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ORIGINAL ARTICLE

Cognitive Neuroscience and Depression: Legitimate Versus Illegitimate Reductionism and Five Challenges

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Abstract The provocative articles in this *Special Issue* underscore the potential of cognitive neuroscience to achieve fruitful integration across diverging levels of analysis. After a discussion of different forms of reductionism and the pragmatic, ideological, and cognitive obstacles standing in the way of achieving integrative explanatory pluralism, I outline five challenges to applying cognitive neuroscience to the study of depression and allied conditions: (1) comorbidity, (2) etiological heterogeneity, (3) ambiguity concerning causal primacy, (4) distinguishing state from trait markers, and (5) distinguishing specific from nonspecific treatment effects. Greater attention to these challenges should assist in bringing about consilience in the conceptualization of depression.

Keywords Cognitive neuroscience · Reductionism · Depression · Comorbidity

Introduction

It scarcely bears repeating that the field of psychology is fractionated (e.g., Henriques, 2004; Staats, 1991; Sternberg, 2006), but it is perhaps reassuring to know that this state of affairs is hardly new. Cronbach's (1957) description of an American Psychological Association (APA) convention of a half-century ago will surely leave many readers with an uncanny sense of deja vu:

> The scene resembles that of a circus, but a circus grander and more bustling than any Barnum ever envisioned—a veritable week-long diet of excitement and pink lemonade...This 18-ring display of energies and talents gives plentiful evidence that psychology is going places. But whither? (p. 671).

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Even today, we find ourselves at a loss to answer Cronbach's rhetorical question. Any recent attendee of an APA convention knows that psychology's intellectual fragmentation has not merely endured, but increased, over the past 50 years. With 54 divisions and more surely on the way, the APA is a loose confederacy of special interest groups, most of which barely communicate with one another. Just look at some of the division's names: Behavioral Neuroscience and Comparative Psychology, Developmental Psychology, Clinical Psychology, Family Psychology, Rehabilitation Psychology, Population and Environmental Psychology, Psychoanalysis, Behavior Analysis, Media Psychology, Addictions, International Psychology, Trauma Psychology, Military Psychology, Exercise and Sport Psychology...the list goes on and on. What do these divisions have in common? It's difficult to tell. Oddly, only one division (Division 1; Society for General Psychology) is dedicated explicitly to cross-disciplinary integration. The remaining divisions rarely visit neighboring islands, and a few show outright disdain for one another.

Moreover, there are troubling indications that the increasing pressures on young academics toward intellectual specialization are exacerbating the problem (Lilienfeld & Waller, 2005; Wachtel, 1980). Such specialization often manifests itself in scholars' decisions to focus on only one level of analysis (e.g., molecular, physiological, sociocultural) in their research programs, with scant effort to integrate findings across levels. Although ''programmatic research'' (the mantra in most academic departments these days) need not be narrow in scope, it has increasingly taken the form of a succession of boilerplate, parametric studies designed to answer relatively circumscribed substantive questions. As Sternberg (2006) noted, ''the median question size seems to be getting smaller'' (p. 15) in many domains of psychology. Clinical psychology is no exception.

Still, amidst the disturbing signs of psychology's intellectual balkanization, there are reasons for cautious optimism. As the thought-provoking articles in this *Special Issue* of *Cognitive Therapy and Research* remind us, the burgeoning paradigm of *cognitive neuroscience* offers the promise of consilience (Wilson, 1998) across diverse levels of explanation: molecular (e.g., alleles for neurotransmitter transporters), physiological (e.g., brain structures), psychological (e.g., cognitions, personality dispositions), and sociocultural (e.g., social support, interpersonal reactions) (see Cacioppo, Berntson, Sheridan, & McClintock, 2002; Ilardi & Feldman, 2001; Ilardi, Rand, & Karwoski, in press). Cognitive neuroscience is not so much a theory of a human behavior as an approach to conceptualizing behavior: it encourages us to consider and explore fruitful bidirectional connections across differing levels of analysis (Ilardi & Feldman, 2001).

As an outsider to, and decided nonexpert in, cognitive neuroscience, I read these articles with interest and enthusiasm. In the more than 30 years since Akiskal and McKinney's (1973) thoughtful attempt to provide a unified model of depression spanning diverse levels of analysis, the field of mood disorders has witnessed relatively few efforts to bridge the differing rungs of Comte's (1830–1842) ladder of the sciences. Instead, most depression researchers have felt content to concentrate their efforts on only a single level of this hierarchy. The articles in this *Special Issue* therefore represent a refreshing step in the direction of vertical integration (Cosmides, Tooby, & Barkow, 1992) and thereby provide stimulating new insights into the etiology and treatment of depression.

My friend Dr. Steven Ilardi has asked me to attempt a minor feat of intellectual integration of my own by offering some summary comments that help to place the articles in this *Special Issue* into context. Although I am afraid that I have little to say that is entirely original, I hope to generate renewed discussion concerning several unjustly neglected issues.

Reductionism: legitimate and illegitimate

When discussing the problem-solving potential (Laudan, 1977) of the cognitive neuroscience perspective, we must first think clearly about a key concept: *reductionism*. In many scientific circles, reductionism seems to be invoked primarily as a term of opprobrium against biologically oriented researchers (''You're being reductionistic!''). As a means of casting aspersions against an investigator's research approach, the invocation of this term can be an effective public relations stratagem. After all, most psychologists enjoy being tarred with the label of ''reductionistic'' about as much as politicians enjoy being tarred with the label of ''unpatriotic.''

Types of reductionism

Nevertheless, the logical nature of the objection against reductionism is rarely clear. This lack of clarity is hardly surprising given that reductionism is not one thing, but many. As philosophical psychologist Robinson (1995) observed,

> ...as ubiquitous as the ''ism'' [in reductionism] is, it cannot be taken to name members of the same orthodoxy or believers in the same sacred texts. Indeed, the term has been applied to so great a variety of perspectives, strategies, formulations, and paradigms that its mere mention these days is a warrant for a page of footnotes. And the footnotes *are* warranted, for ''reductionism'' is a word that really does refer to radically different standpoints, claims, undertakings and achievements. We mislead ourselves in applying the same term innocently across the board, and we often add a numbing contentment to our breathless confusions at the same time (p. 1; emphasis in original).

As a consequence, authors who use this term without explaining which type of reductionism to which they are referring should routinely receive frowning reprimands of dismay from journal editors.

In particular, we must sharply distinguish *constitutive reductionism* from *eliminative or ''greedy'' reductionism* (e.g., Dennett, 1995; Ilardi & Feldman, 2001; see also Robinson, 1995, for a discussion of four types of reductionism). The former brand of reductionism is accepted by all those who are mind-body monists, and should not be controversial among scientists who take materialism for granted as a working assumption. It acknowledges that all mental events are ultimately rooted in the activities in the nervous system, and strives to uncover the physiological correlates of psychological events. Nevertheless, constitutive reductionism does not assume that the physiological level of analysis is always the optimal level of analysis for *understanding* psychological events.

Eliminative reductionism, in contrast, posits that the physiological level of explanation will eventually ''gobble up'' all ''higher'' levels of explanation in Comte's pyramid (e.g., psychological, sociological), rendering them supererogatory. Like the once-popular computer game of *Pacman*, the scientific enterprise envisioned by eliminative reductionists entails a progressive cannibalization of all higher levels of explanation by a ravenously hungry lower-level monster. Ironically, a recent president of the Association for Psychological Science (APS) exemplified this view by declaring that ''psychology itself is dead'' (Gazzaniga, 1998).

Yet as many thoughtful scholars have observed, eliminative reductionism is a dubious strategy and probably an intellectual dead end, as it neglects the crucial point that differing levels of analysis afford different types of explanations. Some levels of analysis

are more informative for certain purposes than others. One could attempt to analyze the words in this paragraph by submitting their contents to an inorganic chemist, who could ascertain the molecules comprising the printed words on this page. Yet no amount of chemical analysis could shed light on what these words mean, let alone what they mean in the context of the other words in the paragraph. In this case, resorting to a lower level of analysis would be worse than useless, because it would waste valuable time in the effort to decipher the meaning of a text.

These two brands of reductionism often imply radically different interventions. To adapt an example from Kendler (2005), let's imagine that you just discovered that the newest version of SPSS's principal axis factor analysis program contained a nasty bug causing it to incorrectly iterate the communalities. One would presumably not respond to this unwelcome news by opening up the back of your computer, finding a screwdriver, removing the motherboard, and attempting to jimmy with the computer's electrical wires and circuits. Instead, you would call up the SPSS crisis hotline and berate the first person who was unlucky enough to pick up the phone.

Similarly, for some psychological problems, certain levels of analysis may be more informative than others. For example, if certain subtypes of depression are characterized primarily by inadequate environment reinforcement (Lewinsohn, 1974), intervening at the neurotransmitter level may be less fruitful than intervening at the level of inadequate social skills or disrupted interpersonal relationships, both of which may be contributing to diminished reinforcement.

Integrative explanatory pluralism

What types of reductionism are most likely to prove helpful for understanding depression and other forms of psychopathology? I am inclined to agree with Kendler (2005) and others that *integrative explanatory pluralism* (see also Mitchell et al., 1997), in which investigators explicitly attempt to combine insights from different levels of psychological analysis, is the best long-term bet. Of course, accomplishing this goal is far easier said than done, and an integrated model of depression—or other forms of psychopathology, for that matter—is unlikely to emerge out of whole cloth.

Therefore, a modest but realistic methodological strategy, at least in the short-term, is to aim for *patchy reductionism* (Kendler, 2005; Schaffner, 1994), in which researchers gradually assemble local linkages among neighboring levels of analysis in a bit-by-bit fashion. Kendler cited Caspi et al.'s (2003) recent discovery of a genotype-by-environment interaction between a serotonin transporter polymorphism and life stressors in triggering depression as one example of an important patchy reduction, in this case across molecular-genetic, psychological, and social levels of analysis. By engaging in similar patchy reductions across levels of analysis, the hope is that ''bridge laws'' that connect these levels will eventually emerge (De Jong, 2002). Most or all of the articles in this *Special Issue* exemplify laudable efforts at patchy reductionism by striving to establish local linkages across different levels of analysis for understanding depression (e.g., among brain event-related potentials, attention, and depressive symptoms).

Resistance

Still, integrative explanatory pluralism routinely meets with resistance from some academic quarters. In part, that is surely because integrative work is difficult. It requires

scholars to possess sufficient knowledge of multiple levels of analysis to be capable of collaborating with researchers in different domains of knowledge.

There are ideological obstacles as well. Throughout the history of science, most disciplines have displayed intellectual defensiveness toward an ''antidiscipline'' that is immediately beneath them in Comte's hierarchy (Walsh, 1997; Wilson, 1998). The antidiscipline has in turn traditionally prided itself on being ''harder''—or at least more rigorous—than the ''softer'' discipline above it. Physics has long been the antidiscipline of chemistry. In 1870s, for example, chemist Benjamin Brodie and many of his colleagues in the London Chemical Society lobbied for an ''atomless'' chemistry (Walsh, 1997), a view that we understandably find laughably absurd today. Yet we occasionally find comparable attitudes in the social sciences of the early twenty–first century. Some sociologists have gone so far as to argue that they can safely dispense with the traditional antidiscipline of psychology in their theoretical formulations. For example, the new discipline of ''pure sociology'' (e.g., Black, 2000) ''eliminates psychology...teleology...and *even people* from social science'' (p. 113; emphasis added). Because psychology's longstanding antidiscipline has been biology, it is hardly surprising that some psychologists continue to resist the ''incursion'' of neuroscience into their preferred level of analysis. Such resistance is counterproductive to scientific progress.

Nevertheless, I suspect that the barriers to explanatory pluralism are not merely pragmatic and ideological, but cognitive as well. All things being equal, it is probably more difficult for us to think about ''vertical'' integrations (Cosmides et al., 1992) across levels of analysis than ''horizontal'' integrations of multiple etiological variables at the same level of analysis. Young children are prone to invoke psychological explanations for psychological events, biological explanations for biological events, and physical explanations for physical events (Keil, 2006). Even as adults, we may be likely to rely on a representativeness heuristic of ''like goes with like'' (Tversky & Kahneman, 1974) when conceptualizing psychological events. As cognitive misers (Fiske & Taylor, 1991) who typically seek the path of least intellectual resistance, we may simply find it easier to think about traditionally ''psychological'' phenomena, such as depression, at their corresponding level of explanation, and traditionally ''biological'' phenomena, such as neurotransmitters, at their corresponding level of explanation. Integrative explanatory pluralism takes mental work.

Challenges

Enthusiastic as I am about the epistemological strategy of integrative explanatory pluralism, I harbor no illusions about its likely pace of progress. In the words of our previous Secretary of Defense, the effort to achieve integration across multiple explanatory levels in the domain of depression is likely to be ''a long, hard slog.''

In the interests of stimulating discussion and debate, I briefly delineate five challenges that investigators must confront when applying cognitive neuroscience to the study of depression. In doing so, I draw largely from the provocative contributions to this Special Issue. Although few, if any, of these five challenges are unique to either cognitive neuroscience or depression, several pose especially potent obstacles to scientific progress in both domains. By attending to and responding to these challenges, cognitive neuroscience researchers should find themselves in a better position to approach the long-term goal of explanatory consilience in the study of depression and allied disorders.

Comorbidity

In many domains of psychopathology, including mood disorders, the phenomenon of *comorbidity* (an unfortunate and semantically ambiguous term that has ''stuck'' despite several efforts to expunge it; e.g., see Lilienfeld, Waldman, & Israel, 1994) is more often the rule than the exception. In this case, I mean that many or most individuals with major depression fulfill diagnostic criteria for one or more disorders, including Axis I and Axis II conditions (Maser & Cloninger, 1990). This extensive diagnostic co-occurrence poses obvious problems for causal and even correlational inference, as it often renders it difficult to know whether a correlate (e.g., a component of the event related potential, activation in a brain area during fMRI) is attributable to depression, a ''comorbid'' disorder (e.g., generalized anxiety disorder, obsessive-compulsive disorder, alcohol dependence), or both.

For example, as Levin, Heller, Mohanty, Herrington, and Miller (this issue) note, the extensive co-occurrence between depression and anxiety muddies efforts to ascertain whether attentional biases and event-related potential components presumably reflecting such biases are due to one or both conditions. Making matters more complicated, they cite important research reviewed by Heller (1993), which suggests that right posterior cortex activity tends to be elevated in anxiety but diminished in depression. As they point out, ''inconsistent results regarding activity in this region for individuals with depression may reflect un-assessed comorbid anxiety.'' Ilardi, Atchley, Enloe, Kwasny, and Garratt (this issue) similarly stress the importance of determining whether P300 anomalies observed in depression are specific to this condition or extend to other conditions marked by elevated negative emotionality (e.g., anxiety disorders, somatoform disorders; see Watson & Clark, 1984). And Chapman et al. (this issue) explicitly recognize the need to differentiate depression from co-occurring obsessive-compulsive personality disorder (OCPD) in their laboratory investigation of risk aversion. Their hierarchical multiple analyses suggest that OCPD, more than depression, is linked to avoidance of risk.

Cognitive neuroscience researchers should therefore be wary about drawing conclusions regarding depression *per se* until they have conducted studies to ascertain the specificity of physiological deficits to mood disorders. Moreover, given the substantial covariation between depression and several other conditions, especially anxiety disorders, they should remain alert to the possibility of undetected suppressor effects in their data. That is, controlling for the statistical effects of covarying disorders may sometimes provide a ''cleaner'' picture of the physiological correlates of depression *per se*, thereby permitting previously undetected patterns of association to emerge.

Etiological heterogeneity

Few psychological disorders are probably unitary entities. Even if all forms of depression represented the outcome of a final common physiological pathway (Akiskal & McKinney, 1973), such as hypoactivity of the brain's reward pathways, it is almost surely the case that individuals reach the end of this long road in dramatically different ways. As a consequence, cognitive neuroscientists must remain cognizant (pun intended) of the possibility (likelihood?) that they are dealing not with one disorder, but with multiple phenocopies that stem from diverse causes.

For example, Levin et al. (this issue) refer to findings suggesting that depressed women with child abuse histories exhibit significantly lower hippocampal volume than women without such histories. If such abuse is itself linked etiologically to heightened risk for depression, it might result in a different ''kind'' of depression than depression not produced in part by early traumatic life events. Therefore, cognitive neuroscience researchers must remain alert to the possibility that certain physiological findings apply largely or entirely to certain subgroups of depressed individuals. In many cases, incorporating putative causal variables into their design and analyses could assist them in parsing such etiological heterogeneity.

Ambiguity concerning causal primacy

As Miller (1996) observed incisively, we reflexively tend to think of physiology as ''deeper'' or more ''fundamental'' than psychology. Hence the frequent use of such terms as ''underlie,'' as in ''increased right frontal activation appears to underlie many cases of depression.'' Yet as Wittgenstein (1953) reminded us, sloppy language can often get us in trouble, as we have already seen in the case of the term ''reductionism.''

In this case, we must be cautious about assuming that ostensible physiological markers (e.g., event-related potential components) are necessarily causally primary to psychological phenomena (e.g., depression, anxiety). Following the lead of Gottesman and Shields (1982), many authors have taken to calling these markers ''endophenotypes'' (internal phenotypes) to distinguish them from the more traditional ''exophenotypes'' of descriptive psychiatry, which are the signs and symptoms of disorders. Nevertheless, some of what we term ''endophenotypes'' may lie causally *downstream* of depressed mood.

For example, the elevated P300 responses following presentation of negative words found by Ilardi et al. (this issue) and the lower levels of cingulate activation found by Hugdahl et al. (this issue) may be as much consequences as causes of depressed mood. For example, these findings could reflect the activation of negative schemata and the diminished attention, respectively, that often accompany depression. Similarly, abnormalities in the error-related negativity discussed by Tucker and Luu (this issue) could in part be a result of the elevated neuroticism found in many mood and anxiety disorders. If the decreased prefrontal activation noted by Siegle, Ghinassi, and Thase (this issue) lies causally downstream of depressed mood, neurobehavioral therapies targeting such activation may not be successful, although it is possible that the causal effects run both ways. If so, it may be possible to intervene effectively at different levels of analysis (see also Ilardi & Feldman, 2001). Cognitive neuroscience researchers must therefore remember that the interplay across differing levels of analysis is often bidirectional.

Distinguishing state from trait markers

Several of the author teams in this *Special Issue*, including Ilardi et al. (this issue), Hugdahl et al. (this issue), Tucker and Luu (this issue), and Levin et al. (this issue) highlight the importance of distinguishing state from trait markers in the study of depression. Certain physiological findings could reflect ongoing depressed mood, whereas others could be vulnerability markers that persist even during symptom remission (Iacono, 1983).

Ilardi et al. (this issue) report that an enhanced P300 response to negative stimuli is present in currently depressed, but not remitted depressed, individuals. This intriguing finding underscores the importance of including both currently and previously depressed individuals in cognitive neuroscience studies of mood disorders. Similarly, Hugdahl et al. (this issue) report that fMRI activations in the inferior frontal gyrus and superior and inferior parietal lobules were present during depression, but not during remission. As they point out, these changes could reflect a return of previously compromised cognitive capacities in depressed individuals.

The distinction between state and trait effects is critical, as this distinction allows investigators to ascertain whether physiological findings reflect enduring diatheses toward depression or transient symptomatic expressions of this condition. In turn, this distinction may ultimately permit psychotherapists to determine whether they have ameliorated lasting propensities that place individuals at heightened risk for subsequent depressive episodes or merely the short-term manifestations of these propensities.

Distinguishing nonspecific from specific treatment effects

As Siegle et al. (this issue) observe, neurobehavioral therapies offer considerable promise in the treatment of mood disorders. Their Cognitive Control Training is an ingenious example of a psychological treatment derived explicitly from knowledge concerning the physiological correlates of depression, and represents translational research at its best.

Nevertheless, cognitive neuroscience researchers who hope to translate basic findings into clinical applications will need to take particular pains to distinguish nonspecific from specific treatment effects. This distinction may be especially critical for depression. Perhaps because depression is largely a disorder of demoralization, it may be especially susceptible to placebo and other nonspecific treatment effects. Indeed, it has been notoriously difficult to detect pronounced treatment specificity for depression, especially in adults (Chambless & Ollendick, 2001), probably reflecting the fact that a large array of interventions that restore hope can be efficacious for this condition. Moreover, because many cognitive neuroscience interventions surely strike many clients as new and interesting, they may be especially susceptible to ''novelty'' effects. As Shadish, Cook, and Campbell (2002) observed, ''when an innovation is introduced, it can breed excitement, energy, and enthusiasm that contribute to success, especially if little innovation previously occurred'' (p. 79). None of this is meant to dampen enthusiasm for neurobehavioral therapies, but instead to emphasize the necessity of methodological rigor and appropriate caution in promoting and disseminating such therapies to the general public. Admirably, Siegle et al. (this issue) acknowledge the importance of testing their promising cognitive training intervention in larger and more tightly controlled clinical trials.

Concluding thoughts

The articles in this *Special Issue* underscore the substantial heuristic value of cognitive neuroscience as an integrative paradigm for understanding—and perhaps one day successfully treating—depression and other psychological disorders. They also highlight the considerable benefits to be accrued from conceptualizing complex psychological phenomena at multiple levels of analysis. With further concerted efforts to achieve

integrative explanatory pluralism by patchy reductions, perhaps the APA convention of 2057 will resemble more a meeting of likeminded thinkers with a shared intellectual perspective than the sprawling 18 ring (and today, 54 ring) circus that Cronbach witnessed a half century ago.

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