Psychology and Neuroscience: Making Peace
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Abstract
There has been no historically stable consensus about the relationship between psychological and biological concepts and data. A naively reductionist view of this relationship is prevalent in psychology, medicine, and basic and clinical neuroscience. This view undermines the ability of psychology and related sciences to achieve their individual and combined potential. A nondualistic, nonreductionist, noninteractive perspective is recommended, with psychological and biological concepts both having central, distinct roles.

Keywords
psychology; biology; neuroscience; psychopathology

With the Decade of the Brain just ended, it is useful to consider the impact that it has had on psychological research and what should come next. Impressive progress occurred on many fronts, including methodologies used to understand the brain events associated with psychological functions. However, much controversy remains about where biological phenomena fit into psychological science and vice versa. This controversy is especially pronounced in research on psychopathology, a field in which ambitious claims on behalf of narrowly conceived psychological or biological factors often arise, but this fundamental issue applies to the full range of psychological research. Unfortunately, the Decade of the Brain has fostered a naively reductionist view that sets biology and psychology at odds and often casts psychological events as unimportant epiphenomena. We and other researchers have been developing a proposal that rejects this view and provides a different perspective on the relationship between biology and psychology.

A FAILURE OF REDUCTIONISM

A term defined in one domain is characterized as reduced to terms in another domain (called the reduction science) when all meaning in the former is captured in the latter. The reduced term thus becomes unnecessary. If, for example, the meaning of the (traditionally psychological) term “fear” is entirely representable in language about a brain region called the amygdala, one does not need the (psychological) term “fear,” or one can redefine “fear” to refer merely to a particular biological phenomenon.

Impressive progress in the characterization of neural circuits typically active in (psychologically defined) fear does not justify dismissing the concept or altering the meaning of the term. The phenomena that “fear” typically refers to include a functional state (a way of being or being prepared to act), a cognitive processing bias, and a variety of judgments and associations all of which are conceived psychologically (Miller & Kozak, 1993). Because “fear” means more than a given type of neural activity, the concept of fear is not reducible to neural activity. Researchers are learning a great deal about the biology of fear—and the psychology of fear—from studies of the amygdala (e.g., Lang, Davis, & Öhman, in press), but this does not mean that fear is activity in the amygdala. That is simply not the meaning of the term. “Fear” is not reducible to biology.

This logical fact is widely misunderstood, as evidenced in phrases such as “underlying brain dysfunction” or “neurochemical basis of psychopathology.” Most remarkably, major portions of the federal research establishment have recently adopted a distinctly nonmental notion of mental health,
referring to “the biobehavioral factors which may underly [sic] mood states” (National Institute of Mental Health, 1999). Similarly, a plan to reorganize grant review committees reflects “the context of the biological question that is being investigated” (National Institutes of Health, 1999, p. 2). Mental health researchers motivated by psychological or sociological questions apparently should take their applications elsewhere.

More subtly problematic than such naive reductionism are terms, such as “biobehavioral marker” or “neurocognitive measure,” that appear to cross the boundary between psychological and biological domains. It is not at all apparent what meaning the “bio” or “neuro” prefix adds in these terms, as typically the data referred to are behavioral. Under the political pressures of the Decade of the Brain, psychologists were tempted to repack-age their phenomena to sound biological, but the relationship of psychology and biology cannot be addressed by confusing them.

Such phrases often appear in contexts that assume that biological phenomena are somehow more fundamental than psychological phenomena. Statements that psychological events are nothing more than brain events are clearly logical errors (see the extensive analysis by Marr, 1982). More cautious statements, such as that psychological events “reflect” or “arise from” brain events, are at best incomplete in what they convey about the relationship between psychology and biology. It is not a property of biological data that they “underlie” psychological data. A given theory may explicitly propose such a relationship, but it must be treated as a proposal, not as a fact about the data. Biological data provide valuable information that may not be obtainable with self-report or overt behavioral measures, but biological information is not inherently more fundamental, more accurate, more representative, or even more objective.

The converse problem also arises—psychology allegedly “underlying” or being more fundamental than biology. There is a long tradition of ignoring biological phenomena in clinical psychology. As Zuckerman (1999) noted, “One thing that both behavioral and post-Freudian psychoanalytic theories had in common was the conviction that learning and life experiences alone could account for all disorders” (p. 413). In those traditions, it is psychology that “underlies” biology, not the converse. Biology is seen as merely the implementation of psychology, and psychology is where the intellectually interesting action is. Cognitive theory can thus evolve without the discipline of biological plausibility. As suggested at the midpoint of the Decade of the Brain (Miller, 1995), such a view would justify a Decade of Cognition.

Such a one-sided emphasis would once again be misguided. Anderson and Scott (1999) expressed concern that “the majority of research in the health sciences occurs within a single level of analysis, closely tied to specific disciplines” (p. 5), with most psychologists studying phenomena only in terms of behavior. We advocate not that every study employ both psychological and biological methods, but that researchers not ignore or dismiss relevant litera-tures, particularly in the conceptual-ization of their research.

Psychological and biological approaches offer distinct types of data of potentially equal relevance for understanding psychological phenomena. For example, we use magnetoencephalography (MEG) recordings of the magnetic fields generated by neural activity to identify multiple areas of brain tissue that are generating what is typically measured electrically at the scalp (via electroencephalo-graphy, or EEG) as the response of the brain associated with cognitive tasks (Cañive, Edgar, Miller, & Weisend, 1999). One of the most firmly established biological findings in schizophrenia is a smaller than normal brain response called the P300 component (Ford, 1999), and there is considerable consen-sus on the functional significance of P300 in the psychological domain. There is, however, no consensus on what neural generators produce the electrical activity or on what distinct functions those gen-erators serve. Neural sources are often difficult to identify with confi-dence from EEG alone, whereas for biophysical reasons MEG (which shows brain function) coupled with structural magnetic resonance data (which show brain anatomy) promises localization as good as any other available noninvasive method. If researchers under-stand the distinct functional sig-nificance of various neural generators of P300, and if only some generators are compromised in schizophrenia, this will be informa-tive about the nature of cognitive deficits in schizophrenia. Con-versely, what researchers know about cognitive deficits will be informative about the function of the different generators.

MEG and EEG do not “underlie” and are not the “basis” of (the psychological phenomena that define) the functions or mental operations invoked in tasks associated with the P300 response. Neu-ral generators implement func-tions, but functions do not have locations (Fodor, 1968). For ex-ample, a working memory deficit in schizophrenia could not be lo-
cated in a specific brain region. The psychological and the neuromagnetic are not simply different “levels” of analysis, except in a very loose (and unhelpful) metaphorical sense. Neither underlies the other, neither is more fundamental, and neither explains away the other. There are simply two domains of data, and each can help to explicate the other because of the relationships theories propose.

Psychophysiological research provides many other examples in which the notion of “underlying” is unhelpful. Rather than attributing mood changes to activity in specific brain regions, why not attribute changes in brain activity to changes in mood? In light of EEG (Deldin, Keller, Gergen, & Miller, 2000) or behavioral (Keller et al., 2000) data on regional brain activity in depression, are people depressed because of low activity in left frontal areas of the brain, or do they have low activity in these areas because they are depressed? Under the present view, such a question, trying to establish causal relations between psychology and biology, is misguided. These are not empirical issues but logical and theoretical issues. They turn on the kind of relationship that psychological and biological concepts are proposed to have.

**CLINICAL IMPLICATIONS**

In psychopathology, one of the most unfortunate consequences of the naive competition between psychology and biology is the assumption that dysfunctions conceptualized biologically require biological interventions and that those conceptualized psychologically require psychological interventions. The best way to alter one system may be a direct intervention in another system. Even, for example, if the chemistry of catecholamines (chemicals used for communication to nerve, muscle, and other cells) were the best place to intervene in schizophrenia, it does not follow that a direct biological intervention in that system would be optimal. A variety of experiences that people construe as psychosocial prompt their adrenal glands to flood them with catecholamines. There are psychological interventions associated with this chemistry that can work more effectively or with fewer side effects than medications aimed directly at the chemistry.

Unfortunately, the assumption that disorders construed biologically warrant exclusively biological interventions influences not only theories of psychopathology but also available treatments. For example, major depression is increasingly viewed as a “chemical imbalance.” If such (psychological) disorders are assumed to “be” biological, then medical insurers are more likely to fund only biological treatments. Yet Thase et al. (1997) found that medication and psychotherapy were equally effective in treating moderately depressed patients and that the combination of these treatments was more effective than either alone in treating more severely depressed patients. Hollon (1995) discussed how negative life events may alter biological factors that increase risk for depression. Meaney (1998) explained how the psychological environment can affect gene activity. The indefensible conceptualization of depression solely as a biological disorder prompts inappropriately narrow (biological) interventions. Thus, treatment as well as theory is hampered by naive reductionism.

**WHAT TO DO?**

“Underlying” (implying one is more fundamental than the other) is not a satisfactory way to characterize the relationship between biological and psychological concepts. We recommend characterizing the biological as “implementing” the psychology—that is, we see cognition and emotion as implemented in neural systems. Fodor (1968) distinguished between contingent and necessary identity in the relationship between psychological and biological phenomena. A person in any given psychological state is momentarily in some biological state as well: There is a contingent identity between the psychological and the biological at that moment. The psychological phenomenon implemented in a given neural circuit is not the same as, is not accounted for by, and is not reducible to that circuit. There is an indefinite set of potential neural implementations of a given psychological phenomenon. Conversely, a given neural circuit might implement different psychological functions at different times or in different individuals. Thus, there is no necessary identity between psychological states and brain states. Distinct psychological and biological theories are needed to explain their respective domains, and additional theoretical work is needed to relate them.

Nor is it viable (though it is common) to say that psychological and biological phenomena “interact.” Such a claim begs the question of how they interact and even what it means to interact. The concept of the experience of “red” does not “interact” with the concept of photon-driven chemical changes in the retina and their neural sequelae. One may propose that those neural sequelae implement the perceptual experience of “red,” but “red” means not the neural sequelae, but something psychological—a perception.

Biology and psychology often are set up as competitors for public mind-share, research funding, and
scientific legitimacy. We are not arguing for a psychological explanation of cognition and emotion instead of a biological explanation. Rather, we are arguing against framing biology and psychology in a way that forces a choice between those kinds of explanations. The hyperbiological bias ascendant at the end of the 20th century was no wiser and no more fruitful than the hyperpsychological bias of the behaviorist movement earlier in the 20th century. Scientists can avoid turf battles by approaching the relationship between the psychological and the biological as fundamentally theoretical, not empirical. Working out the biology will not make psychology obsolete, any more than behaviorism rendered biology obsolete. Scientists can avoid dualism by avoiding interactionism (having two distinct domains in a position to interact implies separate realities, hence dualism). Psychological and biological concepts are not merely different terms for the same phenomena (and thus not reducible in either direction), and psychological and biological explanations are not explanations of the same things. If one views brain tissue as implementing psychological functions, the expertise of cognitive science is needed to characterize those functions, and the expertise of neuroscience is needed to study their implementation. Each of those disciplines will benefit greatly from the other, but neither encompasses, reduces, or underlies the other.

Fundamentally psychological concepts require fundamentally psychological explanations. Stories about biological phenomena can richly inform, but not supplant, those explanations. Yet when psychological events unfold, they are implemented in biology, and those implementations are extremely important to study as well. For example, rather than merely pursuing, in quite separate literatures, anomalies in either expressed emotion or biochemistry, research on schizophrenia should investigate biological mechanisms involved in expressed-emotion phenomena. Similarly, the largely separate literatures on biological and psychosocial mechanisms in emotion should give way to conceptual and methodological collaboration. Research in the next few decades will not need only the improving spatial resolution of newer brain-imaging technologies and the high temporal resolution of established brain-imaging technologies, but also the advancing cognitive resolution of the best psychological science.

Recommended Reading

Anderson, N.B., & Scott, P.A. (1999). (See References)

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Note

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References