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How we think about cognition, emotion, and biology in psychopathology

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Abstract

The variety of potential relationships assumed between psychological and biological concepts fosters considerable misunderstanding of what our data can tell us. A naively reductionistic view of psychological concepts is prevalent, particularly in the psychopathology literature. A series of examples of the application of psychophysiological methods in studies of cognition, emotion, and psychopathology provides a background for a discussion of these problems. Unwarranted distinctions between cognition and emotion, between classes of measures, and between psychological and biological approaches to understanding normal functioning and psychopathology undermine the ability of cognitive neuroscience to achieve its considerable potential. A nondualistic, nonreductionistic, noninteractive relationship is recommended, with psychological and biological concepts both having central, necessary, and distinct roles.

Descriptors: Psychophysiology, Cognitive neuroscience, Cognition, Emotion, Psychopathology

Cognition as a legitimate topic of research has clearly rebounded from the damage done by an overdose of behaviorism, and it looks like emotion is also well on its way to recovery. But in the middle of the Decade of the Brain, there is much uncertainty about where biology fits into such research. This question is especially salient in research on psychopathology, where the most amazing claims on behalf of psychological or biological factors sometimes arise. These claims are directly relevant to some of my work on psychopathology and emotion (for reviews, see Fernandes & Miller, 1995; Miller & Yee, 1994; Taitano & Miller, in press), and they are fundamental to a broad range of research. Here, I will review some empirical work from my lab and elsewhere that poses some challenges for how we commonly think about the relationships among cognition, emotion, and biology in basic research and in studies of psychopathology. I will then address those challenges as best I can and offer some stimulating, perhaps disturbing, and certainly unpopular positions.

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Some Empirical Questions

J. F. Kihlstrom (personal communication, October 16, 1995) recently discussed the role of behavioral data in psychology in a way analogous to how I view the role of biological data in psychology:

> As Skinner's old nemesis, Noam Chomsky, was fond of saying, "Psychology is no more the science of behavior than physics is the science of meter reading." The whole point of psychology, as many of us understand it, is to use behavioral evidence to learn something about mental structures and processes.

I would paraphrase the latter part of Kihlstrom's statement as follows: A major goal of psychophysiology, as many of us pursue it, is to use biological evidence to learn something about mental structures and processes. Although some psychophysiologists pursue biological questions, quite legitimately and fruit-

fully, my interest in biological measures is primarily as a means to address psychological questions, such as when a particular kind of emotional processing is underway or how the cognitive strategies of people at risk for schizophrenia differ from those of people not at risk. I begin with two examples of this emphasis on psychological questions.

Duncan-Johnson, Roth, and Kopell (1984) tested a hypothesis about one of the best known psychophysiological findings in psychopathology. Individuals diagnosed with schizophrenia produce a smaller P300 component of the event-related brain potential (ERP) than do unaffected individuals across a variety of paradigms, but it is not known why. Duncan-Johnson et al. tested the hypothesis that this small P300 results from a failure to encode stimulus sequence. They presented a random series of two tones differing in pitch. The tones were equally probable overall, but any given trial occurred within the context of the previous several trials, and this local probability normally has an impact on P300 amplitude. Unaffected individuals (nonpatients) generally track the recent probability of the two stimuli, with a reliable impact on their P300. Figure 1 presents data from a nonpatient control group in the left panel. At the upper left of the panel is P300 to the A tone, when it occurs after a run of four B tones. So, for the individual at that moment, the A tone was rare, and consequently P300 is large. Moving down that upper limb of the tree, the P300 to the A tone declines as additional A trials are averaged, those that followed shorter runs of B trials. There is a very orderly relationship in these data, reflecting local probability.

The individual has to encode the recent stimulus pattern to categorize the current stimulus as locally frequent or rare. Therefore, if the small P300 seen in individuals with schizophrenia is due to a failure to track the stimulus sequence, then they should fail to show this kind of amplitude tree. In the right panel of Figure 1, the tree for the schizophrenia patients is shifted downward, reflecting the typically reduced overall P300 in schizophrenia. But the key point is that the patient data produced a tree with a perfectly normal shape. This study convincingly ruled out the hypothesis about defective probability encoding in schizophrenia, providing a clear example of using biological measures to address explicitly psychological questions.

The next example employs ERP waveforms to study sentence comprehension. Our understanding of the initial words in a sentence greatly constrains what we expect later in the sentence. Kutas and colleagues have demonstrated the sensitivity of the N400 component to violations of such expectancies (for review, see Kutas & Van Petten, 1994). Figure 2 shows that when the phrase "The pizza was too hot to" ends with the word *eat*, there is very little N400. If the sentence ends with *cry*, there is a very substantial response. There are several things to consider here. First, we know that what prompts N400 cannot be the physical stimulus *cry*. N400 really seems to involve a violation of expectancy, something we conceptualize very much in psychological terms. Second, the rest of the waveform is unchanged. The effect appears to be specific to N400. Third, in addition to that specificity, N400 can be a fairly sensitive measure. The waveform produced by *drink* as the sentence ending produces an intermediate N400, reflecting the lexical priming of *drink* by the context of food consumption provided by the sentence. That parametric sensitivity is rather good for a biological measure tracking a phenomenon we construe psychologically, in this case lexical distance. We should be impressed with the success of our measures in such cases.

The use of ERP measures in the service of cognitive research is now fairly well accepted. However, the value of these measures in emotion research is not widely appreciated. In fact, such work raises difficult questions about the relationship among cognition, emotion, and biological measures.

One of the basic findings in the literature on cognitive factors in depression is a bias in favor of negative information, or what has been called mood-congruent information (Mineka &

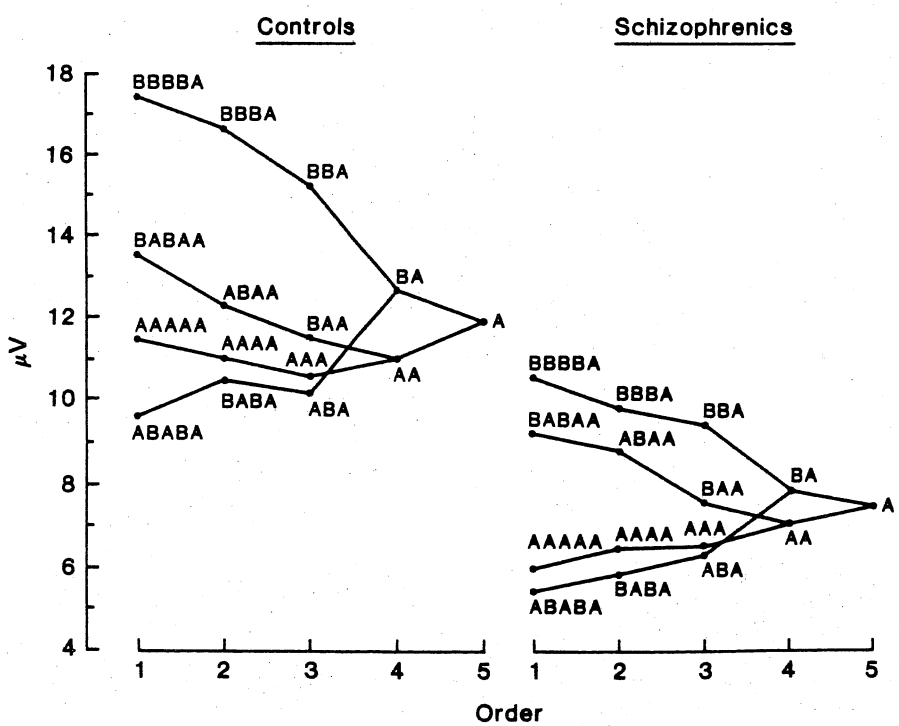

Figure 1. P300 amplitude scores from the Pz recording site for control individuals and those with a diagnosis of schizophrenia as a function of immediately preceding stimulus sequence in a 50/50 oddball paradigm. Redrawn from Duncan-Johnson, Roth, and Kopell (1984).

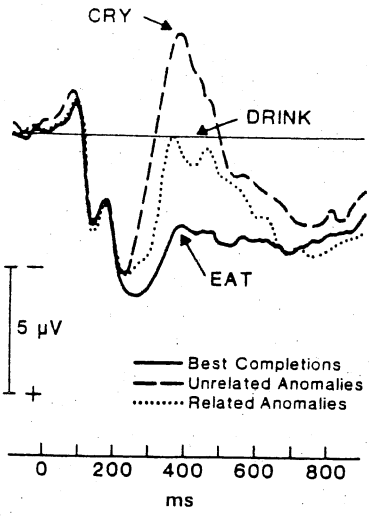

Figure 2. ERP waveforms from the Pz recording site for the final word of sentences such as "The pizza was too hot to . . ." for three different sentence endings (best completions, anomalous completions, and anomalous completions related to the most expected word). Negative is up in this and subsequent ERP waveform figures. The arrow points to the peak of the N400 component. Redrawn from Kutas, Lindamood, and Hillyard (1984).

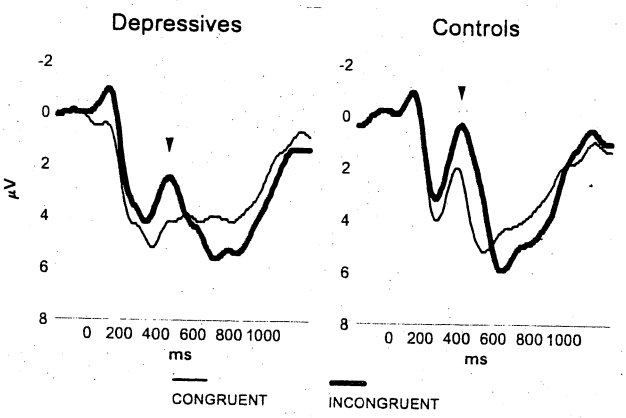

Figure 3. ERP waveforms from the Pz recording site for the final word of sentences ending in semantically congruent and semantically incongruent words, for depressed and control individuals. The arrow points to the peak of the N400 component. Data from Keller (1995).

Sutton, 1992; although see Eich, 1995). In my lab, two ERP studies of cognitive bias in inpatient depressives are underway. For the first, we adapted the basic N400 expectancy paradigm (Keller, 1995). There has been very little work with N400 in psychiatric patients (e.g., Niznikiewicz, 1996). We wondered whether emotionally pleasant and unpleasant words might elicit different responses in depressed and nondepressed individuals because of matching or mismatching the individual's mood.

We have worked with about 20 individuals with major depression so far. As a control group, we recruited matched volunteers from the community, whom we screened for psychopathology. First, we examined how the two groups would do on standard sentences obtained from M. Kutas (personal communication, December 17, 1992). Figure 3 shows the ERPs we obtained. As expected, the controls produced a clear N400 that was larger for the sentences that ended in incongruent words, such as "The pizza was too hot to cry."

Descriptively, the ERPs of depressived individuals were somewhat flatter, and their N400s were smaller, although the small N400 deflection was slightly larger than it should be for the incongruent sentences. Statistically, the incongruent sentence endings provoked a larger N400 enhancement in controls than in depressed individuals, based on a significant topographical interaction.

However, results so far for the emotion sentences are not what we expected. We developed sentences that end in pleasant or unpleasant ways. Figure 4 shows the ERP waveforms for the pleasant sentence endings. Data for the depressed individuals are represented by the thin line and for controls by the thick line. We had predicted that depressed individuals would produce a large N400 here because positive words are mood incongruent for them. Instead, they showed no negative deflection at all for positive stimuli. This outcome makes some sense clinically, however. Depressed people tend to be anhedonic, that is, unresponsive to hedonic or pleasant stimuli on more traditional measures.

If these results hold up, they will suggest that the negative cognitive bias in depression is not due to special attention given to negative information but rather to the neglect of positive information. That interpretation would be consistent with the proposal of Clark and Watson (1991) that depression is distinctive not for the presence of negative affect but for the absence of positive affect.

Our other study of cognitive bias with depressed patients used photographs of human faces posing happy, neutral, or sad expressions (Deldin, 1996). Our interest was in the processing of emotional faces as a function of depression, manifested in ERP components and recall performance. We presented a series of emotional faces in the first block of trials. That block also included emotional words, randomly intermixed with the faces. In the second block of trials, we presented the same stimuli, and we added a second set of face and word stimuli that the participant had not seen before. This is a complex study, for which analyses are incomplete. Two aspects of Figure 5 are particularly worth comment. First, the happy faces, which are the thin solid lines, appear to have the largest mean P300, at about 400–600 ms. This finding is clearer for the controls. Second, there

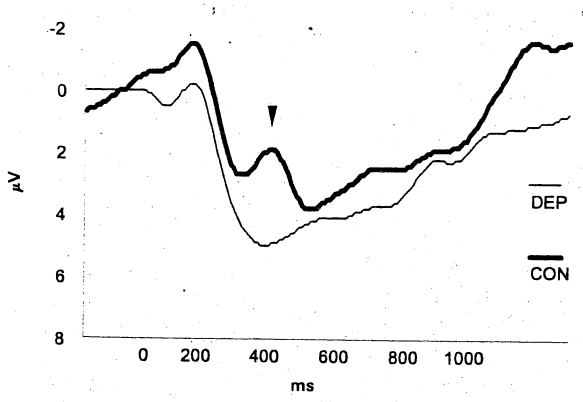

Figure 4. ERP waveforms from the Pz recording site for the final word of sentences ending in emotionally positive words, for depressed and control individuals. The arrow points to the peak of the N400 component. Data from Keller (1995).

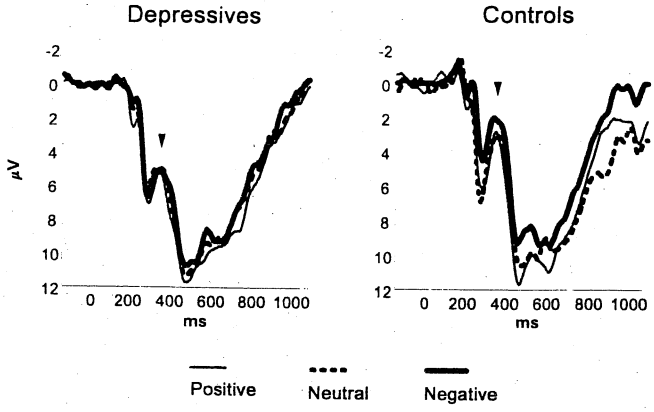

Figure 5. ERP waveforms from the Pz recording site for novel faces. The arrow points to the peak of the N200 component. Data from Deldin (1996).

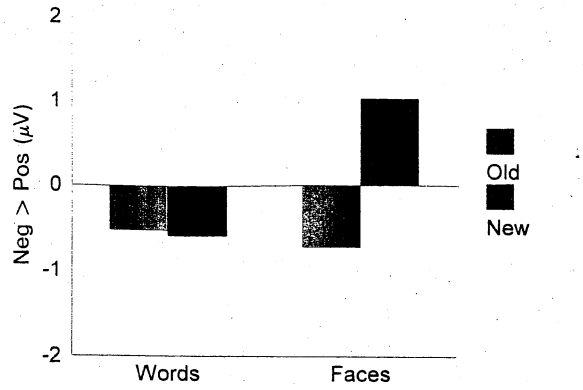

Figure 7. Valence effect on N200 amplitude scores as a function of stimulus type and stimulus familiarity, averaged across nine scalp sites. The bar above the line indicates a larger N200 for novel stimuli. Data from Deldin (1996).

is a clear group difference in a negative-going component peaking at about 300 ms, which I call N200 (marked by the arrows). The depressed patients failed to show much of an N200. Figure 6 presents that difference between the groups as a function of scalp topography, looking down on the head, with the nose at the top of the figure. The group difference is largely over right-parietal cortex, in the lower right of the figure, supported by a significant interaction.

Another finding provides more direct evidence of the sensitivity of N200 to what are conventionally viewed as emotional phenomena. Figure 7 again is a difference score, as a way to represent another significant interaction. The difference score is graphed so that a bar above the line reflects a larger N200 for negative than for positive faces. N200 enhancement was confined to the bar on the right for novel, negative faces.

Emotion has not often been explicitly addressed in ERP studies, but valence plays a prominent role in theories of emotion. Valence and arousal are often put forth as the two principal factors in dimensional models of emotion. This proposal has taken various forms over the years, but the 1990 *Psychological Review* paper by Lang, Bradley, and Cuthbert is a leading example in psychophysiology because of its advocacy of the two-dimensional structure of emotion based on its synthesis of the startle reflex and autonomic literature.

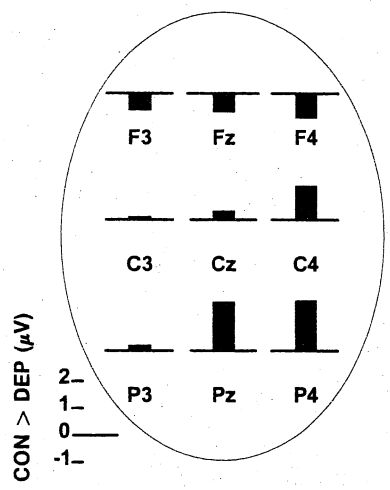

Figure 6. Scalp topography of group differences in N200 amplitude scores. Bars above the line indicate larger N200 for control than for depressed individuals. Data from Deldin (1996).

Neuropsychological theorizing within that broad dimensional tradition has recently focused on depression. Davidson and colleagues have developed a model of lateralized frontal-lobe function and emotion, relying in part on electroencephalographic (EEG) measures such as regional alpha power (for reviews, see Davidson, 1992a, 1993; Sponheim, Allen, & Iacono, 1995). Heller (1993; Heller & Nitschke, in press-a, in press-b), in turn, has construed Davidson's work on frontal laterality as specifically reflecting the valence dimension. To this she added the arousal dimension, which she proposed is managed by activity in right posterior regions. In light of Heller's model, it is tempting to interpret the right parietal N200 deficit in the depressed patients (Figure 6) as a failure of an emotional arousal system, an interpretation with considerable clinical face validity as well as a lot of support in the behavioral neuropsychology literature, especially given evidence that right parietal cortex is involved in face processing.

There may be still more to the N200 story in depression (Fernandes, Hicks, & Miller, 1995). Manipulating one subcomponent of N200, we have found normal mismatch negativity. But the N2b subcomponent of N200 was exaggerated in a mood-disordered group. The data in Figure 8 come from a two-tone oddball paradigm related to the P300 study by Duncan-Johnson et al. (1984), although they were analyzed somewhat differently (Fernandes, Giese-Davis, Hicks, Klein, & Miller, 1996). Trials are averaged according to the stimulus sequence that preceded them. There is a clear N2b enhancement for the depressed group, which is not dependent on a particular kind of stimulus sequence. We have a main effect for diagnosis.

Lest I make too good a case for a specific N200 phenomenon in depression, I must discuss one more study (Yee & Miller, 1988). We adapted a long-interval contingent negative variation (CNV) paradigm from Simons, Öhman, and Lang (1979) and Klorman and Ryan (1980) to look at anticipation of positive and negative emotional slides. We analyzed N200 to the warning tone as a function of the expected emotional valence of the slide, which served as the imperative stimulus, and also as a function of the individual's self-reported mutilation fear. N200 was sensitive to both slide valence and individual fear level. Therefore,

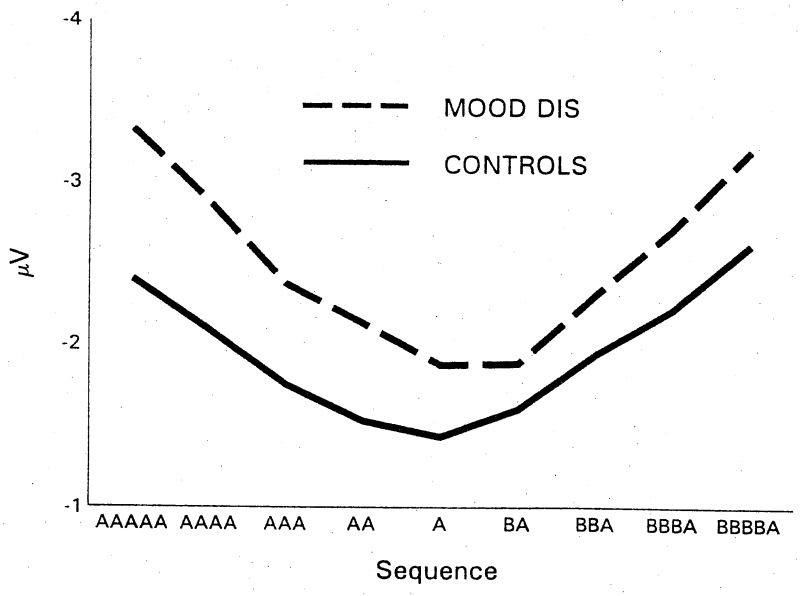

Figure 8. N200 amplitude scores as a function of immediately preceding stimulus sequence in a 50/50 oddball paradigm for nonpatients diagnosed with a mood disorder and for nonpatients receiving no diagnosis. N2b is scored from a difference waveform obtained by subtracting ERPs from an *ignore* condition from those from a *count* condition, removing exogenous components and mismatch negativity. The differencing is done for averages obtained separately for each set of A trials preceded by a given run of A or B tones, shown on the *x* axis. Data from Fernandes, Giese-Davis, Hicks, Klein, and Miller (1996).

clinical effects on N200 are not confined to depression. I have been emphasizing N200 in part because it is almost never used in studies of emotion, and it is rarely used in studies of psychopathology. The available data are interesting, but we need further analysis and follow-up.

With the empirical examples presented so far, I have attempted to establish three things:

1. Biology versus psychology. We can use biological measures to address questions conceived in purely psychological terms, whether or not we thoroughly understand the biophysics of our measures.
2. Cognition versus emotion. We can use "cognitive" ERP measures to study "emotional" phenomena. We can do so without limiting ourselves to what individuals say about their subjective experience.
3. Inferences about psychopathology. We can use those measures in seamless transitions between normative research and studies of psychopathology. Clinical research is not some narrow, occult practice, divorced from what other scientists are doing.

I acknowledge that no strategy can be considered comprehensive as long as it leaves out interesting, relevant phenomena, which for these kinds of studies clearly includes basic neuroscience. The pursuit of psychological and biological questions will have to converge to be comprehensive, a point I will develop below. An example is the finding of a deficit in N200 production in depression, measured over the right parietal cortex. It would be interesting to increase the electrode density to try to verify the anatomical source of that effect in that region, to strengthen the case for invoking the psychological concepts in Heller's neuropsychological model as a basis for interpreting our ERP data.

Some Conceptual Questions

However, in the middle of the Decade of the Brain, the field of psychopathology does not need more preaching about the importance of biology. Rather, we need to tackle some difficult, confusing issues about the logical relationship between biology and psychology, about the role of biological measures in studies of cognition and emotion, and about how all of this applies in research on psychological dysfunction. Here, I revisit those three domains. I intend this survey to be provocative rather than decisive. I wish I could promise to lay all these issues to rest, but I am still stumped myself about some of them.

Biology Versus Psychology

Especially in psychopathology, there often seems to be an ideological war between psychologically and biologically inclined researchers, with intellectual atrocities committed by both sides. Let us consider four examples in some detail.

Biology is more fundamental than psychology. In my reading, I often run across phrases such as "biological underpinnings" (Holden, 1991a, p. 1450), "biological substrates" (e.g., Kandel & Squire, 1992, p. 145), "neural substrates" (e.g., Andreasen, 1984, p. 242), "neurochemical basis of brain functions" (e.g., Zuckerman, 1995, p. 325), "underlying brain dysfunction" (Rubin, Villaneuva-Meyer, Ananth, Trajmar, & Mena, 1992, p. 695), "neuroscience basis" (e.g., Sprague, 1995, p. 514), and "physiological foundations" (e.g., Tiitinen, 1995, p. 50). This is a popular and time-honored motif. William James' view that emotion *is* the peripheral organ activity that accompanies what we call an emotional state is an extreme example. (For further discussion of relevant views of James, see Cacioppo and Berntson, 1992. For numerous additional examples from modern authors, see Ross and Pam, 1995.) Despite the popularity of the view that biology underlies psychology, it rests on an untenable assumption about the relationship between psychology and biology. Most importantly, these phrases appear to assume that biological phenomena are somehow more fundamental than psychological phenomena. Sometimes, this position is carried to the extreme of a naive, breathless reductionism, but often it is more subtle, incomplete, or simply incoherent. Statements that psychological events are nothing more than brain events are clearly logical errors (see the extensive analysis

by Marr, 1982), but more cautious statements, such as that psychological events are "implemented" or "realized" or "embodied" in brain events, are incomplete in what they convey about the relationship between psychology and biology. I encourage this notion of implementation and will suggest some conditions for its use.

Although much rarer, I also run across the converse problem—psychology allegedly underlying biology. For some cognitive psychologists, reaction time is *the* criterion. Whatever biomechanical apparatus is involved in pressing buttons is sometimes treated as inherently uninteresting, simply driven by psychological events and goals. Thus, if one sort of underlying is possible, why not the converse? Biology would then just be the implementation of psychology. But psychology would be where the action is. Perhaps this would justify a Decade of Cognition.

Psychophysiological research provides ready examples of the trouble that this *underlying* idea gets us into. Rather than attributing mood changes to activity in specific brain regions, why not attribute changes in brain activity to changes in mood? In the N400 work I discussed above, does brain activity manifested in N400 underlie language comprehension, or is language comprehension driving brain activity? In Davidson's (1992a, 1993) data on regional brain activity in depression, are people depressed because of low left frontal activity, or do they have low left frontal activity because they are depressed? It is crucial to understand that these are not empirical questions but are logical and theoretical questions. They turn on the kind of relationship that we believe that psychological and biological concepts can have.

I am not sure that it is useful to assert that biology underlies psychology, or vice-versa. By analogy, we would not say that the gears in a clock underlie the concept of time keeping or that the gears are more fundamental than the concept of time keeping. In fact, we can keep time without any gears at all. Conversely, we would also not say that time underlies the gears, in part because we can do things with gears that have nothing to do with time keeping. Time keeping and gears simply belong to different conceptual domains. Quoting Marr (1982, p. 25) on the psychology and biology of vision:

> The explication of each level involves issues that are rather independent of the other [levels of analysis] . . . some phenomena may be explained at only one or two [levels]. . . . In attempts to relate psychophysical problems to physiology, too often there is confusion about the level at which problems should be addressed.

Saying that time keeping and gears belong to different domains does not entail dualism, if one does not ascribe some separate reality to the notion of time keeping. A clock could be described in terms of gears, but it could also be described in terms of the fact that it keeps time (for discussions of such a functionalist perspective, see Dennett, 1995; Fodor, 1968; Kozak & Miller, 1982; Miller & Kozak, 1993; for some reservations about it, see Churchland, 1986). A good explanation of a clock would have to provide both stories. Neither story would render the other redundant, and neither would be sufficient on its own (see Cacioppo & Berntson, 1992, for a similar point about the need for both parts of the story). Kosslyn and Koenig (1992, p. 4) made the same argument with a different metaphor:

> The aim is not to replace a description of mental events by a description of brain activity. This would be like replacing a description of architecture with a description of building materials. Although the nature of the materials restricts the kinds of buildings that can be built, it does not characterize their function or design.

Similarly for Marr (1982, p. 27) on psychological concepts such as perception:

> An algorithm is likely to be understood more readily by understanding the nature of the problem being solved than by examining the mechanism (and the hardware) in which it is embodied . . . trying to understand perception by studying only neurons is like trying to understand bird flight by studying only feathers . . . we cannot understand why retinal ganglion cells and lateral geniculate neurons have the receptive fields they do just by studying their anatomy and physiology.

Underlie is simply the wrong way to characterize the relationship between biological and psychological concepts. The alternative characterization that one sometimes hears, that cognition is *implemented* in neural systems, is much more tractable. Such a construal leaves unanswered some questions about the relationship between psychology and biology, but at least it avoids the logical error of reductionism or the nonsensical claim that psychological phenomena *are* biological phenomena.

We should stop talking in terms of what underlies what. In particular, neural activity does not underlie cognition or emotion or psychotic delusions or memory biases. There is no homuncular brain region "doing" a particular psychological function, in the reductionist sense that everything the psychological concept refers to is fully captured by a description of the brain tissue. Neural activity is clearly necessary for people to think and feel, but the physical implementation of functions such as keeping time or categorizing stimuli or feeling sad is not the function implemented. The mechanism is not the concept it implements. Marr (1982, p. 28) stated, "For far too long, a heuristic program for carrying out some task was held to be a theory of that task, and the distinction between what a program did and how it did it was not taken seriously."

Confusion about causality is especially rampant in psychopathology research. Here is the opening sentence in a prominent article in *Science* about brain imaging technologies, written by a highly regarded researcher: "Psychiatrists have known for at least 100 years that mental illnesses must be fundamentally due to perturbations of normal neural activity in the brain" (Andreasen, 1988, p. 1381). So much turns on the precise meaning of "due to" here. If it is intended in some causal sense, such that biological events are causing (or "underlying") psychological events, then there is no room for psychological trauma, or poverty, or learning history to play a key role in psychopathology, yet these are all well established factors. The problem is not solved or avoided by placing trauma or poverty or learning history earlier in the causal chain than biology. If trauma fosters psychopathology via some mediating biological process, then the psychopathology is not fundamentally due to something biological. The trauma would be the fundamental factor, not the biological phenomenon that results from it, and we would still need to determine the mechanism by which trauma affects biology.

A very prominent example of the problematic implications of such an overly biological view of psychopathology is a print ad placed by the highly respected National Alliance for Research on Schizophrenia and Depression (NARSAD, 1995, p. 8), with the headline: "Depression. A flaw in chemistry, not character." The ad includes the text, "What causes depression? According to recent medical research, depression is caused when an insuf-

ficient level of the neurotransmitter serotonin is passed through the synapses in the frontal lobes of the brain." What role is left by such a statement for learning history, which has a clearly established role in some kinds of depression? What is implied by such a statement regarding suitable treatments for depression, and how does that square with what we know about effective treatments for depression?

A further example of why an entirely biological view about the basis of psychopathology is inadequate is recent evidence about a relationship between educational history and dementia. Prigatano, Parsons, and Bortz (1995, p. 400) reviewed studies indicating that "level of education may serve as a protective factor against cognitive decline" related to Alzheimer's disease, with "a consistent relationship between fewer years of education and decline in cognitive abilities independent of other demographic variables [such as] age, occupation, or income." There is no dispute about whether Alzheimer's disease involves biological changes. But what is driving this disease and the associated cognitive decline? It appears that a purely biological account will not suffice.

I am not arguing for a psychological or sociological explanation of psychopathology *instead of* a biological explanation. I am arguing against framing psychopathology in a way that forces a choice between those kinds of explanations. At present, a hyperbiological bias is ascendant, but this is no wiser than the hyperpsychological bias of the behaviorist movement in clinical psychology earlier in this century.

The hyperbiological bias in some of the foregoing quotations has a number of problematic implications. For example, for the general public, the more biological a given behavior is, the less control the behaver is believed to have over it (Wright, 1987). Thus, "The victim of mental illness has not brought it on himself, and he cannot cure it through his own free will" (Andreasen, 1984, p. 219). Such a viewpoint appears to limit psychological factors to the red herring of willpower, dangerously close to dualism (on the goal of excising willpower, intentionality, etc., from a mature psychological science, see Dennett, 1978; Miller & Kozak, 1993). It is not a large step from saying that alcoholism is (nothing but) a biological disease or that depression is (nothing but) a chemical imbalance to saying that psychopathy and criminal behavior are also biological diseases, for which the psychopath cannot be held responsible. I suspect that most people in and out of science would not be comfortable with the moral and legal implications of that position.

The issue of social or moral responsibility for dysfunctional behavior ought to be understood as entirely distinct from the psychological versus biological nature of a disorder. We might legitimately hold people responsible for some of their biology, and we might legitimately relieve them of responsibility for some of their psychology. Thus, the well-intentioned public service ad quoted above may not protect diagnosed individuals as it hopes to, because construing illness as biological ultimately provides no shelter from social stigma.

Biological theories of psychopathology. This confusing notion that biology underlies psychology sets us up to misconstrue so-called biological theories of psychopathology. A classic example is the misnamed *dopamine theory of schizophrenia.* The basic idea in one form of the theory is that in certain areas of the brain in in individuals with a diagnosis of schizophrenia there are too many dopamine receptors, so that the dopamine neurotransmitter system is too active. Some data are strongly supportive of this theory, and other data are fairly damaging to it (for review, see Davis, Janicak, Preskorn, & Ayd, 1994; Davis, Kahn, Ko, & Davidson, 1991), but the issue here is not the evidence for the theory. The issue is that it cannot really be a theory of schizophrenia in the first place, on logical grounds.

The term *schizophrenia* historically refers to phenomena that are inherently psychological, such as thought disorder and anhedonia. No biological finding about people with schizophrenia could alter the nature of the phenomena the term has historically referred to. Even if we change what the term *schizophrenia* means, its traditional referent, which will always be of interest, remains psychological. An excess of dopamine receptors may indeed be common in schizophrenia. But thought disorder, which is a cognitive construct, is not in the domain of neural systems, it is in the domain of cognitive systems. It is certainly valuable to study the neural systems that implement interesting cognitive phenomena, but an explanation of those neural systems would not be an explanation of the cognitive phenomena (Marr, 1982).

This confusion about the functional implementation of cognition in neurons sometimes goes as far as making claims about the physical location of cognition in neurons. Consider a statement by Kandel and Squire (1992, pp. 143–144):

> Cognitive neuroscience . . . begins with localization within the brain of various cognitive capabilities. . . . It has now become possible to localize mental functions to particular sets of regions. . . . The development of realistic models of cognitive processes requires the ability to locate cognitive function to particular regions of the brain.

But cognitive events do not have a location, any more than the concept of keeping time has a location. Specific physical clocks have locations, but the concept of time keeping is not exchangeable with or reducible to a set of clocks, nor is a description of the architecture of a building interchangeable with a description of building materials.

All *instances* of human psychological processes unfold via brain tissue. But it is a fundamental, logical error to state that psychological processes *are* brain processes or are located in brain tissue. Keeping time is a functional property of a clock; conforming to a school of architecture is a structural property of a building; and particular brain tissue at a particular time in a particular individual may exhibit functional or structural properties of interest. But those respective properties are not located in the clock, the building, or the brain tissue.

Distinguishing implementations from the concepts they implement does not entail dualism if we do not posit a separate reality for the concepts. A geometric proof has no location, but believing in geometry does not make one a dualist. Provided that triangles and mental events are not viewed as having a corporeal reality separate from the usual physical world, standard scientific materialism can accommodate cognitive events without placing them in the brain or anywhere else.

The upshot of this discussion is that the so-called dopamine theory of schizophrenia cannot be a theory about schizophrenia. It can only be a theory about the biochemistry of schizophrenia. (For the harsher stance that it is not a theory at all, see Ross and Pam, 1995.) Even if we end up convinced that the dopamine theory in some form is right about the biochemistry of schizophrenia, we will need other theories to account for other aspects of schizophrenia. If we manage to develop a comprehensive theory, it will have to be about more than just

biochemistry. It will have to account for the psychological phenomena that have traditionally defined schizophrenia. There is clearly a very important biochemical story to be told about schizophrenia, but we should not mistake it for an adequate story about schizophrenia.

Another prominent example of the limits of biological approaches to psychopathology is biochemical models of depression, particularly in the age of Prozac. It may be that the memory bias in depression involves certain consistencies in neural events across individuals and occasions. Even if we are able to outline what goes on at a neural level, we will undoubtedly still refer to the phenomenon as a *memory bias*—a purely psychological term.

This kind of point is routinely made by philosophers of science (e.g., Fodor, 1968; see also Churchland, 1986; Kozak & Miller, 1982; Miller & Kozak, 1993). A favorite example is that the category of *mousetrap* means more than just an enumeration of specific mousetraps. The wood slabs and iron springs of some mousetraps do not underlie the concept of a mousetrap. To build a better one, one has to go beyond the examples already available. No specific physical property defines *mousetrap*. Similarly, we do not need any consistency in neural implementations of thought disorder or memory bias for those concepts to be meaningful. And if we do identify some neural consistency, we will not have reduced the psychological concepts to biological concepts. Cognitive neuroscientists are beginning to understand this.

> The mind is what the brain *does*: a description of mental events is a description of brain *function* [not brain *tissue*]. . . . Consciousness is not the same thing as neural activity; phenomenological experience cannot be described in terms of ion flows, synaptic connections, and so forth. Consciousness and brain events are members of *different categories*, and one cannot be replaced by the other. (Kosslyn & Koenig, 1992, p. 4, 432, emphasis added)

Let us take a more radical step regarding the relationship of biology and psychology in psychopathology. It is well established that measures of what is known as *expressed emotion* predict relapse rate in schizophrenia (Hooley, Rosen, & Richters, 1995; Koenigsberg & Handley, 1986). Expressed emotion, which is something of a misnomer, refers more specifically to expressions of certain sorts of negative emotions, emotional attitudes, or emotional overinvolvement by people working closely with individuals with a diagnosis of schizophrenia, such as family members or hospital staff. It is difficult to imagine that it would ever be possible to account adequately for the relationship of expressed emotion and relapse rate in terms of dopamine. That is not to say that dopamine is unimportant in schizophrenia or even irrelevant to the impact of expressed emotion on affected individuals. In fact, it might turn out that expressed emotion correlates substantially with dopamine receptor density.

But, if we are willing to consider that biochemistry could ever provide a comprehensive account of psychopathology, could we not as an antidote propose conversely that hostile interpersonal behavior might underlie a patient's dopamine problems? Maybe others' expressed hostility pumps up the patient's adrenaline, which is chemically related to dopamine, and maybe the dopamine receptors of individuals with a diagnosis of schizophrenia are unusually responsive to or potentiated by excess adrenaline. Thus, maybe hostility, a psychological concept, drives a biological characteristic of schizophrenia. With equal legitimacy, the hyperbiological camp could counterattack. For example, drugs that alter the system may be an important means of altering the relationship between expressed emotion and relapse.

Such explanatory elitism is all too common. The point to be drawn from the present discussion about the expressed emotion literature is that the evidence for the dopamine theory should not lead us to limit our thinking about schizophrenia to biological conceptualizations or interventions, nor should the evidence for the expressed emotion relationship lead us to limit our thinking about schizophrenia to psychological conceptualizations or interventions.

The first two examples in the ideological war between psychology and biology—the assumption that biology underlies psychology and the faith that biochemical theories of psychopathology could be adequate theories of psychopathology—emphasize the sins of the hyperbiological camp. For the third and fourth examples, let us put the shoe on the other foot and look at some confusions that arise about the wonders of traditional psychological measures in our work at the expense of biology.

Psychophysiology is capable of only gross discriminations. I have heard claims that psychophysiological measures are not up to making specific psychological discriminations and that verbal data are capable of finer distinctions. A common version of this view is the assumption that self-report is a better measure of emotional state than is physiology. I see two problems here. First, I do not know why we should assume that self-report is the best window on mental processes. That notion rests on assumptions about both mental events and the relationship of those events to observable data that we need not make. Second, we actually have plenty of empirical evidence that psychophysiological measures can be fairly precise. Recall the N400 data, where "The pizza was too hot to drink" produced an intermediate N400, reflecting the lexical distance between *drink* and *eat*.

Now, having just read that sentence, how many of you had an N400 at the word *drink*? How many of you can quantify your own semantic distance between *eat* and *drink*? I would rather rely on your N400 than your self-report to address such questions, particularly if I also value temporal precision.

Figure 9 presents another example of psychophysiology doing an impressive job of discriminating mental events. These data are from a dual-task study by Sirevaag, Kramer, Coles, and

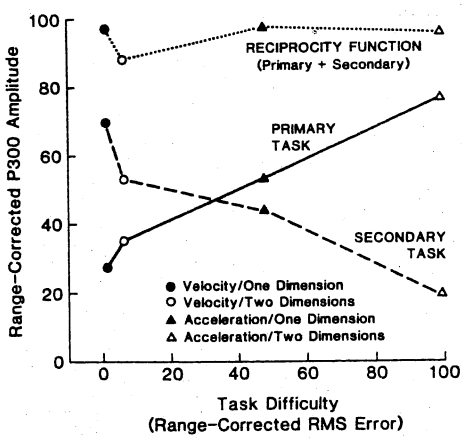

Figure 9. PCA-derived P300 amplitude scores from the Cz recording site as a function of relative task priority. Reprinted from Sirevaag, Kramer, Coles, and Donchin (1989), with kind permission of Elsevier Science.

Donchin (1989). The two diagonal lines show scores for the P300 component of the ERP for each of the two tasks, under different conditions of resource trade-offs. The top line illustrates a remarkable consistency in summed P300 amplitude at different trade-offs between the tasks, implying a consistent pool of total cognitive resources available. It is hard to imagine that self-report data could achieve this kind of precision in such a task.

As a third example of the relative precision of self-report and biological data in the measurement of psychological measures, Cacioppo, Crites, Gardner, and Berntson (1994) obtained ERPs while individuals viewed words representing personality traits. The traits were very positive, moderately positive, moderately negative, or very negative. The choice reaction time task was to judge each trait simply as positive or negative. The negative traits prompted larger P300s than did the positive traits, although that finding must be interpreted carefully because negative traits were rarer, and rareness tends to augment P300. What was impressive for present purposes is that the P300 systematically differentiated all four levels of emotional valence, even though overt behavior differentiated only two levels.

I think that, in general, it is simply wrong to bet that biological measures are less capable of fine discriminations of psychological phenomena than are more traditional psychological measures. Let us not sell biological data short in terms of their potential specificity for measuring psychological phenomena. The specificity we can achieve is probably going to depend on our experimental design and our honing of methodology, not the nature of our measures.

The gold standard. The fourth example of the biology/psychology war also faults the hyperpsychological camp. To extend the critique of the implicit privileging of certain kinds of measures over others, that bias can be framed in terms of what should be the gold-standard criterion for making inferences about mental events.

Claims are sometimes made about biological measures being more objective or less subject to demand characteristics, but such claims are probably groundless (Miller & Ebert, 1988). For example, in a series of studies on heart rate biofeedback, researchers unexpectedly found but then replicated a striking interaction of the interpersonal style of the experimenter with heart rate control strategy. Cuthbert, Kristeller, Simons, Hodes, and Lang (1981) investigated the relative effectiveness of meditation, heart rate biofeedback, electromyogram (EMG) biofeedback, and monetary incentive for control of heart rate. The specific pattern of results is too complex to report here, but Cuthbert et al. (1981, p. 518) concluded that "the effectiveness of any method for achieving relaxation (or physiological control) rests on a complex interaction between the informational and motivational imperatives of the stimulus context and definable aspects of the interpersonal exchange between subject and experimenter."

We should not treat biological data as immune from the interesting confounds that self-report data fall victim to. However, sometimes we seem to assume, at least implicitly, that self-report is the gold standard for measures of emotional state. Most clearly, it is often assumed to have more direct access to subjective experience. Again, I do not see a basis for that assumption. In fact, I have a lot of trouble with that word, *subjective*. I do not understand how measuring so-called subjective self-report is more subjective or closer to some inner core of the subject (cf., e.g., Robinson, 1995) than is measuring blood pressure or CNV. Each measure is about that individual subject; each measure is vulnerable to a host of interesting, problematic phenomena that psychologists study, such as demand characteristics involving the experimenter; and each is potentially driven to some degree by whatever mental phenomena are unfolding. Why not turn *first* to psychophysiological data if your goal is to study subjective experience? Why start, and especially why stop, with self-report?

Michael Kozak and I wrote a paper long ago arguing against treating verbal self-rating data as having special status, closer to internal processes, or as isomorphic with or more directly related to cognitive processes than are other kinds of data, such as physiology or overt behavior (Kozak & Miller, 1982; see also Miller & Kozak, 1993). There is an old debate in social psychology about the validity of introspective reports. Nisbett and Wilson (1977) proposed that introspection is not even what is actually happening when subjects make such reports. Instead, they suggested, subjects are deducing partially informed guesses based on their own theories of themselves. From this view, self-report has no special status as a window on subjective experience. It certainly does not sound very promising as a universal gold standard.

This problem of what should be the gold standard in research on mental events is especially inconvenient in studies of emotion. How do we verify the presence of the emotional state we think we are studying? Imagine all of the measures one could have available in a study of emotion. We would designate a subset of the measures as independent variables and another subset as dependent variables. How would we decide which is which? Whatever our preferred resolution to that dilemma about independent and dependent variable selection, there is little consensus among emotion theorists.

Going a step further, in our grand hypothetical study of emotion, it would be highly desirable to designate a third subset of variables that would serve as a manipulation check. Those variables would tell us when the emotion of interest is actually present or when the processing of interest is happening. But how would we sort our measures into independent variables, dependent variables, and manipulation checks? Should self-report be the criterion for the presence of a given emotion, or should heart rate or N200 or avoidance behavior? Should the answer vary with the emotion?

Partly to deal with that question, Davidson, Ekman, Saron, Senulis, and Friesen (1990) proposed a stringent set of eight criteria that a study of emotion must meet. They made a compelling argument, but virtually no available study meets the criteria. I am not aware of a solution to the issue of what the gold standard should be in emotion research. Until we settle that issue, the emotion literature will remain messy. Conceptual replications will be very difficult as long as we do not agree on the status of our measures, especially which measures should serve as a manipulation check.

The problems I have been discussing come from an unfortunately common antagonism between biology and psychology. In these examples, we have seen that antagonism played out in a variety of ways, to the detriment of our field. We have artificially and unnecessarily set up biology and psychology as competitors for mindshare, for research funding, and for scientific legitimacy. The assumption that one is more fundamental than the other is ill formed. The wager that one will prevail over the other or that we can explain one in terms of the other is philosophically naive.

Cognition Versus Emotion

The second big-picture issue I want to revisit is an antagonism within psychophysiology. The general point is that we often assume stark, unnecessary distinctions between cognition and emotion, and we take on a lot of unfortunate baggage in doing so.

CNS versus ANS. We often seem to view direct measures of central nervous system (CNS) activity, such as EEG and ERPs, as being appropriate for studying cognition and direct measures of visceral or peripheral physiology, such as pulse transit time or electrodermal activity, as being appropriate for studying stress or emotion. However, cognitive studies using peripheral measures are numerous, though they are rarely noted in current cognitive neuroscience literature, including ERP papers. By my count, 11 of the previous 12 Society for Psychophysiological Research presidents have relied heavily on autonomic nervous system (ANS) measures in their work, much of which has addressed cognitive processes. Conversely, although fewer in number, studies of emotion using brain-wave measures are becoming more common.

Figure 10 is from a recent ERP study of emotional stimuli by Naumann, Diedrich, Becker, Maier, and Bartussek (1996). They selected slides from the International Affective Picture System, which was developed and normed cross-nationally by Lang, Öhman, and Vaitl (1988). The slides have been standardized in terms of valence and arousal ratings. Naumann et al. found significant differences in ERP component amplitudes as a function of emotional valence. The earliest two such components are illustrated here. In both cases, positive-valence slides evoked the most positive response. Broadly consistent results showing a more positive ERP response were recently reported by the Lang group (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 1996).

Just as I argued against inappropriate distinctions between psychological and biological stories, the second big-picture point is that one need not choose (and may even have trouble distinguishing) between exclusively cognitive and exclusively emotional accounts of psychophysiological phenomena. The unnecessary conceptual segregation of central and peripheral measures may reflect a widely held, although thoroughly discredited, view of the physiology of emotion as undifferentiated. Given the highly influential work of Stanley Schachter over 30 years ago (Schachter & Singer, 1962), many lay people and even many scientists assume that there is no differential physiology for different emotions and that emotional differentiation is a purely cognitive process. This model seems to appeal both to people holding a dimensional view of emotion and to those holding a categorical view. Thus, one should measure peripheral physiology to assess the degree of nonspecific arousal, but one must rely on self-report ratings to study emotional specificity achieved through cognitive operations.

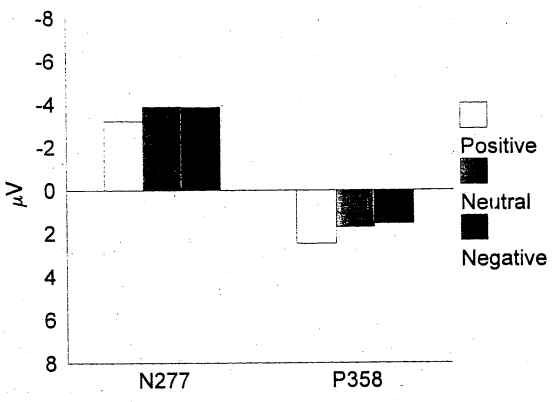

Figure 10. ERP amplitude scores as a function of the emotional valence of a slide. Data from Naumann, Diedrich, Becker, Maier, and Bartussek (1996).

However, this model overlooks the very substantial literature on the psychophysiological differentiation of emotion. The January 1992 issue of *Psychological Science* contains an impressive series of brief reviews of emotion research, several of which make a persuasive case for psychophysiological specificity in emotion. I especially recommend the paper by Lang, Bradley, and Cuthbert (1992) for readers with a dimensional inclination or the paper by Levenson (1992) for those categorically disposed. For those uncertain which way to think about emotion, the paper by Davidson (1992b) incorporates elements of both dimensional and categorical approaches.

There are many dozens of studies providing evidence that different emotional states are associated with different physiological states, although there are relatively few studies aimed primarily at that question (for skepticism about the consistency of the evidence across studies, see Cacioppo, Klein, Berntson, & Hatfield, 1993; Zajonc & McIntosh, 1992; for a particularly sophisticated discussion of the meaning of physiological specificity in emotion, see Stemmler, 1989). I hope that the recent surge in EEG and ERP research on emotion will further undermine the assumption of physiological nonspecificity in emotion. Conversely, I urge ERP researchers to pay more attention to the extensive ANS literature on cognition. Not only would this provide converging measures, but it would foster synergy with the relatively sophisticated concepts and empirical findings available in the ANS literature on some bedrock phenomena in psychophysiology such as orienting.

Process versus state. In addition to the traditional distinction between cognition and emotion on the basis of CNS versus ANS measurement, there is a more subtle distinction that is commonly made, in which cognition is construed as a process, and emotion is construed as a state. This distinction has a number of ramifications, some of which complicate the potential convergence of concepts from the literatures on cognition and emotion. What happens if we reject this distinction? If, for example, the physiological activity that accompanies an emotion is viewed as being *part of*, not a *response to*, the emotion (Lang, 1979, explicated in Miller & Kozak, 1993), then emotion becomes an activity, not a state—something one does, not something one has. Certain tools of cognitive neuroscience immediately become more appropriate for research on emotion. For example, how one would test proposals regarding memory bias in depression or attentional bias in anxiety would depend on how one believes emotion can be engaged. Taitano and Miller (in press) have suggested that we adopt a processing approach in conceptualizing emotion (see also Foa and Kozak, 1986; Rachman, 1980) and that we attempt to model emotion phenomena with what are formally the same kinds of models that are used to model cognition. At minimum, we should recognize how prevailing assumptions about the nature of emotion distance it from cognition, and we should question whether this fundamental gap is optimal.

Precision versus romance. We have an unspoken bias that cognitive measures are precise and that emotional measures are temporally (and maybe even substantively) vague. This bias makes emotion a second-class citizen in science, but it also serves popular, romantic notions about the ineffability of emotional experience.

Some measures of peripheral physiology do not provide the kind of temporal precision available with some direct brain measures. But there are at least two responses to the assumption of the fuzziness of emotion. First, some peripheral measures do provide potentially high temporal precision, such as facial EMG, cardiac cycle-time effects, and the startle reflex. Second, some direct brain measures are rather weak in temporal specificity, such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI). The ERP community likes to point out that ERPs are substantially ahead of fMRI in temporal resolution. Rarely noted is that some peripheral measures are ahead as well.

We often implicitly hold cognition to be scientifically superior, or cleaner, or more accessible. But we can study normal emotion and its role in psychopathology in much the same way that we study cognition. In fact, the distinction between cognition and emotion may be less useful and less valid than we take for granted.

Inferences About Psychopathology

The final big-picture issue is how the biology–psychology relationship plays out in psychopathology research. There has been important progress in improving spatial resolution in the measurement of localized brain activity in recent years with measures such as EEG, magnetoencephalography, and fMRI. Unfortunately, problems then arise in attempts at anatomic localization in studies of psychological functions via comparisons of experimental conditions, emotional states, or diagnostic groups.

Where the action is. A variety of clinical and experimental data encourage us toward very simplistic, premature conclusions about anatomic localization. This is one temptation to resist. For example, in studies of temporal binding, which I have been investigating with Art Kramer (Weber, Kramer, & Miller, in press), the basic empirical issue is this: Because different parts of the brain seem to be involved in processing different features of a stimulus, how does the observer bind together those anatomically distinct areas to perceive the object as a whole?

Even if we accept one proposal that, at a neural level, the different regions somehow briefly entrain each other via phase-locked firing rates, we are going to have trouble with statements such as this one, in a report on PET imaging: "[Visual attention] is done by increasing [neural activity] in the specific regions of the brain involved in processing the attribute you are attending to" (Taylor, 1990, p. 57). Clearly, this account of things cannot be adequate. It does not deal with the importance of the connectedness of the regions; it does not deal with attention as a psychological construct at all. Just as thought disorder cannot be explained purely in terms of dopamine receptors, attention cannot be reduced to activity in the brain region or regions handling a single attribute of the object. Furthermore, attention cannot be accounted for solely in terms of biological concepts about how distinct brain regions communicate. Attention is a psychological construct that has meaning independent of any biological implementation.

More generally, the problem is concluding prematurely that the brain site directly producing what we measure is the location that implements the interesting processing. Instead, the tissue producing the physiological manifestation that you are measuring directly may not be where the action is but may be driven by other tissue, where the action really is (for a similar view, see Robinson, 1995). A telling example is the ongoing search for the generator or generators of P300. This quest has to be approached in at least two ways. First, we must determine what tissue is directly producing the voltage we measure at the scalp. Second, we must determine what psychological processing, implemented in what tissue, is driving the tissue producing the voltage we are measuring. This second problem is more interesting, but it may be much harder to solve because the tissue where the action is may be somewhere far upstream from the tissue producing the voltage we record at the scalp. The same point can be made about other imaging methods.

The importance of not confusing those two senses of *source* is illustrated by work in the 1980s that encouraged the view that P300 is generated in the hippocampus. Initially, there was good circumstantial evidence for this view. But biophysical modeling by Lutzenberger and Elbert (1987) suggested that scalp-recorded P300 could not be generated as deeply as the hippocampus and more likely comes from a broad layer of superficial cortex. Empirically, P300 was shown to be intact after surgical removal of the hippocampus. As a final nail, the latency of P300-like activity recorded directly from the hippocampus tends to be later than the latency of scalp-recorded P300 (for review, see Johnson, 1989). It thus became clear that the hippocampus could not possibly be *the* source of P300, at least in the first sense of *source* described above. If anything, it is more logical to draw the causal arrow the other way, to suggest that P300 and the psychological processes it reflects are driving the hippocampus, although I hesitate to propose that psychological processes underlie hippocampal activity. But the really interesting story may come from determining what tissue is driving both superficial cortex and the hippocampus. That tissue may be where the biological action is. The site of the measure is not necessarily the site of the action.

Where the intervention should be. We can extend this issue of where the action is to involve how to change the action. For trying to prevent or remediate psychopathology, the worst consequence of the biology versus psychology war is the assumption that dysfunctions conceived biologically warrant interventions conceived biologically and similarly for dysfunctions and interventions conceived psychologically. This assumption is rampant in the popular press and common in prominent scholarly works, but it is groundless.

Assume for a moment the validity of the dopamine theory of schizophrenia as a theory about biochemistry in schizophrenia. Although the theory views as crucial that there is excess dopamine activity in schizophrenia, it is important to see that it does not follow from that theory that one must treat individuals with a diagnosis of schizophrenia with drugs that alter the dopamine system. Blocking dopamine receptors certainly sounds like a reasonable thing to try, but there are two caveats. First, the goal is not to alter the dopamine system but to alter schizophrenia. Even if dopamine is crucially problematic in schizophrenia, dopamine may not be the best system in which to intervene. There are probably a wide variety of disrupted systems in schizophrenia. Given that the systems are interdependent, the

best way to alter one system may be a direct intervention in another system.

The second caveat is that, even if the dopamine system turns out to be the best place to intervene in schizophrenia, it does not follow that a direct biological intervention in that system is optimal. We each have ample experience with events that we construe as psychological emptying our adrenal glands and altering our physiology by dumping a jolt of catecholamines into our bloodstream. There may be psychological interventions that will alter the dopamine dysfunction in schizophrenia more effectively or with fewer side effects than medications aimed directly at that system (for a similar view, see Robinson, 1995).

For example, as with expressed emotion, the best way to manipulate dopamine receptors may be to modulate the individual's exposure to the expression of hostility from significant others. In a similar vein, Hollon (1995) recently discussed how negative life events, generally construed psychologically, may alter biological factors in risk for depression. Effective pharmacological and cognitive behavioral treatments for obsessive-compulsive disorder are both accompanied by normalized metabolic rate in the caudate nucleus (for review, see Kozak & Foa, in press). There is a substantial literature on psychological effects on the immune system (e.g., Andersen, Kiecolt-Glaser, & Glaser, 1994; Cacioppo, 1994) and a growing literature on psychological factors controlling gene expression (Holden, 1991b; Johnson, 1990).

One need not choose between exclusively biological and exclusively psychological accounts of schizophrenia. We should not assume that a disorder with some documented biological anomalies is best treated by what we conceive of as biological interventions, and the converse is true for disorders we conceive of psychologically. The present position is sympathetic to calls for viewing psychological and biological realms (whether realms of phenomena or realms of levels of explanation of phenomena) as distinct and interacting (e.g., Zuckerman, 1995). However, the present position goes beyond such a view by arguing against such a distinction, especially if construed as a competitive or hierarchical distinction in which one realm underlies another in any causal or reductionistic sense. *Underlie* is just not the right way to frame the relationship between biology and psychology. Similarly, some well-entrenched traditions distinguishing cognition and emotion are ill founded and detrimental to our field. Psychological and biological domains can be viewed as logically distinct but not physically distinct and hence neither dualistic nor interacting. Psychological concepts and biological concepts are not merely different languages for the same phenomena (and thus not reducible in either direction). Psychological and biological explanations are not explanations of the same things. We can avoid turf battles by recognizing that the relationship between the psychological and the biological is fundamentally logical, not empirical (e.g., working out enough of the biology will not make psychology obsolete). We can avoid dualism by avoiding interactionism (having two distinct domains in a position to interact sounds like having separate realities). As cognitive neuroscientists, we are intrigued by brain tissue in part because of the psychological functions it implements. If we view brain tissue as implementing psychological functions, we will need the expertise of cognitive science to characterize those functions and the expertise of neuroscience to study the biological mechanisms that implement them. Neither of those disciplines encompasses, reduces, or underlies the other.

I hope to leave you wondering if I am right about these issues. I do not expect to convince you outright. If it were that easy, we would have resolved these issues long ago. These are tough problems, and I hope to at least make you painfully aware of how often we overlook them, stumble over them, and inadvertently foster them.

Psychophysiologists are less confidently wrong headed about these issues than many people in adjacent disciplines. The nature of our training and our work encourages us to respect and integrate biology and psychology rather than to favor one over the other. I hope we can do the same for cognition and emotion and that we can bring to bear all that we know to improve clinical intervention.

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