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Re-Socializing Psychiatry

Critical Neuroscience and the Limits of Reductionism

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Contemporary neuroscience is advancing our understanding of the role of the brain in psychiatric disorders. These successes, allied with broader social forces, have allowed biological psychiatry to largely displace psychodynamic and social psychiatry, which emphasized the importance of meaning and experience in psychopathology. In contrast to these traditions, biological psychiatry tends to treat experience as an epiphenomenon of neural activity and the social world as an independent set of external stimuli or adaptive contexts. As a result, psychiatry reduces phenomenology to a list of symptoms and signs, and reduces the social world to a set of learned behaviors, attitudes, and social contingencies. In fact, the social world plays a fundamental role in human functioning and experience, with causal effects on mental health and illness. In this chapter we critically review the reductionist picture in contemporary psychiatry and provide illustrations of the importance of the social world in psychopathology from research in social neuroscience and psychiatric epidemiology.

In an editorial in *JAMA* (*Journal of the American Medical Association)* in 2005, Thomas Insel and Remi Quirion, the scientific directors of the US and Canadian national institutes that fund mental health research, argued that psychiatry is a discipline of "clinically applied neuroscience" (Insel & Quirion, 2005). Given their influential positions, this vision of psychiatry is important not only for the immediate future of funding psychiatric research, but for the direction of the whole field. The examples of neuroscience they described as providing a new foundation for psychiatry were drawn mainly from genetics and neuroimaging research. There is no question that these fields have made dramatic progress in recent decades. It is equally clear, however, that psychiatry as currently practiced includes a far more varied and complex array of human problems than can be neatly fitted into a biologically driven nosology, set of theoretical models and corresponding treatments. Twenty years ago, Leon Eisenberg warned of the stunting effects on psychiatry of ignoring either the brain

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or the mind (Eisenberg, 1986). To this we must add the continuing tendency to downplay the social and cultural origins of disability and distress as well as resilience and healing. Defining psychiatry as applied neuroscience valorizes the brain but urges on us a discipline that is both mindless and uncultured. Critical neuroscience can work against this conceptual shrinkage to locate psychiatric research, theory, and practice in a wider social, cultural, and political world.

Critical neuroscience aims to trace the social origins and implications of claims like those of Drs Insel and Quirion. Behind their enthusiasm for neuroscience as a foundation for psychiatry is a reductionistic view of the origins and nature of human behavior and experience as rooted in neurobiology. This neuroreductionism seems attractive and even compelling for several reasons: (1) the technologies of neuroscience have made the activities of the brain visible in new and vivid ways; (2) in some instances, neuroscientific research has generated partial explanations for specific symptoms, diseases or disorders; (3) in the social sphere, neurobiological explanations for mental illness have been embraced by many because they shift causality away from human agency and so work to exculpate individuals and their families as the causes of their own suffering; (4) the biological turn has been heavily promoted with many inflated claims because this serves powerful interests in the pharmaceutical industry; and (5) more broadly, the emphasis on neurobiology diverts attention from social, structural, and economic factors that are politically contentious. Ultimately, neurobiological reductionism in psychiatry serves a larger ideology that locates human problems in our brains and bodies rather than in our histories and social predicaments.

In this chapter we want to challenge the logic of this neuroreductionist program, especially as it applies to psychiatry. Our position can be expressed simply: the social environment makes a difference to mental life and to mental illness. Therefore, a reductionist psychiatry which restricts itself to the processes inside the brain is doomed to be incomplete. We begin by surveying some types of reductionism and challenge its commitment to the idea that a single level of explanation of human behavior is possible. We then illustrate the importance of social processes in psychopathology through examples from social neuroscience and social psychiatric epidemiology. Finally, we consider why, despite the obvious importance of higher order cognitive and social processes in psychiatry, many continue to believe that the future of psychiatry rests with the discovery and clinical application of lower-level biological explanations.

Varieties of Reductionism

Reductionism has many forms or versions, encompassing methodological strategies, ontological claims, and epistemological commitments. Some forms of reductionism are useful while others may promote work that is profoundly misleading and potentially damaging to individuals, groups, and communities. Conflating the different forms of reductionism makes it hard to see the virtues and costs of each.

In the domain of psychiatry, there are at least three different versions of neuroreductionism to distinguish:

(1) *Methodological reductionism* assumes that it is a necessary and sufficient methodological strategy to break down complex systems and phenomena into simpler components or analogues to study. This includes focusing on animal models—even though these cannot address the more complex processes of narrative construction, reasoning, or imagination—and studying simple unidirectional or linear causal effects, even when it is clear that most biobehavioral systems involve circular feedback loops or mutual causality. Even when psychological processes are recognized as important, the assumption is that clinical science can advance by approaching such higher order phenomena (like pathological behavior and experience) in terms of lower-level (neurobiological) processes.

(2) *Ontological reductionism* claims that the higher order phenomena are constituted by the lower, that is, that there is no additional entity that is introduced to give rise to these higher order (mental) phenomena. Thus, mind is nothing other than the brain (or the brain at work) and we can, therefore, ultimately dispense with our folk language that treats the psychological (or social) domain as something distinct.

(3) *Epistemological reductionism* argues that there is no need for information about the higher order levels to explain human behavior and experience; everything that can be or needs to be known can be derived or deduced from our knowledge of lower order mechanisms. Hence, self-reports can ultimately be by-passed when we can measure what is going on inside the other person with a brain-imaging device like the philosopher's science-fictional "cerebroscope." In seeing that certain patterns of brain activation have occurred, we would have all the same information about the person we derive from statements like "I am in pain" or "I see red" or "The CIA has planted a bug in my brain."

Methodological reductionism has proved an enormously productive strategy for the advance of science—though it always risks losing sight of the crucial phenomena to be explained. In fact, successful reduction often depends on using the higher order phenomena to guide the search for lower level explanations and to recognize an adequate explanation when it has been found. The mathematical biologist Robert Rosen (1968) made this argument using the example of the relationship of statistical mechanics and thermodynamics. The kinetic molecular theory represents one of the best examples of a successful reduction; it shows how the macroscopic thermodynamic properties of a gas can be reduced to (explained by) the movements (dynamics) of the particles making up the gas. However, there are an immense number of ways to describe the ensemble of gas molecules and so, Rosen argued, the rules of statistical mechanics could only be discovered because an adequate description of the macroscopic properties existed against which to develop and test the lower level theory.[1] Therefore, even the most successful cases of reductionism in science argue against the adequacy of a program of research focused only on the simpler (lower-level) system as the sole methodological strategy.

[1] Indeed, the existence of molecules themselves was demonstrated through macroscopic phenomena like Brownian motion.

On the face of it, human beings are comprised of many systems at many different levels of organization: molecules, organelles, cells, tissues, organs, physiological systems, neural circuits and information processing systems, psychological faculties and functions (some of which have been called "modules"), memories, schemas and other knowledge structures, habits and dispositions to respond, patterns of interpersonal interaction, and so on. There may well be additional levels between these well-identified levels of structure. Clearly, there is no need to posit different types of substance to encompass these different levels of organization: they are all biological in the sense that there is an unbroken continuity of material constitution as one moves up and down the hierarchy. Ontological reductionism in the form of physicalism is widely accepted as part of the scientific worldview (though challenged in some traditions as still in conflict with religious values that insist on a fundamental dualism or supervenience of the spirit and the sacred in human existence). One version of this physicalism results in eliminative materialism: the idea that we can dispense with our notions of mind and experience and replace them with an empirically grounded vocabulary of neural or brain processes.

Nevertheless, explanations of human behavior employ multiple sets of conceptual models or descriptive languages that reflect different levels of organization: the social level of interpersonal relationships; the psychological level of cognitive schemas, motivations, and emotions; the neuropsychological notions of brain functions, regions, and circuits; the neurophysiological vocabulary of axons and synapses; the molecular language of neurotransmitters and receptors; and so on. Reductionism assumes that the higher levels in this list of descriptive languages either have no causal efficacy or else can be explained entirely in terms of the lower level descriptions. This means that we can dispense with the higher order language and replace it with a more fundamental conceptual vocabulary that will yield complete explanations.

The "decade of the brain" witnessed a thorough biologization of psychiatry, justified in part by this reductionist view. In psychiatric theory, reductionism amounts not only to a basic confidence in the adequacy of neurobiological mechanisms to explain psychopathology, but in a preference for lower level explanations. In this view, molecular biology represents the most basic descriptive and explanatory level of psychopathology. This reductionist view ignores the extent to which neurophysiology, psychology, interpersonal interaction, group and family process, and other social processes represent emergent levels of organization with their own structure and dynamics (Morowitz, 2002).[2] As such, these levels require their own languages of description and provide their own modes of explanation (Prosser, 1970). In a sense, they are all "biological" in so far as we are dealing with a single (material) world with many hierarchically structured levels of complexity. However, this is a systems biology that makes use of concepts and constructs from many disciplines to describe

[2] A thorough-going reductionism seems to require that one aim for reduction to the most fundamental of the sciences—physics. Even if one restricts oneself to those sciences that are most basic to the phenomena of interest—here, mental phenomena—then presumably one should aim for a reduction to molecular biology. But that seems absurd. A satisfactory theory of the mind given exclusively in terms of neurons (say) would surely count as a reductionist success. Whereas reductionism aspires to fundamental explanation, science aims at good explanations, at whatever level they can be found (see Fodor, 1997)

and explain basic processes. The systems involved are not only molecular or neurophysiological but also social and cultural.

Indeed, there is a substantial literature on neural networks that demonstrates how even simple systems can exhibit complex properties (Scott, 2002). However, the implications of this for psychiatry are not always drawn out. Instead, we follow a "neo-humoral" approach of treating disorders as the result of too much or too little of some neurotransmitter. Psychopathology, on this view, reflects a chemical imbalance. This is not only a way to simplify the complexities of neurochemistry for popular consumption—in a form that fits with prevalent metaphors of balance and harmony as intrinsic to well-being. It is used equally in clinical texts on psychopharmacology and in research on animal models of psychopathology. This model ignores the fact that neurotransmitters do not map in any simple way onto specific functions, behaviors, or disorders. Neurotransmitters are associated with pathways that perform different functions in different circuits and generally do not code for a specific type of information processing or adaptive system. As a result, a drug treatment that affects one type of neurotransmitter or receptor will have an enormous number of concurrent effects. However, the neo-humoral approach to partitioning psychiatric disorders into categories based on their putative association with disturbances of specific neurotransmitters fits with the technology of psychopharmaceuticals and so it serves powerful economic interests.

The architecture of current psychiatric diagnostic systems was underwritten by observations of the differential effects of certain classes of medication on psychiatric disorders (Healy, 2002; Wilson, 1993). In particular, the distinction between schizophrenia and bipolar disorder became very important when evidence accumulated that lithium had some specificity for bipolar disorder. Antipsychotic or neuroleptic medications, by contrast, were clearly effective at suppressing psychotic symptoms across a very wide range of different disorders. The simplification imposed by a psychiatric nosology organized according to drug classes works in part because manufacturers exaggerate the specificity of medications (most of which, in fact, work for a wide range of symptoms) and patients and clinicians are encouraged to focus on one salient therapeutic effect and ignore all of the other effects—or to view them as more or less troublesome "side-effects."

Accounts that try to explain behavior in terms of neurotransmitters often jump levels, leaving out the interaction of networks and circuits that traverse the brain—a highly differentiated "organ" with many anatomically distributed subsystems. This seems to represent a confusion between a reductionist viewpoint—which can and should make use of a wide range of biological data—and the (unargued for) view that there is a privileged biological level at which deep explanations of mental life are to be found. Similarly, attempts to correlate activity in specific regions of the brain with behaviors leave out the intervening processes of coordinating perception and activity over time. These leaps across levels sometimes work because some problems can be traced to a global problem at the level of neurotransmitters or other cellular or biochemical processes. However, the ultimate expression of most developmental problems depends on individuals' unique learning history (their character, personality, and idiosyncratic psychology) and the environmental contexts in which they live (their social world). These other levels can sometimes be minimized or ignored

because: (1) there are developmental trajectories that are influenced by isolable changes in single genes or other crucial steps in epigenetic processes that persist over time and across diverse environments; (2) there are final common pathways or "attractor basins" such that the developmental history of the brain's networks do not matter much for the final forms of pathology; or (3) some of the "degrees of freedom" (types of behavior) associated with a new level of organization are held constant so that the dynamics of the system can be described in fewer dimensions or simpler terms (that is, the levels of a single neurotransmitter). This last simplification also can occur because we take our psychological constructions and social worlds as static and unchangeable.

On the other hand, it is easy to construct models of even a few interacting neurons (cell assemblies, circuits) that exhibit very complex behavior and a whole range of perturbations that could have various pathological effects. In particular, it is possible to construct a system in which the parameters associated with neurotransmission are all within "normal" limits at each location initially, but the effect of the overall pattern is to create instability or mutually amplifying interaction that is abnormal. The essential point is that systems have different dynamical properties than their components—and systems of systems have still other dynamics. As a result, each level can have its own pathological dynamics that arise from patterns of connection and coordination that are not reducible to the activity of single units—or even families of units grouped together on the basis of their use of a common neurotransmitter or other molecular characteristics. Reduction to a different level may fail to capture the patterns of interest. Systemic pathologies cannot always be reduced to problems with components of the system. The trouble may lie in the connectivity, circuitry, or activity of the system as a network—and, in the case of psychopathology, the relevant networks may include loops through the social environment of family, community, and society.

The picture given to us by biology then is of a hierarchy of systems with emergent levels of structure and dynamics at each level. Emergence, in the sense used here, refers to the appearance of new structures and dynamics in a system that were not present in the elements of the system (Bedau & Humphreys, 2008; Meehl & Sellars, 1996; Morowitz, 2002). The notion of emergence recognizes that systems have properties that are not present in their components. This is true in a trivial sense for most things: a house made of bricks gives shelter in ways that an individual brick, or even a heap of bricks, does not. But to count as an interesting case of emergence, the new level of systemic organization must have radically new properties that cannot be predicted from the properties of the components or from simpler systems. In fact, there are many examples of phenomena that occur only in the context of the larger system; and even when the rules of interaction of the components are known it may not be possible to predict the system's properties except through modeling or simulation of the system as a whole. Even when prediction is possible, it may not be the case that the more "fundamental" level of description is the most perspicuous; the emergent level may provide more illuminating explanations. Even if molecular genetics were reducible to fundamental physics, for example, it does not follow that physical genetics would be a better theory. The structure of the phenomena may be most clearly revealed at the molecular level.

Nonlinear dynamical systems display a wide range of emergent phenomena that are not obvious from the rules that govern the interaction of their components (Mainzer, 2004; Nicolis & Prigogine, 1989).[3] These dynamic properties require new languages of description. The processes of one level organize themselves to create the structures of the next higher level which then allow new processes to occur (Pattee, 1973). An example would be the assembly of the receptor proteins at a synapse that allow neural transmission. Looking from the top down, macro-structures like a synapse can be decomposed into molecular processes. But describing the synaptic arrangement of the molecular processes requires additional sorts of information, to characterize how multiple components are arranged in space and time in relation to each other to give rise to new processes. These arrangements then give rise to specific dynamics with new properties not present in (and, arguably, not even inherent in) the elements of the lower level. The properties inhere in the *arrangement* of the molecular components, not in the components alone. It is the arrangements, organization, and spatio-temporal pattern that supply the missing ingredients needed for processes to emerge and move from one level to the next. This arrangement may be spontaneously self-assembling (as we assume it was in the origins of life or in the developmental processes of embryogenesis) or receive top-down influences from previously constructed higher order structures of greater or lesser complexity (Kauffman, 1993). Even when it appears spontaneous or autonomous, such emergence always involves specific environmental circumstances—at least in terms of the energy supplied to an open system but often in terms of the ordering effects available from interactions with other external structures. Thus, the higher level of order or organization may not be exclusively constituted by or dependent on the lower level, local system but depends also on cooperative interactions with an emerging "macro" level or environmental context that surrounds the local system.

Against the assumptions of methodological and ontological reductionism that would direct scientific (and clinical) attention to the fundamental building blocks of nature as holding the ultimate causal efficacy and explanatory power, the hierarchical systems view of nature introduces orders of magnitude of complexity and requires that we consider a local system in its interactions with an environmental context that is partly shaped and constituted by the emerging system itself (Rosen, 1991). Studying these processes of *autopoeisis* requires specific methodologies that examine systemic properties that cannot be found in (or even predicted from) the isolated components (Kauffman, 1993; Maturana & Varela, 1980).

There is debate about the sense in which these emergent levels are really ontologically distinct. Certainly, they are all physically instantiated with the same raw materials that make up the rest of the world, but their new properties (complex behaviors,

[3] A linear system can be reduced to a weighted sum of its components, which leads to the notion of linear causality (Scott, 2002): if a certain cause C_1 leads to an effect, E_1 and another cause C_2 leads to effect E_2, then the co-occurrence of both causes will lead to a state that is a sum of the two independent effects, that is $C_1 + C_2 \rightarrow E_{1,2} = E_1 + E_2$. In contrast, nonlinear systems do not have such independent effects of causes on outcomes and hence we can speak of nonlinear causality in which $C_1 + C_2 \rightarrow E_{1,2} \neq E_1 + E_2$. The system is literally more (or other) than the sum of its parts. Much has been learned about a variety of nonlinear dynamical systems but many systems remain mathematically intractable and can only be studied through computer models or other analogues.

reproduction, self-repair, adaptation to new environments) seem substantially different in kind from those of simpler systems, in that they demand different theoretical formulations and may, in turn, be more or less informative about the mental phenomena of interest. It is more contentious whether this systems view demands a new epistemology of science (Maturana & Varela, 1987; Wolfram, 2002). However, the higher levels of organization of the nervous system do pose special problems for our notions of the nature of knowledge—of what can be known—and how we come to know it.

Ontologies of Mind

Social factors are implicated in the development of mental phenomena. Where does this leave the question of reduction? Methodological reductionism, as a set of prescriptions about how to do science, is largely a pragmatic question. How best to decompose a system for study is a question that continues to be addressed in the conduct of scientific research itself. No one doubts that along with taking a system apart, one must also be able to reconstruct its functioning within a successful theory. As a result, methodological reductionism can be taken as a family of techniques that are demonstrably effective for studying particular processes but that must be guided by theories of the higher order phenomena that the reductionist method aims to address.

Ontological reduction is, perhaps surprisingly, an area of continuing controversy. Leaving dualist options entirely to one side, the fact of the significant interactions between psychological processes and the environment raises the possibility that mental life requires more than the brain; it can include tools or aspects of the outside world crucial to mental life. There is a long tradition arguing that mental life extends into the environment through processes of embodiment and enactment. The anthropologist Gregory Bateson (1972) argued that tools were extensions of mind, which emerged from a social ecology. Maturana and Varela (1980) argued that cognitive processes could only be understood in terms of the organism's interactions with the environment. These theories do not claim that mind is a different substance than body, but that there are emergent processes that are new and different in substantial ways from the prior or lower level of organization from which they arise. In some sense, therefore, they represent new phenomena with distinct ontologies.

A controversial version of this "extended mind" hypothesis was articulated by Andy Clark and David Chalmers (1998; see also Clark, 2008) in a paper in which they offer the following simple thought experiment: seeing an advertisement for an exhibition at the Museum of Modern Art (MoMA) in New York City, Inga remembers that the MoMA is on 53rd Street and starts walking in that direction. Otto also sees the advertisement and decides to visit the exhibition. Unfortunately, Otto suffers from Alzheimer's disease and is losing his memory. In an effort to cope with the disability, he has begun to carry around a notebook in which he keeps various bits of information. Consulting his notebook, Otto finds that he has written in it the address of the MoMA. With the address now available, he too heads in the direction of the museum. Clark and Chalmers argue that there is no principled reason to think that Otto's

notebook is not part of his mind, despite the fact that it is, of course, not part of his brain. Since it performs precisely the same function as Inga's memory, it is no more than prejudice in favor of the biological that leads us to exclude it from the domain of the mental.

It seems clear that the mind does not involve a new physical substance but there are nevertheless new sorts of processes, entities, and events that come into being as a result of social arrangements and interactions that may both augment and constrain brain activity (Hacking, 1999; Searle, 1995). Recognizing the importance of the social world could lead one to reject ontological reductionism even though one does not believe in non-physical entities like souls. The very words you are now reading emerged from a collaboration between two authors that has resulted—we would like to think, at any rate—in an intellectual product that is more than the sum of its parts. If one were inclined to see the mind as extended beyond the skin, then social interactions of an intellectual kind would regularly engender cognitive activity that would be *ontologically* different from the mental activity of a single person.

The social arrangements of interpersonal interaction can give rise to new sorts of cognitive and brain activity. Some of these interactions are governed by rules and institutions, others by the physical configuration of space and place. To the extent that we accept that the social world creates new sorts of things with their own structures and processes, we can speak of new ontologies, with a social and cultural history and a contemporary politics (Hacking, 2002).

Whether or not we grant the social world a distinct ontological status, it clearly can be decisive for individuals' health and illness (Wilkinson & Pickett, 2009). Psychiatric theory and practice therefore must include knowledge of social context. The crucial question then is what a social view of mental illness does to epistemological reduction. Commitment to this form of reduction is the most theoretically and practically significant because it is here that the question of the right approach to a science of mental illness must be decided. Leaving aside the question of the extended mind, there is broad agreement that the mind is ontologically nothing over and above the interactions of brain, body, and environment. But that fact does not constrain what theories of the mind or mental illness will turn out to be correct, any more than the fact that the universe is made up entirely of quarks implies that every scientific theory must be a theory of quarks. Larger-scale phenomena have their own dynamics and hence require their own languages of description of macro-level processes. "The world," as Jerry Fodor (1997, p. 162) puts it, "runs in parallel, at many levels of description."

Even with respect to the brain itself, there is controversy over the levels of description needed. While there may be wide agreement among neuroscientists that the emergent levels of organization seen in the nervous system do not require a different physical ontology, it is less clear whether they require a different epistemology. Cognitive systems are intentional—they refer to events in the world and can only be rightly understood as parts of loops that involve perception and action in the world. This leads to an epistemological problem when efforts are made to understand the cognitive system by isolating it from the environment. This dilemma is still more contentious when one considers the phenomena of consciousness and self-awareness. Whether or not subjectivity requires different ontology (following Chalmers (1996), Jackson (1982), and Nagel (1974)), it certainly requires a different epistemology. Moreover, this

epistemology must not only respect the privileged (though also biased and distorted) perspective of the subject and the role of their agency in constructing both their own experience and the larger social world, but also the emergence of many aspects of mind and self through that self-constituting interaction with the social world.

Subjectivity and the Social Construction of the Self

A special type of emergent phenomenon characterizes the human brain: that is the ability to construct representations or descriptions and to operate at this logical level. Abstractly, this is what makes the brain utterly different than the liver or the lungs. The brain does not secrete or exchange information with the world the way other organs operate: it manipulates patterns. This has been captured in the notion of the brain as simultaneously a dynamical system and a cognitive or linguistic system (Pattee, 1977). Another analogy that leads to a similar distinction was introduced by von Neumann in his comparison of the brain and the digital computer: both require hardware and software to process information (von Neumann, 1958). In principle, these are distinct and dissociable.[4] For digital computers, the hardware may vary in speed and other characteristics but as long as it can carry out a basic set of computations it suffices to run any conceivable program. In reality, of course, knowing the characteristics and constraints of the hardware allows programmers to devise more efficient programs that run especially well on specific hardware. In the case of the brain, the software is instantiated as changes in the hardware; that is, the abstract manipulation of symbols and its physical realization in terms of neural networks are thoroughly intertwined. The structure of the brain exerts constraints on what is easy or difficult to compute—resulting not only in the limits of specific cognitive abilities but in the bounded nature of everyday rationality and our propensity for certain types of systematic biases, errors and akrasias.

The programs that are inscribed in the brain reflect our developmental histories and the demands of the contexts or environments in which we dwell. A unique set of these programs concern our abilities to monitor, represent, control, and reflect on our own behavior and experience. These control processes include efforts to match or reconcile our behavior to various standards we have, some of which are attached to a sense of our social personhood or to our subjective sense of selfhood. A lot goes on both in and around the construction and reconstruction of the sense of self as one or more images, plans, or narrative centers that include a sense of personal history (grounded in memory), agency, and subjectivity.

[4] The links between hardware and software may include the ability of software to modify hardware—this lay behind von Neumann's notion of self-reproducing automata (von Neumann, 1951). Because any physical instantiation of a program requires energy to make order out of disorder in the course of its computations, running a program inevitably has physical effects on the substrate that conducts its computations. Thus, a program that runs in a rapid loop could overheat the processor and set the machine on fire or cause a meltdown. Even computers, therefore, have bidirectional causal pathways between hardware and software. Nevertheless, the functioning of the software (the linguistic level) can be described in hardware-free terms and has its own logic and "pathologies."

The sense of subjectivity and selfhood we experience from the inside interacts with a social construction of personhood seen from the outside. As persons then, we have emergent levels of organization of behavior associated with subjectivity and self-awareness and with our social roles and the corresponding responses of others. The self cannot be fully reduced to any lower level of structure or representation.[5] The self is not an arrangement of synapses and the cultural world is not an aggregate of individuals' cognitive or neural representations. The brain cannot stand in for the person and the person cannot stand in for society or culture. So, to achieve and maintain a person-centered viewpoint, we need to understand the ways in which people use and are used by their brain and their culture.

The recognition that, as subjective agents, we are not simply manifestations of brain activity but that we *use* our brains, reflects the supervenience of the self as an organizing system that can reflect on and work with the idiosyncrasies of the brain and the body it inhabits. Our brains are plastic and pluripotent and we can feed and nurture them or abuse them with chemical substances we ingest or experiences we seek out—indeed, we can choose to expose them to new environments where they are shaped, sculpted, and transformed (Malabou, 2008).

On the other hand, conscious self-direction is not the only determinant of behavior. Non-conscious cognitive processes and non-cognitive regulatory processes—like the activity of the cardiovascular system or the gut—constantly influence our behavior (and our experience of agency). Some of these non-conscious processes may organize behavior in a planful or purposive way. In a sense then, to the extent we identify the self with the conscious "I," we might think of the brain as using us for its own purposes, compelling us to do things we would rather not (Wilson, 2004). The awkward locution of "being used by one's brain" is not meant to misplace agency, but to counteract the tendency to exaggerate the autonomy and agency of the self that comes from a person-centered view of the world. It also opens the door to recognizing that our brains can betray us or can be hijacked by others—the domains of psychodynamic theory and the social psychology of persuasion, respectively, each with its own hermeneutics of suspicion.

Similarly, contemporary social sciences tend to exaggerate the agency of the individual against the constitutive and countervailing forces of the social world. Ascribing agency and purpose to society is not meant to personify impersonal networks (though for groups and communities this does make sense), but to acknowledge that we live

[5] Though, to the extent that the self reflects distinctive patterns of responding to context, it may be partially inscribed in lower level dispositions to respond, that persist even when self-awareness is damaged or constricted. Consider, for example, the person with Alzheimer's who, while showing an alteration of personality or "loss of self" (Cohen & Eisdorfer, 2001), nevertheless, reveals flashes of their old self in certain turns of phrase, emotional responses, or other patterns of behavior. The self, like other complex representational processes, may be holographically distributed in the brain so that destruction of some areas does not simply eliminate its processes but degrades their specificity or detail; much as cutting up a hologram results not in a fragment of the original image but in a blurry version of the whole image. The notion of distributed networks in the brain has a long lineage that antecedes the invention of holography (Pribram, 1990). Of course, to the extent that the self resides in (or is sustained by) interactional processes, its preservation or loss—in Alzheimer's or other neurological disorders—depends on interpersonal processes (how others perceive and respond to the afflicted person) as well as on the neural machinery of memory and self-reflection (Herskovits, 1995).

in and among dense networks of interpersonal and institutional processes that shape our developmental trajectories. These processes are not expressions of a passive social matrix in which we can freely locate ourselves, but are themselves determined by political and economic interests. One way in which these larger political-economic formations influence us is by structuring the social worlds that afford us identities, power, and purpose. They underpin the collective notions of personhood that define our goals and aspirations. They influence the narratives that regulate our sense of autobiographical memory and identity and the forms of embodiment through which we acquire our sense of self. And, with technologies both old and new, they may reach past the self to directly manipulate the neural substrates that subserve the programs of the self.

In the face of this complex hierarchy of levels of organization and the emergence and supervenience of subjectivity and agency, the epistemology of biomedicine requires some rethinking. Biomedical practitioners generally assume that we can treat verbal reports as more or less accurate indices of bodily experience (Kirmayer, 2008). When a patient says "I am in pain," the assumption is that there is a specific physiological process (or one of a family of processes) going on in the body and the brain that yields a specific experience, which the person can then reliably report. Of course, patients may be "unreliable historians" and either exaggerate, minimize, or deny their experience. But this only reinforces the sense that the normal condition allows a direct link between bodily events, symptom experience, and clinical presentation. With such naive semiotics, biomedicine ignores the way in which experience is shaped by an array of psychophysiological and psychological processes that depend on past learning, cognitive schemas, memory, and attention. In addition to this cognitive and attentional mediation, both experience and its verbal report depend on context and may involve more or less conscious attempts at rhetorical self-fashioning and positioning. A symptom report, autobiographical story or response to a question, must then be understood not just in terms of the individual's history but also in terms of their relationship to the interlocutor, to unseen participants in their social world who wait beyond the doors of the consulting room, and indeed, in terms of the circulation of ideas and ways of construing oneself in local communities and global systems (Kirmayer, 2000).

The complexity, ambiguity, and indeterminacy of verbal reports is not simply a matter of "noise" in a communication channel confounding what would otherwise be a clear communication. There are aspects of experience that can only be known in and through language because they are made up of language in the first place and reside in cognitive structures and corresponding ways of thinking, or else are located in a conversation as a discursive formation or way of speaking. On this view, knowledge and experience are socially constituted and not reducible to an internal representation in the mind or brain of an individual (Bloor, 1983). Nor is it merely a matter of an "epidemiology of representations," each carried by an individual and distributed according to social position (Sperber, 1996). Rather, the discursive formations that constitute complex experiences of selfhood reside in culturally constituted forms of life.

This points to an important limitation of current work in social neuroscience which, despite its recognition of the importance of the social world in the evolution and

development of the brain, tends to focus on lower-level biological phenomena (Insel & Fernald, 2004). For example, studies that show how important the neurohormones oxytocin and vasopressin are for our feelings of love and attachment, have important consequences, including alerting us to the possibility that psychiatric treatments like SSRIs might undermine romantic love and stable attachments in couples (Fisher, Aron, & Brown, 2006). But this model captures only a small part of the tapestry of thoughts and feelings, interactions, and interpersonal responses that go into the experience of different forms of love. Recognizing the power of a hormonal system may give us an understanding of some of our vulnerabilities and some leverage in responding to the human predicament—but it does not eliminate the choice of stance and strategy to pursue our lives. That requires a different level of analysis and a different language of description.

We can see this in studies on the psychobiological effects of an affectionate hug, in which holding another person close for a time stimulates the release of oxytocin, which in turn causes feelings of comfort, calm, trust, and, eventually, attachment to the other (Carter, 1998; Insel & Fernald, 2004). The more frequent the hugs, the greater the oxytocin release and the stronger the induced feelings of calm and trust, with health benefits in terms of reducing heart rate and blood pressure (Light, Grewen, & Amico, 2005). But the effects of oxytocin interact with contextual factors that shape the meaning of the embrace. Women in a warm, supportive relationship experience stronger oxytocin effects in response to physical contact with their partner (Grewen, Girdler, Amico, & Light, 2005). Of course, even before any contact, we have the opportunity to anticipate and interpret the meaning of an embrace, which may be desired or unwanted, socially appropriate, or transgressive. And during the embrace, thoughts and competing emotional responses can give the experience layers of reinforcing or contradictory meaning that may override any hardwired or previously learned propensity to respond.

On a larger temporal and social scale, love involves a refiguring of our personal identities, biographies, and life trajectories. We locate ourselves in relation to the loved one, and space itself is reoriented to define the familiar places of hearth and home and the unfamiliar spaces of the public realm, which are progressively more unfamiliar. So love involves cognitive maps as well, even if there are some contour lines drawn by gradients of comfort and response that are based on experiences linked to hormonal mechanisms of attachment. The affective systems revealed by social neuroscience interact with other biobehavioral systems, as well as cognitive and interpersonal processes to create a map of our local social worlds with hills and vales corresponding to places of safety and danger, comfort and distress. But this is only a sketch of a social world, with its own exigencies, that exceed in complexity any of our cognitive constructions. Love and marriage have their own interpersonal dynamics that are not reducible to psychological or biological processes (Gottman, Murray, Swanson, Tyson, & Swanson, 2002). In addition the local system of a marriage is embedded in larger social institutions that regulate its meaning and durability.

Social neuroscience certainly gives us insight into the dynamics of attachment in prairie voles and other animals and the same systems can be shown to be operating in humans. At the same time, it is unclear how far this takes us in an understanding of human love and attachment. As Insel and Fernald (2004) note, "Less clear is the

relevance of these observations to the primate brain, where visual processing trumps vomeronasal signals and cortical networks may override the neuropeptide signals from the hypothalamus." (p. 715). It is not simply that visual processing or wider cortical networks have more influence in the primate brain but that, in humans, vision and cortical associations bring information about others in a complexly configured social world. In what sense, then can we view love as "an emergent property of the nervous system" (Porges, 1998)? The social meanings of love are only possible because of the autonomic and neurohormonal systems that enable certain types of strong emotional response, memory, and attachment to others. At the same time, the neural systems that contribute to feelings of comfort and attachment only become the processes we call "love" given the socially guided use of our cognitive capacities for desire, imaginative fantasy, and commitment (Griffiths & Scarantino, 2009; Gross, 2006; Reddy, 2001). Deprived of its biological substrate, love would be a weak or non-existent force in the world; deprived of its social history, embodiment, and enactment, it would be literally unimaginable.

Social Origins of Psychiatric Disorder

The failure of a reductive epistemology of the mind can best be seen when we reflect on the role of self and personhood in psychopathology. The social world allows us to recognize certain aspects of our self-fashioning and compels us to treat other aspects as natural or given. Cross-cultural comparison is important then not only to respect human diversity, but to look behind the curtain of our commonsense constructions of the person—which may not only serve vested interests but obscure the very processes that constitute mind itself. It is always easiest to see this by looking at other peoples' cultures. The field of cultural psychiatry uses such cross-cultural comparison to identify the role of social processes in the origins, course and outcomes of mental health and illness. One of the most striking recent findings in this area is evidence for social influences on the incidence of schizophrenia.

As some of the most severe forms of psychopathology, psychotic disorders tend to be viewed as the exemplars of biological psychiatric disorders. Indeed, after a period of interest in the importance of social factors in the causes, course, and outcome of schizophrenia in the 1950s and 1960s, there has been a decline of research on, and interest in, social factors in schizophrenia in North America (Jarvis, 2007). This de-emphasis of social determinants has gone hand-in-hand with a search for genetic causes—a goal which, to date, has proved elusive. At the same time, however, there is substantial evidence for profound social influences on the causes and course of schizophrenia.

Perhaps the most important source of relevant evidence for social effects on the etiology of psychosis comes from investigation of the effects of migration on the incidence of schizophrenia (Cantor-Graae, 2007; Coid et al., 2008). Over the last 30 years, a number of studies of African and Caribbean migrants to Britain have found higher rates of schizophrenia in these populations, ranging from rates that are twice to 14 times higher than the white population (Fearon & Morgan, 2006). A meta-analysis conducted by Cantor-Graae and Selten (2005; see also Bourque, van der Ven, & Malla, 2011) produced

a mean weighted relative risk for developing schizophrenia of 2.7 (95% CI 2.3–3.2). Remarkably, the relative risk in the children of these immigrants (either born in the country of migration or brought up there from a young age) was higher still (mean weighted relative risk of 4.5, 95% CI 1.5–13.1). This demonstrates that the effect on mental health cannot be attributed exclusively to the stress of the process of migration itself. Whatever factors are operative seem to affect the second generation still more strongly.

While striking, there are many methodological challenges involved in conducting these studies, so the findings must be interpreted with caution (McKenzie, Fearon, & Hutchison, 2008). These studies do not usually distinguish between types of immigrant (for example, economic migrants versus refugees) whose psychological profile and reaction to the process of migration might be expected to vary considerably. Nor do these studies distinguish well among different ethnic groups. While most studies make use of first-admission or first-contact cases, it is known that members of different social groups typically come to the attention of the mental health system in different ways. It is thus not possible to be sure that the numbers of cases in different populations are being measured equally accurately. Moreover, if members of some groups are more likely to seek care than others, then the numbers of clinical cases may not be representative of the numbers in the general population. There are also concerns about comparing the incidence of schizophrenia in migrant groups with the incidence in the country of origin given that diagnostic methods are not uniform cross-culturally. Finally, there are questions about the accuracy of diagnoses across cultures and the possibility of ethno-racial bias in assessment.

Despite these difficulties, the size of the increase in the incidence of schizophrenia and the consistency of findings strongly suggests that the phenomenon is real and no mere artifact (McKenzie et al., 2008). In addition, the AESOP study carried out by Fearon and colleagues (2006) controlled for some of the relevant variables, and their findings confirmed those of the earlier studies. Incidence rate ratios (IRRs) were calculated for each ethnic group in comparison to the White population and were found to be very high for schizophrenia and manic psychosis in African-Caribbeans (9.1 and 8.0, respectively) as well as in Black Africans (5.8 and 6.2, respectively) in both men and women. Thus, whatever the stresses of migration, they act somewhat selectively, affecting some mental processes more than others, increasing vulnerability to—or undermining protective factors against—schizophrenia and mania in particular.

There is no consensus about what actually does the psychological damage either to immigrants themselves or to their children, but there is no evidence that the differential incidence of schizophrenia is genetic in nature; the incidence of schizophrenia in the countries from which most Caribbean migrants come is no higher than in the White population of the UK (Hickling & Rodgers-Johnson, 1995; Mahy, Mallett, Leff, & Bhugra, 1999). Whatever is increasing the vulnerability, or decreasing the efficacy of protective factors, seems to be social in nature. At the very least, genetic vulnerabilities are being manifest by changes in social conditions. Leading candidates include poverty and, more generally, socioeconomic disadvantage, racism, and living in an urban environment (McKenzie et al., 2008).

The effect of the urban environment has been studied in some detail and may constitute one of the strongest risk factors for the development of psychosis (Krabbendam & van Os, 2005). Studies over many decades have repeatedly shown that the rates of

schizophrenia are influenced by exposure to urban environments and that there is also a dose effect: the larger the city, the higher the incidence of psychosis. Indeed there is evidence dating from the nineteenth century showing the same effect (Torrey, Bowler, & Clark, 1997). Furthermore, the effect is greater according to the number of years one spends in an urban region between birth and 20 years of age (Pedersen & Mortensen, 2001). In addition, the effect of urban life increases psychosis-like symptoms in non-clinical populations (van Os, Hanssen, Bijl, & Vollebergh, 2001). Most importantly, the urban effect seems to be specific to psychosis. Bipolar disorder, for example, is no more common in cities than in rural areas (Mortensen et al., 1999).

In order to assert the causal role of the urban environment, however, one must be able to exclude at least two alternative hypotheses: (1) that psychotic individuals, or those in the prodromal phase of psychosis, are more likely than non-psychotic or prodromal individuals to move to the city (the "social drift" hypothesis); and (2) that those who are mentally ill are less likely than those who are not psychotic to leave the city for more attractive (rural) communities—the "social residue hypothesis." Dauncey and colleagues (Dauncey, Giggs, Baker, & Harrison, 1993) investigated the place of residence of psychotic patients during the five-year period before admission and found no evidence for the social drift hypothesis. Mortensen and colleagues (Mortensen et al., 1999) argue that for this drift to have occurred in the previous generation would require an extremely high degree of movement from rural to urban areas.

A number of other potentially confounding factors have also been examined, including obstetric complications, adverse life events, and season of birth, and do not account for the effect of urban environment (Boydell & McKenzie, 2008). While socioeconomic disadvantage might be expected to account for at least some of the urbanicity effect, many of the relevant studies have been carried out in countries in which the standard of living is higher in urban than in rural regions. Drug use, in contrast, may constitute part of the explanation for the urbanicity effect in psychosis, though the effect remains even when adjusted for the use of cannabis.

It is worth noting that there seems to be a complex interaction between the effect of urban life and genetic predisposition to psychosis. Those with a genetic vulnerability to schizophrenia seem to be disproportionately affected by urban life, so that the urban environment constitutes a greater stress on vulnerable individuals than on those who are not (van Os, Pedersen, & Mortensen, 2004). A parallel synergy occurs at the social level. Van Os and colleagues (van Os, Driessen, Gunther, & Delespaul, 2000) found that people without partners are disproportionately at risk for psychosis if they are city-dwellers. We will return to this issue below.

Although it is at present unclear just what causes the urbanicity effect, it seems to be a function of human relations, an idea supported by the fact that within cities the effect is distributed differentially across neighborhoods (Kirkbride et al., 2006). The incidence of schizophrenia is higher in economically deprived areas with a high proportion of single-person households and high levels of population mobility (Boydell & McKenzie, 2008). This suggests that the effect is determined by the structure of particular communities and is thus fundamentally social. In the case of immigrants, there is evidence that the ethnic density of the neighborhood affects risk for psychosis (Veling et al., 2008). Those living in areas where there is a smaller proportion of their own ethnic group are at greater risk.

Taken together, these studies suggest that social factors are crucial determinants of the risk of schizophrenia. The nature of these factors and their differential distribution and impact on individuals from different backgrounds result from processes that can only be adequately described at the level of the social world, in terms of the impact of the histories of colonialism, migration, racism, and discrimination on social and economic inequalities. Although neuroscience can help us understand the proximate mediators of these social effects, it can never predict their spatial or geographic distribution and may misdirect attention away from crucial, modifiable social structural factors that demand remediation.

Socializing Biological Psychiatry

The evidence for social determinants of health—and of mental health in particular—is compelling. All of this might be granted, yet the biological psychiatrist could claim it lies outside the purview of psychiatry, which studies only the proximate neural mediation of the effects of the social environment. However, the whole thrust of our argument is that there should not be an either/or in considering brain–society interactions. Instead, psychiatry needs theories of social and cultural biology that recognize the fundamental role of social processes not only as determinants of health and illness but as the mediators and mechanisms of psychopathology as well as of healing and recovery.

We raised, in passing, the possibility that genetic factors could contribute to the increase in the incidence of schizophrenia seen in migrants, that arises as a result of uncovering of genetic vulnerability when protective factors—for example, the organization of family or social life in the home country—are no longer present in the destination country. We also noted the possibility of synergies between genetic susceptibility and the urban environment. This raises the possibility that social factors interact in some way with genetic mechanisms.

There are at least three ways in which this could be happening. The first is that genes could predispose to behavior in ways that feed back on mental life. Kendler and Prescott (2006, pp. 264–265) provide an apt, if hypothetical, example of the basic idea:

> A cancer geneticist has collected a sample of 400 patients with lung cancer and 400 control participants. She scans a chromosome looking for gene variants that differentiate the two groups and finds a gene that is much more common among the lung cancer patients. With great excitement, she writes up her results and submits them to a major scientific journal, claiming to have found a new oncogene (i.e., a gene that can cause cancer). However, unbeknownst to her, the gene has no effect on the risk for cancer at a physiological level. Instead, it exerts an indirect effect, through behavior, on the risk for chronic cigarette smoking. For example, genetically controlled variation in nicotine receptors, which stimulate the pleasure centers in the brain, might affect the chances that individuals will seek repeated exposure to carcinogenic compounds. Has this researcher really found a new oncogene? Yes and no. Traditional oncogenes act via inside-the-skin pathways (e.g., by influencing cell division), whereas this oncogene acts via an outside-the-skin pathway. This oncogene will have a few unusual properties not possessed by

traditional oncogenes. In a culture in which tobacco is not smoked, it will have no effect on cancer risk. Any social process that reduces the frequency of heavy tobacco smoking (such as reduced social acceptability or increased taxation) will reduce the impact of the oncogene on risk for lung cancer.

"Outside-the-skin" gene expression could of course also occur in psychiatric disorder. Consider another researcher who finds a gene that correlates with schizophrenia. She infers that the gene is likely to code for a protein that is implicated in dopamine function, which in turn is associated with the cardinal symptoms of schizophrenia. It turns out, however, that the gene is actually associated with temperament; people who have it tend to be unassertive and therefore are more likely to be bullied as children—and bullying may play a causal role in the later development of psychosis (Bebbington et al., 2004). Has this researcher found a gene for schizophrenia?[6] Not really. Like the putative oncogene, the effect of this gene has to be understood in the context of the environment in which it is expressed. The social environment may thus be part of a loop that affects mental life, and ignoring the potential role of the environment may lead to a misunderstanding of biological function of the gene.

A second possibility is that mentioned in relation to the effects of the urban environment on those disposed to schizophrenia. If a genetic disposition renders one individual more vulnerable to a social stressor than others, then this is evidence that there is a synergy between biological and social features that must be understood together. For example, individuals with a particular form of the serotonin transporter (5-HTT) gene are more susceptible to stress and, therefore, to depression and suicide than those without it (Caspi et al., 2003). This same sort of genetic polymorphism might confer adaptive advantages in other environmental and social contexts (Suomi, 2006).

A third way in which the social world may be interacting with our biology is via epigenetic processes—that is, processes in which the expression of genes, rather than the genes themselves, is altered. Research on epigenetics has begun to reveal how interactions of the genome with the environment over development lead to structural changes in the methylation patterns of DNA that regulate cellular function. These changes may be lasting so that experience remodels the functional genome. For example, there is compelling evidence in rodents and primates that early parenting experiences alter the regulation of stress response systems for the life of the organism via the hypothalamic-pituitary-adrenal stress response (Meaney, 2001; Meaney & Szyf, 2005; Zhang & Meaney, 2010). This process occurs in humans as well. In a recent paper, McGowan and colleagues (McGowan et al., 2009) reported a post-mortem study of hippocampal tissue that showed differences in glucocorticoid receptors' gene expression in suicide victims with a known history of abuse compared to suicide victims without such a history. Gene expression was reduced in individuals who suffered from abuse, but no difference was found between suicide victims without

[6] Kendler (2005) discusses the assumptions in the phrase "X is a gene for Y," pointing out that since psychiatric disorders have multiple causes and the causal pathway from any genetic variation to any specific type of behavioral disturbance is usually long, complicated, and context dependent, it will rarely if ever be appropriate to say that "X is a gene for psychiatric disorder Y."

a history of abuse and controls. It is reasonable to infer, therefore, that the changes in gene expression are correlated with the abuse itself and not with some aspect of the suicide behavior or its prodrome. This seems to be compelling evidence that the social world—in this case, home life—has a direct influence on gene expression and therefore, perhaps, on behavior in humans. This important finding shows that the nervous system is reshaped by experience not only at the synaptic level but in its underlying genetic regulation as well.

Recent work suggests that schizophrenia might be associated with specific epigenetic modulation of multiple systems (Mill et al., 2008). This points to a more refined way of thinking about the interactions between the brain and the social environment (Mill & Petronis, 2009; Petronis, 2004). The types of social adversity faced by immigrants, described above, may exert influences over the course of development through epigenetic processes that render individuals more vulnerable to schizophrenia. The epigenetic effect of social stressors will interact with ongoing social processes that constrain individuals' adaptation and expose them to prolonged and persistent stresses such as those associated with poverty, inequality, marginalization, and discrimination (Wilkinson & Pickett, 2009).

We thus need models and corresponding languages of description that allow us to recognize, study, and intervene in patterns and processes of adversity and resilience that are located outside the brain—even if, through learning and development, the social world comes to have shadows, refractions, or reflections in the functional genome and the circuitry of brain. The social world has its own organization—it is not comprised of isolated risk or protective factors but of coordinated systems with persistent effects over time that reflect dynamics that are irreducibly social.

Conclusion: Beyond Reductionism

We have tried to show that (1) as a methodological strategy, biological reductionism is useful but not sufficient to understand the origins of human behavior and experience in health and sickness; (2) as an ontological position, biological reductionism is undermined by the higher level of organization at which mental life must be understood, which includes interactions between the brain and the social world; and (3) partly in consequence of these first two conclusions, epistemological reductionism will never be adequate as a comprehensive understanding of human behavior and experience. In fact, promoting such reductionism in psychiatry does real violence to our conceptual models and the production of knowledge and, ultimately, to clinical practice that aims to be person-centered and integrative.

Given that the non-reductionist view we have described has a long lineage and is grounded in solid observation and argument about the nature of hierarchical systems—and more specifically about the nervous system—the persistent enthusiasm for reductionist epistemologies requires some explanation. This is a task for critical neuroscience. We think the answers for this bias will be found not only in the methodological advantages of reductionism for scientists seeking to design experiments, or their desire to argue for the utility of simple models to address important mental health problems. We believe that they will also be discovered in the ways in which

biological explanations draw attention away from highly contested social and political issues—issues that would demand much political consensus and will to address—and focus instead on a level of explanation distant from everyday experience, that can be framed as a politically neutral arena for scientific explanation and technical mastery. This neutralization of the politically loaded issue of the social origins of mental health disparities goes hand-in-hand with the economic exploitation of biological theory by pharmaceutical companies.

When Insel and Quirion express the view that psychiatry is "clinically applied neuroscience," they are expressing a form of epistemological reductionism—a form of reductionism according to which mental illness will ultimately be understood and treated by a successful theory of the brain. If, however, as we have argued, one cannot understand mental illness without reference to social causes of mental illness, then no theory that is exclusively about the brain can be complete. At best, a neuroscientific theory can articulate the end result of the complex interactions of the organism with its environment. Even if it turns out that a disorder of dopamine, for example, is a necessary and sufficient condition for the symptoms of schizophrenia, it would be a profound error to ignore the social world that contributes to the causes, course and outcome of that disorder as scientifically insignificant. A successful theory of the brain will undoubtedly explain a great deal about mental life and mental illness, but on its own it will provide no more than a keyhole view of the mind. It seems likely, therefore, that unless economic forces conspire to shrink it to a narrow technical domain in the future psychiatry will become not just behavioral neurology or applied neuroscience but also clinically applied social science.

References

Bateson, G. (1972). *Steps to an ecology of mind*. New York: Ballantine Books.

Bebbington, P. E., Bhugra, D., Brugha, T., Singleton, N., Farrell, M., Jenkins, R., Lewis, G., & Meltzer, H. (2004). Psychosis, victimization and childhood disadvantage: Evidence from the second British National Survey of psychiatric morbidity. *British Journal of Psychiatry, 185*, 220–226.

Bedau, M. A., & Humphreys, P. (Eds.). (2008). *Emergence: Contemporary readings in philosophy and science*. Cambridge, MA: MIT Press.

Bloor, D. (1983). *Wittgenstein: A social theory of knowledge*. London: Macmillan.

Bourque, F., E. van der Ven., & Malla, A. (2011). A meta-analysis of the risk for psychotic disorders among first- and second-generation immigrants. *Psychological Medicine*, *41*, 897–910.

Boydell, J., & Mckenzie, K. (2008). Society, place and space. In Morgan, C., Mckenzie, K., & Fearon, P. (Eds.), *Society and psychosis*. New York: Cambridge University Press.

Cantor-Graae, E. (2007). The contribution of social factors to the development of schizophrenia: A review of recent findings. *Canadian Journal of Psychiatry, 52*, 277–286.

Cantor-Graae, E., & Selten, J.-P. (2005). Schizophrenia and migration: A meta-analysis and review. *American Journal of Psychiatry, 162*, 12–24.

Carter, C. S. 1998. Neuroendocrine perspectives on social attachment and love. *Psychoneuroendocrinology, 23*, 779–818.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., Mcclay, J., Mill, J., Martin, J., Braithwaite, A., & Poulton, R. (2003). Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science, 301*, 386–389.

Chalmers, D. J. (1996). *The conscious mind*. New York: Oxford University Press.

Clark, A. (2008). *Supersizing the mind: Embodiment, action, and cognitive extension*. New York: Oxford University Press.

Clark, A., & Chalmers, D. (1998). The extended mind. *Analysis, 58*, 7–19.

Cohen, D., & Eisdorfer, C. (2001). *The loss of self: A family resource for the care of Alzheimer's disease and related disorders*. New York: Norton.

Coid, J. W., Kirkbride, J. B., Barker, D., Cowden, F., Stamps, R., Yang, M., & Jones, P. B. (2008). Raised incidence rates of all psychoses among migrant groups: Findings from the East London first episode psychosis study. *Archives of General Psychiatry, 65*, 1250–1258.

Dauncey, K., Giggs, J., Baker, K., & Harrison, G. (1993). Schizophrenia in Nottingham: Lifelong residential mobility of a cohort. *British Journal of Psychiatry, 163*, 613–619.

Eisenberg, L. (1986). Mindlessness and brainlessness in psychiatry. *British Journal of Psychiatry, 148*, 497–508.

Fearon, P., Kirkbride, J. B., Morgan, C., Dazzan, P., Morgan, K., Lloyd, T., & Murray, R. M. (2006). Incidence of schizophrenia and other psychoses in ethnic minority groups: Results from the MRC AESOP Study. *Psychological Medicine, 36*, 1541–1550.

Fearon, P., & Morgan, C. (2006). Environmental factors in schizophrenia: The role of migrant studies. *Schizophrenia Bulletin, 32*, 405–408.

Fisher, H. E., Aron, A., & Brown, L. L. (2006). Romantic love: A mammalian brain system for mate choice. *Philosophical Transactions of the Royal Society of London B Biological Sciences, 361*, 2173–2186.

Fodor, J. (1997). Special sciences: Still autonomous after all these years. *Noûs, 31*, (Suppl 11), 149–163.

Gottman, J. M., Murray, J. D., Swanson, C. C., Tyson, R., & Swanson, K. R. (2002). *The mathematics of marriage: Dynamic nonlinear models*. Cambridge, MA: MIT Press.

Grewen, K. M., Girdler, S. S., Amico, J., & Light, K. C. (2005). Effects of partner support on resting oxytocin, cortisol, norepinephrine, and blood pressure before and after warm partner contact. *Psychosomatic Medicine, 67*, 531–538.

Griffiths, P. E., & Scarantino, A. (2009). Emotions in the wild: The situated perspective on emotion. In P. Robbins & M. Aydede (Eds.), *Cambridge handbook of situated cognition*. Cambridge: Cambridge University Press.

Gross, D. M. (2006). *The secret history of emotion: From Aristotle's rhetoric to modern brain science*. Chicago: University of Chicago Press.

Hacking, I. (2002). *Historical ontology*. Cambridge, MA: Harvard University Press.

Hacking, I. (1999). *The social construction of what?* Cambridge, MA: Harvard University Press.

Healy, D. (2002). *The creation of psychopharmacology*. Cambridge, MA: Harvard University Press.

Herskovits, E. (1995). Struggling over subjectivity: Debates about the "self" and Alzheimer's disease. *Medical Anthropology Quarterly, 9*, 146–164.

Hickling, F. W., & Rodgers-Johnson, P. (1995). The incidence of first contact schizophrenia in Jamaica. *British Journal of Psychiatry, 167*, 193–196.

Insel, T. R., & Fernald, R. D. (2004). How the brain processes social information: Searching for the social brain. *Annual Review of Neuroscience, 27*, 697–722.

Insel, T. R., & Quirion, R. (2005). Psychiatry as a clinical neuroscience discipline. *Journal of the American Medical Association, 294*, 2221–2224.

Jackson, F. (1982). Epiphenomenal qualia. *Philosophical Quarterly, 32*, 127–136.

Jarvis, G. E. (2007). The social causes of psychosis in North American psychiatry: A review of a disappearing literature. *Canadian Journal of Psychiatry, 52*, 287–294.

Kauffman, S. A. (1993). *The origins of order: Self-organization and selection in evolution*. New York: Oxford University Press.

Kendler, K. S. (2005). "A gene for…": The nature of gene action in psychiatric disorders. *American Journal of Psychiatry, 162*, 1243–1252.

Kendler, K. S., & Prescott, C. A. (2006). *Genes, environment, and psychopathology: Understanding the causes of psychiatric and substance use disorders.* New York: Guilford.

Kirkbride, J. B., Fearon, P., Morgan, C., Dazzan, P., Morgan, K., Tarrant, J., & Jones, P. B. (2006). Heterogeneity in incidence rates of schizophrenia and other psychotic syndromes: findings from the 3-center AeSOP study. *Archives of General Psychiatry, 63*, 250–258.

Kirmayer, L. J. (2008). Culture and the metaphoric mediation of pain. *Transcultural Psychiatry, 45*, 318–338.

Kirmayer, L. J. (2000). Broken narratives: Clinical encounters and the poetics of illness experience. In C. Mattingly & L. Garro (Eds.), *Narrative and the cultural construction of illness and healing.* Berkeley, CA: University of California Press.

Krabbendam, L., & van Os, J. (2005). Schizophrenia and urbanicity: A major environmental influence conditional on genetic risk. *Schizophrenia Bulletin, 31*, 795–799.

Light, K. C., Grewen, K. M., & Amico, J. A. (2005). More frequent partner hugs and higher oxytocin levels are linked to lower blood pressure and heart rate in premenopausal women. *Biological Psychology, 69*, 5–21.

Mahy, G. E., Mallett, R., Leff, J., & Bhugra, D. (1999). First-contact incidence rate of schizophrenia on Barbados. *British Journal of Psychiatry, 175*, 28–33.

Mainzer, K. (2004). *Thinking in complexity: The computational dynamics of matter, mind, and mankind.* New York: Springer.

Malabou, C. (2008). *What should we do with our brain?* New York: Fordham University Press.

Maturana, H. R., & Varela, F. J. (1987). *The tree of knowledge: The biological roots of human understanding.* Boston: Random House.

Maturana, H. R., & Varela, F. J. (1980). *Autopoiesis and cognition: The realization of the living.* Dordrecht, Holland: Reidel Publishing Company.

McGowan, P. O., Sasaski, A., d'Alessio, A. C., Dymov, S., Labonte, B., Szyf, M., & Meaney, M. J. (2009). Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature Neuroscience, 12*, 342–348.

McKenzie, K., Fearon, P., & Hutchison, G. (2008). Migration, ethnicity and psychosis. In C. Morgan, K. McKenzie & P. Fearon (Eds.), *Society and psychosis.* New York: Cambridge University Press.

Meaney, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annual Review of Neurosciences, 24*, 1161–1192.

Meaney, M. J., & Szyf, M. (2005). Maternal care as a model for experience-dependent chromatin plasticity? *Trends in Neuroscience, 28*, 456–463.

Meehl, P. E., & Sellars, W. (1966). The concept of emergence. In H. Feigl & M. Scriven (Eds.), *Minnesota studies in the philosophy of science.* Minneapolis, MN: University of Minnesota Press.

Mill, J., & Petronis, A. (2009). The relevance of epigenetics to major psychosis. In A. C. Ferguson-Smith, J. M. Greally & R. A. Martienssen (Eds.), *Epigenomics.* New York: Springer SBM.

Mill, J., Tang, T., Kaminsky, Z., Khare, T., Yazdanpanah, S., Bouchard, L., & Petronis, A. (2008). Epigenomic profiling reveals DNA-methylation changes associated with major psychosis. *American Journal of Human Genetics, 82*, 696–711.

Morowitz, H. J. (2002). *The emergence of everything: How the world became complex.* New York: Oxford University Press.

Mortensen, P. B., Pedersen, C. B., Westergaard, T., Wohlfahrt, J., Ewald, H., Mors, O., & Melbye, M. (1999). Effects of family history and place and season of birth on the risk of schizophrenia. *New England Journal of Medicine, 340*, 603–608.

Nagel, T. (1974). What is it like to be a bat? *Philosophical Review, 83*, 435–450.
Nicolis, G., & Prigogine, I. (1989). *Exploring complexity: An introduction*. New York: W. H. Freeman.
Pattee, H. H. (1977). Dynamic and linguistic modes of complex systems. *International Journal of General Systems, 3*, 259–266.
Pattee, H. H. (1973). *Hierarchy theory: The challenge of complex systems*. New York: George Braziller.
Pedersen, C. B., & Mortensen, P. B. (2001). Evidence of a dose-response relationship between urbanicity during upbringing and schizophrenia risk. *Archives of General Psychiatry, 58*, 1039–1046.
Petronis, A. (2004). The origin of schizophrenia: Genetic thesis, epigenetic antithesis, and resolving synthesis. *Biological Psychiatry, 55*(10), 965–970.
Porges, S. W. (1998). Love: An emergent property of the mammalian autonomic nervous system. *Psychoneuroendocrinology, 23*, 837–861.
Pribram, K. H. (1990). From metaphors to models: The use of analogies in neuropsychology. In D.E. Leary (Ed.), *Metaphors in the history of psychology*. New York: Cambridge University Press.
Prosser, C. L. (1970). Levels of biological organization and their physiological significance. In J. A. Moore (Ed.), *Ideas in evolution and behavior*. New York: Natural History Press.
Reddy, W. M. (2001). *The navigation of feeling: A framework for the history of emotions*. Cambridge: Cambridge University Press.
Rosen, R. (1991). *Life itself: A comprehensive inquiry into the nature, origin, and fabrication of life*. New York: Columbia University Press.
Rosen, R. (1968). Hierarchical organization in automata theoretic models of the central nervous system. In K. N. Leibovic (Ed.), *Information processing in the nervous system* (pp. 21–35). New York: Springer.
Scott, A. (2002). *Neuroscience: A mathematical primer*. New York: Springer.
Searle, J. R. (1995). *The construction of social reality*. New York: Free Press.
Sperber, D. (1996). *Explaining culture: A naturalistic approach*. Oxford: Blackwell Publishers.
Suomi, S. J. (2006). Risk, resilience, and gene x environment interactions in rhesus monkeys. *Annals of the New York Academy of Science, 1094*, 52–62.
Torrey, E. F., Bowler, A. E., & Clark, K. (1997). Urban birth and residence as risk factors for psychoses: An analysis of 1880 data. *Schizophrenia Research, 25*, 169–176.
van Os, J., Driessen, G., Gunther, N., & Delespaul, P. (2000). Neighbourhood variation in incidence of schizophrenia. Evidence for person-environment interaction. *The British Journal of Psychiatry, 176*, 243–248.
van Os, J., Hanssen, M., Bijl, R. V., & Vollebergh, W. (2001). Prevalence of psychotic disorder and community level of psychotic symptoms: An urban-rural comparison. *Archives of General Psychiatry, 58*, 663–668.
van Os, J., Pedersen, C. B., & Mortensen, P. B. (2004). Confirmation of synergy between urbanicity and familial liability in the causation of psychosis. *American Journal of Psychiatry, 161*, 2312–2314.
Veling, W., Susser, E., van Os, J., Mackenbach, J. P., Selten, J. P., & Hoek, H. W. (2008). Ethnic density of neighborhoods and incidence of psychotic disorders among immigrants. *American Journal of Psychiatry, 165*, 66–73.
von Neumann, J. (1958). *The computer and the brain*. New Haven: Yale University Press.
von Neumann, J. (1951). The general and logical theory of automata. In L. Jeffress (Ed.), *Cerebral mechanisms in behavior*. New York: John Wiley & Sons, Inc.
Wilkinson, R. G., & Pickett, R. (2009). *The spirit level: Why more equal societies almost always do better*. London: Penguin.

Wilson, E. A. (2004). *Psychosomatic: Feminism and the neurological body*. Durham: Duke University Press.

Wilson, M. (1993). DSM-III and the transformation of American psychiatry: A history. *American Journal of Psychiatry, 150*, 399–410.

Wolfram, S. (2002). *A new kind of science*. Champaign, IL: Wolfram Media.

Zhang, T. Y., & Meaney, M. J. (2010). Epigenetics and the environmental regulation of the genome and its function. *Annual Review of Psychology, 61*, 439–466, C1–3.