



## Self-Regulation and Depression

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*This article describes an emerging perspective on depression as a disorder of self-regulation. It is proposed that the concept of brain/behavior system, though insufficient for explaining depression, can be reformulated as self/brain/behavior system to address both psychological and neurophysiological aspects of depression. A set of hypotheses conceptualizing depression (in self/brain/behavior system terms) as a disorder of self-regulation is offered, and evidence in support of those predictions is summarized. Implications of the self-regulation model are discussed and potential advantages of a self-regulation perspective on depression are suggested.*

Why do people become depressed? In some respects we know a great deal about depression. The probability of suffering a major depressive episode is influenced by risk factors such as gender, parental loss, pathogenic rearing practices, personality dynamics, a history of traumatic experiences, previous episodes of depression, low social support, recent stressful events, and genetic influences (Kendler, Kessler, Neale, Heath, & Eaves, 1993). However, as yet there is no comprehensive, empirically validated model of vulnerability to depression.

To the afflicted individual, depression often is experienced as an “injury to the self” following loss, disappointment, or failure (Whybrow, 1997). But how could injury to the self induce dysphoric and anxious symptoms, alterations in behavior, cognition, and physiology, and increased vulnerability to subsequent episodes? The premise of this article is that we must broaden our perspective to view depression as a disorder of *self-regulation*. I begin by discussing how the concept of brain/behavior system, though insufficient to account for depression, becomes a viable framework for conceptualizing depression when expanded into the broader concept of self/brain/behavior system for self-regulation. Next, I present hypotheses concerning depression as a catastrophic response of this system to failure of self-regulation. Finally, some predictions, implications, and advantages of a self-regulation model are discussed.

### Self/Brain/Behavior Systems

Many theories invoke the concept of *brain/behavior system* to account for functional associations among classes of stimuli, patterns of activity in the central nervous system, and affective as well as behavioral outcomes. Emotion and motivation theorists postulate two fundamental brain/behavior systems, for approach and avoidance respectively, each of which is characterized by particular stimulus

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sensitivities, motivational tendencies, and affective states. These hypothetical systems originated in the animal literature but have been adapted as models of human motivation and affect (Watson, Weise, Vaidya, & Tellegen, 1999). Yet limitations of these models have been noted; for instance, the brain regions involved in representation and processing of the abstract or symbolic rewards that have motivational significance to humans are substantially more complex than those mediating responses to rewards in animals, suggesting the need for a broader systems perspective (Zinbarg & Mohlman, 1998).

Theories of depression also have incorporated the brain/behavior system concept. These models characterize depression as a dysfunction of the approach system with resulting deficits in positive affect and incentive motivation. This view is supported by studies of brain lesions, cortical activation patterns, neurotransmitter systems, pharmacologic challenges, and the efficacy of biological treatments for depression (Davidson & Henriques, 2000).

Although the brain/behavior system construct has had a major impact on theories of motivation and emotion, it does not provide a sufficient explanation for depression (Tomarken & Keener, 1998). In particular, it is silent regarding the role of self-regulatory cognition in vulnerability to depression, and so cannot provide a complete explanation of why certain people become depressed in certain circumstances (Strauman, 1999). To account for vulnerability to depression, which for initial episodes are most likely to occur in response to perceived failures or losses (Brown, 1998), brain/behavior systems must be reformulated to include the self. Given the critiques summarized above, we propose that the brain/behavior systems for pursuit of approach and avoidance goals are more accurately conceptualized in humans as self/brain/behavior systems, hypothetical constructs encompassing the psychological processes and biological structures that enable the pursuit of self-regulatory goals.

The theoretical implications of expanding the brain/behavior system concept to incorporate self-regulatory cognition are profound. For example, consider the idea that human psychological capabilities evolved in response to an increasingly complex social environment. One likely evolutionary pressure would be for motivational systems to acquire the capacity to monitor the individual's status in reference to others upon whom he or she depends for survival (Posner & Rothbart, 2000). In developmental terms, children must learn to behave in ways that maintain the nurturance and security they require from caregivers (Kochanska, 1995). If the approach and avoidance systems operate in service of maximizing positive outcomes and minimizing negative outcomes respectively, then to be effective those systems must be capable of self-representation and self-evaluation within the social context, and the representational, monitoring, and evaluative functions must be integral parts of each system.

Furthermore, if self-regulatory cognition is integral to the approach and avoidance systems, then the systems must be fundamentally different in human than in nonhuman animals. For instance, most animals have a restricted repertoire of approach goals and a limited set of strategies for achieving those goals; for such organisms, it is accurate to describe the positive-outcome motivational system as an "approach" system. In contrast, the kinds of positive outcomes that humans pursue can be as mundane as sating one's hunger or as abstract as achieving world peace. For many of the goals that people pursue, there is no literal or spatiotemporal "moving toward"; rather, the strategies people use instantiate a different sense of approach, namely, "setting about." Higgins (1997) noted these differences when he

proposed the terms *promotion* and *prevention* for the approach and avoidance systems respectively.

Another implication of the self/brain/behavior system construct is that the psychological processes within each system develop and operate *in relation to each other* rather than in isolation. Emotion, motivation, and cognition each reflect the individual's ongoing appraisal of, and response to, progress toward important goals—that is, the goal-oriented activity of the system as a whole. Such a hypothetical system operates at three levels in the pursuit of the individual's goals (Matthews, Derryberry, & Siegle, 2000). The *neural* level refers to the operation of CNS structures which support motivation, emotion, and self-regulatory cognition; the *cognitive* level refers to information processing events that are supported by the CNS; and the *knowledge* level refers to the individual's goals, intentions, and beliefs about self in relation to the social world.

Conversely, if self-regulation is coordinated by the promotion and prevention systems, then the social cognition of self-regulation needs to be studied in relation to the system as a whole (and vice versa). Table 1 lists proposed components of each hypothetical system, though it should be noted that every instance of self-regulation need not involve every component.

Expanding the approach and avoidance systems into self/brain/behavior systems for self-regulation permits generation of novel hypotheses regarding how various psychological and biological processes are functionally associated—both under normal circumstances and as the systems break down. This perspective allows us to study depression within a framework that acknowledges the complex processes contributing to the onset and maintenance of the disorder.

### **Depression as a Disorder of Self-Regulation**

Akiskal and McKinney (1973), in a classic article that foreshadowed contemporary efforts at building integrative models of depression, proposed that depression was a functional disorder that represented a “final common pathway” resulting from a range of distal contributory causal factors. In their view, the core of depression was the loss of motivation to respond effectively to cues for reward, a dysfunction manifested throughout the organism—in neurotransmitter systems, cognition, and behavior. Although acknowledging that vulnerability to depression was partly genetic and that repeated episodes of depression could cause permanent changes in the CNS, Akiskal and McKinney (1973) argued that depression began as an organism-wide response to loss or failure. Since their proposal, much evidence has appeared consistent with the view that depression is a situationally-triggered dysfunction of approach motivation.

Although self-regulation was not a dominant concept in behavioral science at the time of Akiskal and McKinney's article, their model is compatible with the self/brain/behavior system construct. If approach and avoidance motivation are manifestations of two regulatory systems, then there is reason to hypothesize that depression arises from malfunction in one or both systems. Furthermore, their view of the core of depression—loss of the motivation to and/or capacity to respond effectively to cues for reward—can be translated directly as *failure of self-regulation within the promotion system*. Current knowledge concerning the etiology and treatment of depression is consistent with such a view, but only if the approach and avoidance systems are reconceptualized in self/brain/behavior system terms.

**TABLE 1** Components of the Two Hypothetical Self-Regulatory Systems**Promotion System:**

- *CNS substrates:* Left prefrontal and frontal cortex, ascending dopaminergic system
- *Motivational impetus:* Maximizing positive outcomes
- *Personality/temperament correlates:* Extraversion, openness
- *Parenting/socialization antecedents:* Nurturance, encouragement
- *Self-evaluation:* Strong ideal standards (high accessibility, high commitment, high coherence)
- *Strategic orientation:* Approach; insure hits, insure against errors of commission
- *Interpersonal goals:* Agency and communion
- *Situational triggers:* Gain/non-gain
- *Affective states:* Cheerfulness (when goal attained), dejection (when goal not attained), eagerness (when pursuing goal)

**Prevention System:**

- *CNS substrates:* Right frontal cortex, septohippocampal system and associated neocortical structures
- *Motivational impetus:* Minimizing negative outcomes
- *Personality/temperament correlates:* Neuroticism, constraint
- *Parenting/socialization antecedents:* Punishment, control
- *Self-evaluation:* Strong ought standards (high accessibility, high commitment, high coherence)
- *Strategic orientation:* Avoidance; insure correct rejections, insure against errors of commission
- *Interpersonal goals:* Security and responsibility
- *Situational triggers:* Loss/non-loss
- *Affective states:* Quiescence (when goal attained), agitation (when goal not attained), vigilance (when pursuing goal)

A model of depression as a disorder of self-regulation suggests hypotheses such as the following:

1. *Depression results from cumulative failure of the promotion system.* Depression is not a brain disorder, or a cognitive disorder, or a behavioral disorder; it is a system disorder which is manifested throughout all levels of the promotion system and beyond. To understand depression, it must be studied within the context of how the two regulatory systems operate.
2. *An initial episode of depression is a functional disorder of the promotion system resulting from failure of self-regulation.* Such self-regulatory dysfunction may result from a single experience or an accumulation of experiences. The most likely scenario is that depression results from a “downward spiral” of failure to make progress toward promotion goals, with increasingly more severe and prolonged physiological, cognitive, and interpersonal consequences. Beyond a

certain level of cumulative failure, the system can no longer function normally and a new state of the promotion system, manifested as a depressive episode, ensues. Note that by “functional disorder” we mean that although the promotion system is responding maladaptively, there is (as yet) no structural change in the system itself.

3. *Core symptoms of depression reflect dysregulation within the promotion system* (e.g., mood, appetite, anhedonia, energy, concentration, worthlessness, hopelessness, low self-esteem) or *dysregulation of reciprocal inhibition between the promotion and prevention systems* (e.g., sleep disturbance, guilt, agitation/anxiety, HPA axis dysfunction). The approach and avoidance systems normally operate in a reciprocally inhibitory manner but this pattern breaks down in depression (Watson et al., 1999); this translates in our model to a combination of promotion system hypoactivity and prevention system hyperactivity.
4. *Depression alters the two regulatory systems*. This hypothesis is intended to account both for increased subsequent vulnerability to depression and for the observation that the disorder itself appears to change over subsequent episodes. That is, irreversible changes can occur within any level of the systems, which in turn increases risk for subsequent episodes. The more episodes, the greater the risk for irreversible change within each system.

A number of studies have produced data consistent with these hypotheses. For example, we have observed that covertly exposing people to their own promotion or prevention goals reliably induces motivational and affective states consistent with the individual's perception of whether or not they were meeting the goal—suggesting that each system is characterized by continuous automatic evaluation of progress toward such goals (Carver & Scheier, 1990). Similar but more pronounced responses to priming with self-regulatory goals has been observed in clinically depressed and anxious individuals (Strauman, 1989). Other priming studies have shown that activating self-regulatory cognition alters physiological responses hypothetically associated with the promotion and prevention systems. Our current research examines the impact of existing treatments for depression on dysfunctions in self-regulation and the development of novel treatment strategies from a self-regulation framework (Strauman et al., 2001).

There are a number of potential advantages of a self-regulation perspective on depression. First, existing theories of depression are translatable into self-regulation terms. This is critical because in order to be sufficiently comprehensive a theory of depression must both account for existing findings and make novel predictions. So, for example, hopelessness, the core feature of Abramson, Metalsky, and Alloy's (1989) model, can be conceptualized as the acute state of breakdown in the promotion system that signals the onset of a depressive episode. Second, a self-regulation perspective can account for correlations across different components of the hypothesized regulatory systems, such as associated changes in hedonic capacity, self-representation, approach motivation, interpersonal effectiveness, and HPA axis function following an experience of failure. At present no model of depression uses such associations to make predictions about regulatory system function in non-depressed and depressed individuals. Third, expanding brain/behavior systems by incorporating self-regulatory cognition addresses the concern that the experience of loss or failure (and its affective consequences) is frequently in the eye of the beholder and therefore only predictable based on the goals and beliefs of a particular individual.

A self-regulation perspective also provides for novel predictions concerning the etiology and treatment of depression. The following are examples of topics for research derived from the basic predictions of the model.

- *Onset of depression:* Because depression is a system disorder involving dysfunction of the promotion system, the onset of the disorder will manifest the characteristics of nonlinear or catastrophic changes in self-regulating systems. These changes will be detectable by comparing real-time sequences of neural function, information processing, and experience between nondepressed and depressed individuals.
- *Genetic vulnerability:* If depression results from failure of self-regulation and vulnerability to depression is partly genetic, what vulnerability factors are “inherited”? This perspective predicts a more subtle and complex pattern of heritability than many contemporary models have hypothesized. Specifically, we predict that genetic factors that increase vulnerability to depression do so via their impact on the *resilience of the system to self-regulatory failure*. Individual differences in the intensity and/or duration of dysphoric affect or in sensitivity to cues for loss/failure increase vulnerability to depression because of the acute and chronic “strain” placed on the promotion system.
- *Treatment matching:* In order to improve the effectiveness of treatments for depression, additional knowledge is needed regarding how different treatment modalities work. The self-regulation perspective provides an easily operationalizable framework for studying treatment mechanisms of action modeled on the three levels of self/brain/behavior systems (neural, cognitive, goal). System-based functional assessment can improve our capacity to select the most efficacious treatments for a particular individual. In addition, comprehensive treatment programs for recurrent depression can be designed to take into account the impact of repeated episodes on the two self-regulatory systems.
- *Risk for relapse:* Similarly, this perspective on depression suggests that episode-induced changes in the operation of the promotion system at any level can increase the likelihood of subsequent episodes (as well as reducing the magnitude of “stress” required to trigger a system breakdown). However, in order to improve our ability to predict risk for relapse or recurrence, we need to investigate how the two hypothetical systems “recover,” spontaneously or in response to treatment, from a depressive episode.
- *Computer simulation:* A particularly exciting prospect for applying this perspective to the study of depression is the opportunity to develop neural network models of the promotion and prevention systems under normal conditions and then “perturb” them to see whether the computer model can indeed simulate or predict differences in the two systems between normals and depressives. In addition, by extending connectionist modeling to capture the developmental processes that lead to the initial emergence of the two systems, it may be possible to identify early pathways of vulnerability and translate those findings into community-based primary prevention.

Depression poses an enormous public health burden that is increasing in prevalence. A problem of this magnitude deserves the fullest possible commitment of scientific resources to remediate, and ultimately prevent, such a devastating disorder. It is my hope that research guided by the self-regulation perspective will make a significant contribution to this effort.

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