that to a maturation of the field.

Kuhn argues that to be considered a mature science, a field has to agree on a basic scientific paradigm (36). Psychiatry, by this criterion, would be in an immature "preparadigmatic" state. Although vastly underspecified and in need of being "filled in" in different ways for each of the major psychiatric and drug abuse disorders, explanatory pluralism might form the substrate of such a shared paradigm.

Acceptance of Patchy Reductions Leading to Piecemeal Integration

What should be our goals in seeking to understand the extraordinarily complex casual networks within the mind-brain system and its interaction with the psychosocial environment that lead to psychiatric illness? Another assertion of the biological reductionists is that the value of a causal explanation is directly related to how far down it goes on the causal chain—the more basic and biological the better (10). While tempting, this "zeitgeist" should be resisted.

A thought experiment might help. Imagine that there are 15 discrete levels, with the mind-brain system between DNA on one hand and the clinical manifestations of schizophrenia on the other. Researcher 1 is conducting linkage and association studies that attempt to directly relate levels 1 and 15 but would provide no insight into the intervening levels. Researcher 2 is trying to understand, at a basic molecular level, the actions of a putative altered gene transcript, thereby trying to move from level 1 to level 2 or 3. Meanwhile, researcher 3 is seeking to understand the neuropsychological deficits in schizophrenia, trying to clarify the link between levels 13 and 15. Although biological reductionists might declare the work of researcher 2 to be more "scientific" and valuable because it is more basic, I hope that this thought experiment makes it clear that we can make no such judgments a priori. There are many links in the chain, and their ultimate value and scientific fruitfulness are unlikely to bear any strong relationship with where on the causal chain (or, more realistically, network) they sit.

This thought experiment leads to a final point. Although developing the "grand theory" is attractive and may provide a fruitful heuristic framework, we are not close to developing a full casual network for any psychiatric disorder. Nor should this now be our primary goal. Rather, we should settle for what we have called "bit-by-bit" efforts of integrative pluralism. Schaffner (40, p. 282) has expressed a similar idea in what he calls "patchy reductions" in "a structure of overlapping interlevel causal models." Such efforts should, over time, result in clarification of parts of the causal network from which it may be possible to move toward a more complete etiological understanding of the extremely complex mind-brain dysfunctions that it is our task to understand and treat.

Integration and Conclusions

Working in the field of psychiatry inevitably involves us in some of the most important and perplexing questions that humans can face. Two are of paramount importance for our field: how do mind and brain interrelate, and how can we integrate the multiple explanatory perspectives on psychiatric illness? I have tried to pose tentative answers to these questions in the hope that they might contribute toward providing, for psychiatric research, a pragmatic integrated rubric. We need to move from sterile, ideologically driven debates toward critical, creatively conceptualized empirical questions. How much real explanatory power is provided by the many possible etiological perspectives on a given psychiatric disorder? How can we begin to understand how the various explanatory levels interrelate with one another?

Our hope should be for the scientific maturation of psychiatry that will in turn allow us to use and integrate the coming scientific advances. This will require our moving beyond the clumsy and outdated baggage left us by Cartesian dualism. We should not, however, thereby reject our fundamental roots within the mental and psychosocial spheres or succumb to the temptations of simplistic reductionist models. Psychiatric disorders are, by their nature, complex multilevel phenomena. We need to keep our heads clear about their stunning complexity and realize, with humility, that their full understanding will require the rigorous integration of multiple disciplines and perspectives.

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