Self-Regulation and Psychopathology: Toward an Integrative Translational Research Paradigm

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Abstract

This article presents a general framework in which different manifestations of psychopathology can be conceptualized as dysfunctions in one or more mechanisms of self-regulation, defined as the ongoing process of managing personal goal pursuit in the face of internal, interpersonal, and environmental forces that would derail it. The framework is based on the assertion that self-regulation is a critical locus for the proximal influence on motivation, cognition, emotion, and behavior of more distal factors such as genetics, temperament, socialization history, and neurophysiology. Psychological theories of self-regulation are ideal platforms from which to integrate the study of self-regulation both within and across traditional disciplines. This article has two related goals: to elucidate how the construct of self-regulation provides a unique conceptual platform for the study of psychopathology and to illustrate that platform by presenting our research on depression as an example.

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INTRODUCTION

Psychopathology Through the Lens of Self-Regulation

The nature and consequences of human behavior cannot be fully understood without taking into account the many ways in which people try to regulate their own thoughts, emotions, and behaviors in the service of becoming a particular kind of person (Allport 1955). All organisms have multiple mechanisms for behavior regulation, but the complexity of those mechanisms and their overall organization is most evident in humans. Within the discipline of psychology, the term self-regulation frequently is used to denote the processes by which people initiate, maintain, and control their own thoughts, behaviors, or emotions, with the intention of producing a desired outcome or avoiding an undesired outcome (Carver & Scheier 1990, Karoly 1993). Self-regulatory processes can be identified at both psychological and neural levels and can be automatic as well as intentional. Self-regulation has been shown to be both reflexive/mechanistic and intentional/agentic (Bandura 2001), which is consistent with the complexity of its component mechanisms. It is precisely this complexity that makes self-regulation research so challenging and also so important to our understanding of adaptive as well as maladaptive functioning within the social world.

Human behavior shares much of the genomic, neural, and physiological bases of animal behavior. However, human beings also purposefully regulate behaviors, thoughts, and emotions to achieve desired goals, to bring behavior in line with internal or external standards, and to function effectively in social interactions (Posner & Rothbart 2000). It appears that these capabilities evolved to allow us to deal with challenges and opportunities that arise in our physical and social environments, but also they render us vulnerable to a range of significant health problems, including psychological disorders such as depression and anxiety (Leary & Buttermore 2003).

Although humans have a remarkable capacity for effective self-regulation, there is no shortage of evidence that we are easily knocked off track when it comes to being the kind of person we desire to be. Self-regulatory dysfunction is implicated when people engage in self-harm, overeat, fail to control their temper, succumb to ethical lapses, procrastinate, and underachieve.

Self-regulation failure is at the core of health problems such as obesity, eating disorders, cardiovascular disease, smoking, and substance abuse (Ryan et al. 1997). Problems in self-regulation can arise in connection with people's efforts to achieve important goals, eat healthfully, exercise, practice safe sex, follow medical regimens, and drive safely (Bonin et al. 2000, Polivy et al. 1994, Sayette et al. 2001). Evidence also suggests that domestic violence, child abuse, and sexual assault arise in part from the inability to effectively regulate goal-directed behavior in the face of competing urges. In each case, dysfunction of self-regulation can create and maintain dysfunction within other regulatory mechanisms, and vice versa (Barkley 2001).

This article presents a general framework in which specific manifestations of psychopathology can be conceptualized as dysfunctions in one or more mechanisms of self-regulation. The framework is based on the postulate that self-regulation is a critical locus for the proximal influence on motivation, cognition, emotion, and behavior on more distal factors such as temperament, socialization history, physiology, and the physical and social environments (Karoly 1999, Strauman 2002). Furthermore, I propose that social psychological theories of self-regulation are ideal platforms from which to integrate the study of self-regulation within and across traditional disciplines. This article has two related goals: to elucidate how the construct of self-regulation provides a unique conceptual platform for the study of psychopathology and to illustrate that platform by presenting a summary of our own research on depression as an example.

One of the defining characteristics of human behavior is that it is organized and motivated by goals, which are mental representations of desired end states (Miller et al. 1960). Goal-directed behavior is embodied throughout the central nervous system (CNS), and the CNS itself has evolved to support multiple levels of behavioral organization, which facilitate goal pursuit within a complex social world (see Miller 2010 for a thoughtful discussion of the relation between neurobiological and psychological levels of analysis, which is beyond the scope of this review). Austin & Vancouver (1996) identified approach and avoidance goals as among the most important classes of goals for both organization and motivation of social behavior. Goal-directed behavior is instantiated within the mammalian brain largely as two brain/behavior systems for approach and avoidance, known as the behavioral activation system (BAS) and the inhibition system (BIS). The BAS and BIS underlie the temperament BAS-based, spatiotemporal approach and avoidance as well as dispositional positive and negative affectivity (Carver & Scheier 1990, Watson et al. 1999). The neuroanatomical structure and function of the BAS and BIS have been widely explored for both normal behavior and psychopathology (Fowles 1980). Likewise, the influence of the BAS and BIS on social behavior has been clearly documented (Watson et al. 1999). Individual differences in the BAS and BIS strength and sensitivity to relevant cues are well-documented, but all individuals are presumed to possess the same basic circuitry and networks for the BAS-mediated approach and BIS-mediated avoidance and to respond to the environmental cues for which the BAS/BIS evolved.

Human goal pursuit also has been explored from a social-cognitive perspective. In social psychology, the term self-regulation frequently is used to describe the ongoing process of managing personal goal pursuit in the face of internal, interpersonal, and environmental forces that would derail it (Hoyle & Gallagher 2015). Social-cognitive theories of self-regulation mirror the binary approach/avoidance conceptualization of animal models, but incorporate our advanced abilities for planning, strategy, and abstraction. Such theories typically emphasize higher-order goals that are cross-situational and often are integrated within the individual's identity and sense of self [e.g., James 1948 (1890), Freud 1961 (1923), Rogers 1961)]. People characteristically strive to attain their personal goals and standards—to become the kind of person that they see as desirable, their "ideals," and as obligatory, their "oughts" (Higgins et al. 1986). In their ongoing pursuit of personal goals, people continuously compare their actual behaviors with their representations of the kind of person they are striving to become (Carver 1998, Gollwitzer 1999). In turn, this ongoing

self-evaluation has significant repercussions for the individual's emotional and motivational state and ultimately for both well-being and social adaptation (Karoly 1993). Social-cognitive mechanisms for self-regulation are presumed to emerge largely from early socialization experiences, particularly from contingent interactions with parents, although temperament plays a moderating role. These social cognitive models, orthogonal to the BAS and BIS, suggest numerous combinations of dispositional and social-cognitive tendencies to respond preferentially to reward versus threat cues (Strauman & Wilson 2010).

Although a robust literature links the BAS and BIS with vulnerability to internalizing disorders (e.g., Griffith et al. 2010), research and theorizing regarding the role of social-cognitive processes of self-regulation in psychopathology also have been thriving. The present article draws valid and practical generalizations from the self-regulation literature, primarily on the basis of social-cognitive approaches. In that regard, it is important to note at the outset that the study of self-regulation has substantial overlap with other basic science and clinical science constructs relevant to well-being and psychopathology. For example, emotion regulation has been defined as the processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions (Gross 1998). One could easily conceptualize self-regulation as relevant to each of those specific processes, and vice versa. Therefore, although researchers inevitably break down cycles of behavioral adaptation into more manageable components, we must remember that the boundaries between constructs are permeable and constantly shifting.

The challenge is substantial to create truly comprehensive, multidisciplinary self-regulation-based theories of vulnerability to psychiatric disorders. The self-regulation of behavior involves genetic, physiological, cognitive, motivational, emotional, and social systems (Tucker & Williamson 1984). Factors operating at each of these levels can elicit, facilitate, or impede people's efforts to attain their goals, and of course those different factors and processes influence one another as well. Nonetheless, the construct of self-regulation represents a valuable organizing principle for theory and research, one that is ideally suited for this challenge (Carver & Scheier 1990). Self-regulation occupies a unique place in biological and psychological functioning, one that makes it especially appropriate for investigating how the mind and the body interact. That is, self-regulation involves many of the biological and psychological processes most important for adaptive social functioning.

Self-Regulation Theory as a Shared Language

Heatherton (2011) offered a cogent psychological assessment of the importance of the self-regulation construct for the study of the brain and behavior. As a social species, humans have a fundamental need to belong that encourages behaviors consistent with being a good group member (which has obvious survival value). In turn, being a good group member requires the capacity for self-regulation, which allows people to (intentionally or automatically) alter or inhibit behaviors that would place them at risk for exclusion and other negative interpersonal consequences. Heatherton (2011) also proposed that self-regulation requires four psychological components, paraphrased as follows: (*a*) People need to be aware of their behavior and its consequences, particularly in reference to salient norms, standards, and goals; (*b*) people need to understand how others are reacting to their behavior and how others' reactions can influence them in turn; (*c*) people need to detect and anticipate negative outcomes, especially in complex social situations; and (*d*) people need to resolve discrepancies between their actual behavior and their desired (or undesired) outcomes. From this perspective, the self-regulation construct is relevant to a broad range of psychiatric disorders: schizophrenia (Fortune et al. 2011), autism (Bachevalier & Loveland 2006), and psychopathy (Koenigs et al. 2011), among others. Although this article focuses on

unipolar depression as an example of the utility of a self-regulation perspective on psychopathology, it should be acknowledged that the perspective summarized by Heatherton applies equally to other disorders. This perspective aligns with the Research Domain Criteria developed by the National Institute of Mental Health as a transdiagnostic framework for psychopathology research (Cuthbert & Insel 2013).

Although the term self-regulation has only recently become a unifying concept in psychopathology research, numerous theories and models have addressed one or more of the psychological components postulated by Heatherton. Over the past several decades, growing interest in the role of cognition in emotional disorders produced an abundance of research (Coyne & Gotlib 1983). Three major issues confronted social-cognitive research on anxiety and depression (Strauman 1992): (*a*) whether cognitive processes are implicated in the onset or maintenance of full clinical syndromes and are not simply normal sadness or anxiousness, (*b*) whether cognitions represent contributory causal factors in emotional disorders and are not simply consequences or epiphenomena of such disorders, and (*c*) whether specific cognitive processes are associated with particular disorders. The value of self-regulation theories of psychopathology depends in part on their predictive specificity, identifying discriminant associations among particular dysfunctions and specific disorders or phenotypes.

Many psychological theories of emotional disorders have postulated that self-evaluation processes were associated with vulnerability to distress. For example, clinical and experimental data documented the relation between low self-esteem and chronic negative affect (e.g., Wylie 1979). Theorists as diverse as James [1948 (1890)], Freud [1961 (1923)], and Rogers (1961) described how within-self inconsistencies can produce discomfort. Bandura's (2001) pioneering work on self-efficacy helped to highlight the critical role of self-evaluation in basic motivational and interpersonal processes and in the onset and maintenance of psychiatric disorders. Likewise, Mischel's (2004) cognitive/affective model of personality was formulated both as a general model of social cognitive processes and as a means by which to conceptualize emotional vulnerability. And attachment theory (e.g., Bowlby 1969), although not originally intended as a model for self-regulation of behavior and affect, nonetheless has had an enormous influence on psychological theories of emotional disorders.

Admittedly, the notion that self-regulation offers a common framework for psychopathology research, particularly (but not exclusively) in the domain of internalizing disorders, is itself a retrospective construction. Nonetheless, my colleagues and I have made this proposal previously (e.g., Strauman & Merrill 2004), and ongoing developments in research on depression and other disorders have supported our assertions regarding the utility of a self-regulation perspective. I will simply suggest that there is both conceptual and clinical value in applying this perspective to the diagnosis, treatment, and prevention of psychopathology, and I support that assertion by reviewing some of our own research findings.

SELF-REGULATION AND PSYCHOPATHOLOGY

This section examines two core issues that arise from conceptualizing psychopathology in terms of disordered self-regulation. Specifically, the issues that deserve attention are (*a*) identifying and implementing relevant levels of analysis in applying a self-regulation perspective to psychopathology and (*b*) clarifying the antecedents, processes, and consequences of self-regulation (particularly in terms of personal goal pursuit) in the domains of interest. After considering those core issues, I propose some general parameters for a self-regulation model of psychopathology, before turning to a discussion of self-regulation in depression.

Levels of Analysis in Models of Self-Regulation

Although most scientists who study self-regulation have focused their research primarily at a single (i.e., psychological) level of analysis, human self-regulation occurs simultaneously at multiple interacting levels. Thus, a range of regulatory factors must be considered, including genetic/genomic processes; neurophysiology (particular executive processes and emotional/motivational processes); psychological processes (cognitive, motivational, and emotional); and interpersonal, social, and cultural factors that elicit, facilitate, or impede people's efforts to self-regulate. From a psychopathology standpoint, it is important to recognize that the psychological processes by which self-regulation is made possible are themselves linked bidirectionally with physiological and social processes that are critical to the disorders of interest.

Theories of self-regulation are among the most influential and most extensively validated models in behavioral science. Although different theories make unique predictions, all intend to explain how perceived progress, or lack of progress, toward goals influences motivation, cognition, affect, and behavior (Carver 2004). The current interest in self-regulation among psychologists stemmed largely from work on delay-of-gratification (Mischel 1966, Mischel & Moore 1973) and from discrepancy-reduction and cybernetic models of self-evaluation (Carver & Scheier 1981, Higgins 1987), which themselves were derived from the classic work of Miller et al. (1960), Duval & Wicklund (1972), and others. Self-regulation research has examined the nature of internal guides to behavior (e.g., goals, values, life tasks, personal strivings), factors that enhance and diminish people's ability to self-regulate, the automaticity of regulatory processes, strategies for behavioral control, and individual differences in the capacity to self-regulate.

Although the psychological mechanisms for self-regulation operate within the head of the individual, so to speak, psychologists have long recognized that people's capacity for self-regulation is tied to the social context in which they live. For example, our views regarding what kind of person we ought to be and which behavioral impulses must be controlled (as well as how to control them) are strongly determined by social factors, and motivation to regulate behavior is often facilitated by a desire to conform to other people's standards for us (Higgins et al. 1987). People often enlist others to assist in their self-regulatory efforts, and belonging to groups composed of individuals who are struggling with similar self-regulation failures (such as AA or domestic violence groups) can be of substantial benefit. Even when others are not present, people often imagine potential reactions of friends, family members, or coworkers as they try to control undesired impulses [Cooley 1964 (1902), Mead 1934].

Cognitive and behavioral neuroscientists have been engaged in also studying mechanisms for self-regulation, typically focusing on neural processes that underlie so-called executive functions, which are critical for successful self-regulation (Lewis & Todd 2007). Processes such as selective attention, attentional switching, sustained attention, memory, emotional control, choice, self-reflection, and decision making all have been implicated in goal pursuit and behavior regulation, intentional and automatic. Much of this work has confirmed the primary role of the prefrontal cortex (PFC) and frontal cortex in executive functions, particular in the context of goal pursuit (Stuss & Levine 2002). For instance, damage to the ventromedial-orbitofrontal cortex results in disinhibition, failure to consider long-term consequences of one's behavior, lapses in decision-making, an inability to behave in ways that one knows are morally correct, and an inability to use other people's social cues to regulate one's own behavior (Blair & Cipolotti 2000, Damasio et al. 1990). Furthermore, the PFC and frontal cortex are involved in representing goals, initiating action, and maintaining ongoing behavior, and these brain regions have been also implicated in addictions, mood disorders, and anxiety (Volkow & Fowler 2000). Other areas of the brain, including the anterior cingulate cortex, the hypothalamus, and the amygdala, are involved in regulatory

processes. Experimental data have been supplemented by case studies from neuropsychiatry that have examined patients who have lost specific self-regulatory capacities (e.g., Hart et al. 1999). Similarly, computational models of brain function have been developed to describe the dynamic neural basis of self-regulation (Cooper & Shallice 2000). Finally, a complementary approach to self-regulation can be found in the hybrid field of neuroeconomics (e.g., Glimcher & Kanwisher 2006), which seeks to understand how models of choice and economic behavior elucidate brain mechanisms of behavioral control.

Functional neuroimaging studies, in turn, implicate the importance of neurotransmitter systems in both voluntary and involuntary mechanisms of self-regulation (Depue & Collins 1999). For instance, it is well documented that the modulation of fast neurotransmission by monoamines is critically involved in numerous physiological functions and psychopathological conditions (Gainetdinov & Caron 2003). Transporters for neurotransmitters such as dopamine, serotonin, and norepinephrine are established targets of many psychoactive drugs, and data regarding the impact of cognitive and behavioral phenomena on neurotransmitter availability and transport are becoming available (e.g., Dunlop & Nemeroff 2007). Furthermore, emerging developmental research linking behavioral, functional neuroimaging, and neurophysiological measures of self-regulatory processes (e.g., Viding et al. 2006) offers intriguing glimpses of interactions among the processes and levels of dynamic organization underlying human self-regulation.

Perhaps the broadest biobehavioral perspective on self-regulation to emerge over the past two decades focuses on bidirectional pathways of influence between genomic processes and behavior (Moffitt et al. 2005). Although phenotypic differences among genetically identical organisms have long been observed, only recently have behavioral variables been examined systematically as potential consequences of, or explanations for, these differences. It has been hypothesized for a number of disorders that during specific phases of development, variations in environment or behavior can trigger or suppress gene expression. The proliferating number of examples in which behavioral self-regulation leads to epigenetic differences between monozygotic twins over the life course (e.g., Fraga et al. 2005) indicates that genomic processes must be included within a truly comprehensive science of self-regulation (Hariri et al. 2006), particularly with reference to psychopathology. For example, a relatively frequent single nucleotide polymorphism in the catechol-*O*-methyltransferase (*COMT*) gene has been shown to influence dopamine neurotransmission and, in turn, to have implications for cognitive, motivational, and affective processes regulated by the orbitofrontal cortex. In a now classic study, Drabant et al. (2006) examined the role of the *COMT* genotype as a predictor of individual differences in affect regulation and associated patterns of brain activation. Using a within-subject functional magnetic resonance imaging (fMRI) experimental design based on a perceptual task that involved matching angry and fearful facial expressions, Drabant et al. (2006) observed that heritable variation in dopamine neurotransmission associated with the *COMT* genotype impacted the functional reactivity of frontal and limbic circuitry implicated in the regulation of emotional arousal. In the section titled *COMT* Genotype and Regulatory Focus, I present data linking the *COMT* genotype with a self-regulation-based risk phenotype for depression.

Foundations, Mechanisms, and Outcomes

Following the self-regulation taxonomy proposed by Hoyle & Gallagher (2015), this section offers an organizing framework for conceptualizing psychopathological dysfunction in terms of self-regulation: (*a*) self-regulatory foundations, (*b*) self-regulatory mechanisms, and (*c*) goal pursuit outcomes and consequences. This framework also draws upon theorizing by Karoly (1999), who proposed a self-regulatory goal process perspective that integrated a broad range of psychological processes from goals to self-efficacy to individual differences in temperament.

Foundations of self-regulation. Self-regulation in pursuit of a goal is enabled by a set of mental structures and processes. Hoyle & Gallagher (2015) referred to these structures and processes as foundational because they are required at one or more points in the ongoing process of self-regulation. For that reason, a deficit or strength in any of them influences the degree to which the individual routinely struggles or succeeds at managing goal pursuit. Hoyle & Gallagher proposed three classes of foundational processes: executive function (Miyake et al. 2000), temperament (Rothbart & Bates 2006), and personality traits. Executive functions are of particular interest within the self-regulation/psychopathology interface, because they manifest as basic skills that allow for planning, decision making, error correction, and inhibition of prepotent responses. Research on schizophrenia emphasizes deficits and disruptions in executive function as both an endophenotype and a core feature of the disorder, but only recently has that body of research been linked with social-cognitive perspectives on self-regulation specifically (e.g., Orellana & Slachevsky 2013). Experimental psychopathology research has focused also on executive functions for depression (Snyder 2013) and anxiety (MacLeod & Mathews 2012).

Self-regulatory processes and mechanisms. In their taxonomy of self-regulation, Hoyle & Gallagher (2015) distinguished between goal pursuit across time and situation and goal pursuit that is temporally and situationally constrained, occurring whenever situational factors make the individual's goals salient. For present purposes, we focus on the mechanisms that operate both within and across situations. The Hoyle and Gallagher model describes an ongoing self-regulatory cycle on a momentary scale and a cross-time and cross-situational scale. Hoyle & Gallagher identified three rough phases of ongoing goal pursuit, beginning with forethought. During this phase, the individual sets goals and makes plans to achieve them via analyzing the task itself and the immediate or overarching context. Assuming sufficient planning, resources, and motivation, the process moves to the performance phase. This phase requires skills of self-control to stay engaged in goal-relevant behaviors. The performance phase also requires metacognition, the ability to monitor one's behavior and evaluate progress in relation to the goal. Eventually, the individual enters the third phase, self-reflection. Adaptive self-reflection (in contrast to depressive rumination, for example) involves an accurate appraisal of one's efforts as well as the extent to which those efforts were successful or unsuccessful. This three-phase model is particularly useful as a template for conceptualizing self-regulatory dysfunction in specific disorders, and each phase can involve both intentional/agentic and unintended/automatic aspects.

Goal pursuit outcomes and consequences. Hoyle & Gallagher (2015) identified a series of self-regulatory challenges and consequences that have significant implications for both immediate and long-term well-being. For example, goal pursuit initiation requires sufficient opportunity, motivation, and psychological resources and is critical to the overall self-regulation process. Likewise, once pursuit of a goal has begun, self-regulation involves the management of pursuit, which requires actions, such as persistence, maintenance of interest, and monitoring of effectiveness. Ultimately, effective goal pursuit may dictate that a particular task, or overarching goal, is either irrelevant or unattainable, calling for goal disengagement or substitution. Difficulty with initiation, maintenance or management, and resolution (especially disengagement) of goal pursuit behavior is a prominent feature of both DSM Axis I and II diagnostic categories (Ruscio et al. 2011). For example, perseverative behaviors are characteristic of a number of personality disorders. This seeming inability to respond adaptively to failure feedback leads to the frequently reported sense of bewilderment and frustration for both the suffering individual and their family and friends when maladaptive behavior patterns stubbornly persist.

Principles of Self-Regulation Applicable to Psychopathology

The brief historical overview provided at the beginning of this section clearly indicates that effective self-regulation is fundamental to mental health and well-being. Central to life's pleasures and pains is success or failure in pursuit of personal goals, including knowing when to keep what one is or has and when to make changes. Surprisingly, however, relatively few interventions for mood and anxiety disorders are based explicitly on the psychological principles that underlie self-regulation in general and approach/avoidance in particular. In turn, the application of self-regulation theory to the treatment and prevention of psychological disorders depends on well-elaborated and validated models of specific disorders or pathways to disorder. To determine whether and how psychological processes of self-regulation contribute to psychopathology, a general, flexible conceptual framework would be helpful. At the heart of such a framework is the question of dysfunction: What accounts for the transformation (either quantitative or qualitative, either abrupt or gradual) from effective to ineffective self-regulation, particularly insofar as such dysregulation has profound consequences for neurobiology, affect, motivation, and behavior? This section highlights some important conceptual challenges for self-regulation models of psychiatric disorders.

Antecedent, correlate, or consequence. The rise of cognitive psychology and cognitive neuroscience over the last 50 years triggered a reexamination of the nature of psychological causality among philosophers of science and behavioral scientists. These issues are particularly vexing for theories of psychopathology, which, in principle, can locate critical causal influences at any of a number of levels of analysis. As such, one question to be considered is, What do theories of self-regulation imply about the status of psychological mechanisms as contributory factors for psychopathology, particularly when such disorders are understood as neurobiological as well as psychological?

Investigators have been exploring integrative models in which self-regulatory processes both contribute to and are influenced by the onset of a disorder or episode (e.g., Karoly 1999, Little 2006). However, I am not aware of any truly comprehensive self-regulation-based model for any single disorder. Of course, such a model likely represents an ideal, which any particular theory should not be expected to attain. Nonetheless, without considering how psychological processes that underlie self-regulation influence, and are influenced by, processes at nearby levels, our understanding of psychopathology will be inherently limited. In particular, we will be vulnerable to simplistic explanations that elevate one level of analysis to primary causal status and relegate others to consequences or epiphenomena (Kendler 2005).

Self-regulatory failure: gradual/degenerative versus abrupt/catastrophic. Psychological theories of self-regulation, which were intended to model basic psychological processes, typically are silent on a particular issue of critical importance to the study of psychology: Exactly how does dysfunction of the mechanisms underlying self-regulation lead to a clinically significant disorder (here using depression as an example)? This question, of course, is a more specific version of the ubiquitous challenge for all theories of psychopathology—namely, the transformation from adaptive to maladaptive functioning. Logically, at least two distinct pathways linking self-regulatory failure with the onset of a depressive episode can be hypothesized: a gradual, degenerative course or an abrupt, catastrophic one. The degenerative course is consistent with a downward spiral of perceived failure in goal pursuit, culminating over time in a generalized state in which the perceived likelihood of a positive outcome is zero (Carver 1998). The catastrophic course has been conceptualized in association with major negative life events (Paykel 2003) but could likewise be

related to less traumatic events that signal failure in pursuit of a goal with which an individual's self-worth is too strongly identified or upon which their self-regulatory orientation is primarily based (Showers et al. 1998). Patients' experiences of the onset of depression have been described both ways (Blatt 2004), and the presumed heterogeneity of depression itself suggests that translation of self-regulation theories to account for the onset of depression will require explication of multiple sequences of contributory causal events and processes.

Moderating processes. Current theories of self-regulation provide robust and thoughtful accounts of how individuals pursue personal goals and the kinds of motivational and emotional states they experience when they see themselves as attaining, or failing to attain, such goals. However, as noted above, these theories themselves do not articulate comprehensively how the experience of acute emotional distress following failure to attain important goals becomes the chronic emotional distress characteristic of mood and anxiety disorders. Because only a subset of individuals who experience chronic difficulties with attaining goals go on to meet diagnostic criteria for major depressive episode or an equivalent disorder, there are likely to be other factors that determine whether a particular individual responds adaptively in the face of continued failure feedback or becomes mired in a downward spiral of negative self-evaluation, doubt, and distress.

One candidate for such a factor is maladaptive repetitive thought, also known as rumination. The tendency to engage in specific types of ruminative thought in response to goal blockage predicts the likelihood of self-regulatory failure (Carver & Scheier 1990, Pyszczynski & Greenberg 1987). Martin & Tesser (1996, p. 7; 1999) proposed a theory of rumination, which they defined as a "class of conscious thoughts that revolve around a common instrumental theme and that recur in the absence of immediate environmental demands requiring the thoughts." Martin & Tesser posited that rumination is instigated by a failure to make progress toward a desired goal and that attaining or disengaging from the blocked goal will terminate the ruminative process. However, redirecting thoughts away from the distressing content or reducing negative affect associated with the goal blockage is likely to only temporarily halt the process, because the continual cueing of goal-related thoughts by features of the social environment makes effective distraction difficult. An unfavorable assessment of the expectancy of succeeding at goal-directed action instigates rumination, especially if the blocked goal is central to the self (Carver 1998).

Nolen-Hoeksema et al. (1993) studied rumination as a coping style both contributing to and occurring in depression, and defined ruminative coping as "behaviors or thoughts that focus an individual's attention [on] the possible causes and consequences of that mood" (p. 20). In this account, rumination is not instigated by a self-regulatory failure but instead is a maladaptive response to dysphoric mood. Indeed, dysphoric individuals who ruminate demonstrate lower problem-solving and less ability to generate effective solutions to interpersonal problems when compared to nondysphorics and dysphorics who use distraction (Lyubomirsky et al. 1999).

As this cursory examination of issues underlying self-regulation models of psychopathology hopefully illustrates, substantial evidence favors a more integrative, multilevel perspective on self-regulation in psychological disorders. To the extent that emerging theories can address these issues—causal status, the role of failure experiences, and potential moderators—such models may provide valuable insights for diagnosis and treatment. I offer one final observation: Investigators in each field need to examine their assumptions (often implicit) regarding what causes psychopathology if truly integrative new models are to be realized. Kendler (2005) proposed a multilevel perspective on psychopathology that acknowledged the reality of both brain-to-mind and mind-to-brain causality in the pathogenesis of mental disorders. Moreover, Kendler called for adoption of explanatory pluralism, in which investigators focusing on particular levels of analysis are more forthright about the importance of complementary levels and are more ambitious in their

efforts to collaborate in cross-level studies. The study of self-regulation deserves a place at the table among contributors to a better understanding of psychopathology. I turn now to our own research on self-discrepancy and regulatory focus as contributory causal factors in depression as examples of the value of self-regulation theory and the challenges inherent in applying such theory to psychopathology.

Example: Self-Discrepancy Theory and Regulatory Focus Theory

The hedonic principle—that people approach pleasure and avoid pain—is one of the fundamental motivational assumptions within psychology. In spite of the wide applicability of this principle, however, its limitations have become apparent over the past several decades. The problem with the hedonic principle is not that it is wrong, but rather that its dominance has taken attention away from other principles that concern the different ways that people approach pleasure and avoid pain (Higgins 1997)—different ways that influence the emotional and motivational consequences of perceived success and failure in goal pursuit.

Self-discrepancy theory (SDT; Higgins 1987) was developed to conceptualize how problems in self-regulation of personal goal pursuit contribute to mood and anxiety disorders. SDT identified two types of personal goals or self-guides: hopes and aspirations (ideal self-guides) versus duties and obligations (ought self-guides). The theory predicted that when individuals failed to meet their ideals, they would suffer from dejection and dysphoria, whereas when individuals failed to meet their oughts, they would suffer from agitation and anxiety. According to SDT, what produces these different emotional syndromes are the different psychological situations that people experience depending on which type of self-guide they are using. When events are construed in reference to ideals (hopes and aspirations), people experience success as a gain and failure as a nongain. This gain/nongain construal triggers emotions such as happiness, joy, and satisfaction when we succeed and sadness, frustration, and disappointment when we fail. In contrast, when events are construed in reference to oughts (duties and obligations), people experience success as a nonloss and failure as a loss. This loss/nonloss construal triggers emotions such as calmness and quiescence when we succeed and worry, guilt, and anxiety when we fail.

SDT provided an integrative translational model that links self-regulatory cognition with the behavioral science literatures on motivation and emotion. In addition, SDT recognized that specific situations could influence whether a person's ideals or oughts were more accessible at that moment. Whichever type of self-guide was more accessible would determine whether that particular situation was construed in reference to the person's ideal or ought guides, which in turn would determine which affective experiences resulted. Evidence for such emotional variability across situations as a function of the accessibility of ideal and ought guides from contextual priming has been found in numerous studies (e.g., Strauman & Higgins 1987).

Regulatory focus theory (RFT; Higgins 1998) is a more general model of self-regulation that extended SDT by distinguishing between a promotion system that is concerned with nurturance, advancement, and fulfilling hopes (ideals) and a prevention system that is concerned with security, safety, and fulfilling duties (oughts). RFT emphasizes that promotion failure and prevention failure, along with their accompanying affective and motivational experiences, are psychological states. If either the promotion or prevention system were activated in any specific situation and a personally significant failure were to occur in that situation, then acute system-specific distress would occur: dejection and dysphoria in the case of promotion failure and agitation and anxiety in the case of prevention failure. In contrast to BAS/BIS, which operate as bottom-up systems in response to cues for spatiotemporal approach and avoidance (Depue & Collins 1999, Watson et al. 1999), the promotion and prevention systems are top-down socialization-based systems for

strategic approach (eager strategies) and avoidance (vigilant strategies) in response to activation of generalized goals or concerns (Scholer & Higgins 2010, Strauman & Wilson 2010). Functional neuroimaging studies provide evidence that these two sets of approach/avoidance systems have distinguishable neural activation correlates (Strauman et al. 2013).

As had been postulated originally in SDT, promotion and prevention goal failure are associated with specific affective and motivational consequences. Depression is associated with discrepancy between the actual and the ideal, which is a promotion system failure, whereas anxiety is associated with discrepancy between the actual and the ought, which is a prevention system failure (Strauman 1989, 1992; Strauman & Higgins 1987). But RFT makes additional predictions about the antecedents and consequences of personal goal pursuit. Promotion failure is experienced as the absence of a positive outcome (a nongain), whereas prevention failure is experienced as the presence of a negative outcome (a loss). Recent research on RFT has found that when the promotion system is active, what matters to individuals at that moment is to advance from a current status quo "0" to attain a better "+1" state. In contrast, when the prevention system is active, what matters to individuals at that moment is to maintain a safe status quo "0" and not fall to a worse "−1" state.

This mechanistic distinction is important because it clarifies the critical difference between an active promotion state versus an active prevention state in what makes unsuccessful goal pursuit distressing, i.e., what constitutes a failure. What is critical is not only the particular kind of personal goal that the individual is pursuing (e.g., ideal versus ought) but also the meaning of the individual's current state 0. In the prevention system, 0 is positive and it is moving below 0 that is a failure. In contrast, in the promotion system, remaining at 0 is a failure and moving from 0 to +1 is positive. The critical nature of this distinction is revealed by considering what happens when individuals construe themselves as being in a worse (−1) state compared to the status quo 0—which are the circumstances in which individuals with depressive or anxious symptoms regularly find themselves. Although being in a worse state is clearly negative within both systems, how to make things better presents a different challenge for promotion versus prevention. When individuals are in a prevention state, any behavioral option that gets back to the safe status quo 0 state is desirable, that is, the psychological mandate is to get back to 0. However, in a promotion state there is no value in simply getting back to 0 because it still constitutes a failure (Scholer & Higgins 2010). Thus, RFT suggests that to help people who are construing themselves as failing in personal goal pursuit requires creating different interventions for a prevention failure versus a promotion failure. Furthermore, individuals who experience both dysphoric and anxious symptoms are likely to be experiencing two different kinds of perceived failure at different times and, therefore, are likely to benefit from both kinds of interventions at different times—targeting promotion failure when they experience dysphoric symptoms and prevention failure when they experience anxious symptoms.

Recent research has elucidated the neural correlates of promotion and prevention goal pursuit (e.g., Cunningham et al. 2005, Touryan et al. 2007). Eddington et al. (2007) used incidental goal priming to examine neural activation associated with promotion and prevention goals via fMRI. They found that an area of the left PFC was activated during idiographic promotion goal priming, and the magnitude of activation in this left PFC region was correlated significantly with a self-report measure of individual differences in strength of orientation to promotion goals. The locus of activation was found in the region of PFC that is postulated to play a critical role in modulating emotional and motivational responses to goal-relevant stimuli (Ramnani & Owen 2004) and in integrating outcomes across separate cognitive operations in pursuit of abstract, higher-order goals (which has been associated with activation of the promotion system).

Strauman et al. (2013) used a different goal priming procedure to further elucidate the neural correlates of the promotion and prevention systems. When we applied a rapid masked exposure

technique, in which participants were exposed subliminally to their own promotion and prevention goals, we observed distinct patterns of neural activation associated with promotion versus prevention goals. Promotion priming led to activation in frontal and occipital regions and in the caudate and thalamus, whereas prevention priming was associated with activation in the precuneus and posterior cingulate cortex. Individual differences in chronic dysphoric/anxious affect and in regulatory focus, but not differences in the BAS/BIS strength, predicted differential activation following promotion versus prevention priming. The regions activated in response to promotion and prevention goals mapped broadly onto the cortical midline network (Northoff 2007) shown to index self-referential processing.

There is no implied claim here that SDT and RFT can provide sufficient, comprehensive models for diagnostic categories such as depression and anxiety as disorders of self-regulation. Nonetheless, from an experimental psychopathology perspective, the two related theories have substantial support for their predictions regarding the acute and chronic affective impact of goal pursuit feedback and for their implications regarding self-regulatory dysfunction in depression versus anxiety. Clearly, SDT/RFT is only one viable model for conceptualizing depression as a disorder of self-regulation. The next section focuses on applying principles of self-regulation more specifically to unipolar depression, returns to the SDT/RFT example, and then summarizes related models of self-regulation and depression.

SELF-REGULATION AND DEPRESSION

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 et al. 1993). However, as yet no comprehensive, empirically validated model of vulnerability to depression exists. 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? This section begins by highlighting models of depression that laid the groundwork for contemporary self-regulation approaches.

Precursors of Self-Regulation Models

The current literature on self-regulation dysfunction in depression draws heavily on a number of conceptual and empirical contributions. Several of the most influential are summarized here.

Depression as a final common pathway. Akiskal & 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 they acknowledged that vulnerability to depression was partly genetic and that repeated episodes of depression could cause permanent changes in the CNS, Akiskal & McKinney 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 at the time of Akiskal & McKinney's article, their model is compatible with current understandings of human self-regulation. Specifically, if approach and avoidance motivation are manifestations of self-regulation brain/behavior systems, then it is reasonable to hypothesize that depression arises from malfunction in one or both systems. Furthermore, their view of the core of depression—loss of the motivation or capacity to respond effectively to cues for reward—can be translated directly as failure of self-regulation within the promotion system (or, alternatively, the BAS). Many of the postulates from Akiskal & McKinney's model were limited by lack of knowledge about the cumulative effects of multiple episodes of depression and the field's inability to differentiate early-stage depression from late-stage melancholia, but nonetheless, the core predictions of their model appear to be robust and prescient.

Self-esteem and depression. Of all facets of psychology with potential relevance to depression, self-esteem has been the most extensively studied. Clinical, epidemiological, social, and personality researchers have long theorized that inadequate or fragile self-esteem can lead to (Roberts & Monroe 1994), exacerbate (Roberts & Gotlib 1997), and result from (Hayes et al. 2004) episodes of unipolar depression. Although the origins of self-esteem are complex and not completely understood (Hoyle et al. 1999), the implications of self-esteem for psychopathology and health are undeniable (Kemeny et al. 2004).

Substantial evidence indicates that low self-esteem is a significant risk factor for depression, beginning with the groundbreaking work of Brown & Harris (1978). In a series of retrospective and prospective surveys, Brown and colleagues demonstrated that chronic low self-esteem was a statistically and clinically significant factor in vulnerability to depression (e.g., Andrews & Brown 1993, 1995). They proposed that self-esteem was a critical proximal (immediate) causal locus for the indirect effects of distal factors on affective vulnerability, including childhood loss or abuse experiences, temperament, and socioeconomic factors. Subsequently, Roberts & Monroe (1994) argued that overall level of self-esteem was not a robust predictor of the onset of depression. They proposed a multidimensional model of self-esteem in depression that included an emphasis on structural deficits within the self (such as few, rigid, or externally based sources of self-worth); low levels of self-esteem exacerbated by dysphoric mood, stressful events, or schema-congruent experiences; and temporal instability of self-worth. This model, in turn, was elaborated by Crocker & Wolfe (2001) and other investigators.

Depression and attributional style. Personality and clinical psychologists have long noticed that variability in explanatory style was associated with psychopathology. Social psychologists, in turn, have determined that people explain their outcomes through reference to a wide variety of causal factors. A vast body of evidence indicates that Heider's (1958) classic four factors (ability, effort, luck, and task difficulty) are among the most frequently offered explanations for positive or negative outcomes. For the past 40 years, Weiner's (1985) theory of attribution has been a dominant model of how individuals interpret their successes and failures. Attribution theorists emphasize that individuals' interpretations of their outcomes determine subsequent strivings (or lack thereof) and the psychological consequences of perceived success and failure (Anderson et al. 1996). Weiner's model classified attributions into three causal dimensions: locus of control, stability, and controllability. In turn, each of these causal dimensions has identifiable influences on motivation, behavior, and affect. Some degree of self-serving attributional bias (a tendency to attribute positive, but not negative, outcomes to one's own efforts, ability, etc.) appears to be universal, and greater positive bias is associated with greater psychological and physical health. Theories of explanatory style and attributional biases have been enormously influential in the

development of cognitive models for depression (Gibb et al. 2004), anxiety, and related psychological problems (Bell-Dolan & Anderson 1999), and particularly the hopelessness theory of depression (Abramson et al. 1989).

Self-Regulation and Depression: General Principles

Even from this limited review, it is possible to assemble a set of general principles to conceptualize unipolar depression as a disorder of self-regulation. These principles are summarized in broadly applicable language rather than theory-specific language:

1. Depression results from cumulative or catastrophic failure of the individual's neurobiological and psychological capacity for successful goal pursuit. Following the logic of Kendler (2005) and Akiskal & McKinney (1973), a self-regulation perspective postulates that depression is not a brain disorder, or a cognitive disorder, or a behavioral disorder; it is a systemic disorder that is manifested throughout all levels of human goal pursuit. To understand depression, we must study it from a multidisciplinary context that acknowledges complementary perspectives (molecular, neural, cognitive, and interpersonal) on the specific dysfunctions and symptoms that characterize the disorder.
2. An initial episode of depression is a functional disorder resulting from failure of self-regulation. This assertion is intended as an alternative to a disease model of depression, while acknowledging the critical importance of distinguishing between initial and subsequent episodes (both for pathophysiology and for psychological sequelae). Such self-regulatory dysfunction may result from a single experience or an accumulation of experiences. One likely scenario is that an initial episode of depression results from a downward spiral of failure to make progress toward approach or promotion goals, with increasingly more severe and prolonged physiological, cognitive, and interpersonal consequences. Beyond a certain level of cumulative failure, the mechanisms that sustain goal-based behavior can no longer function normally, and a downregulated behavioral state, manifested as a depressive episode, ensues. Note that by functional disorder we mean, as postulated by Akiskal & McKinney (1973), that although the mechanisms of goal pursuit are responding maladaptively, there is (as yet) no permanent, structural change in the affected systems—although with further episodes such structural change may be inevitable (see number 4, below).
3. Core symptoms of depression reflect dysregulation of approach/promotion (e.g., mood, appetite, anhedonia, energy, concentration, worthlessness, hopelessness, low self-esteem) or dysregulation of reciprocal inhibition between approach/promotion and avoidance/prevention [e.g., sleep disturbance, guilt, agitation/anxiety, hypothalamic–pituitary–adrenal (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). The same type of dynamic reciprocal inhibition has been postulated for the promotion and prevention systems, along with the prediction that in depression such an efficient and flexible motivational capacity becomes impaired (Strauman & Wilson 2010). In turn, promotion hypoactivation is predicted to cause and maintain symptoms such as dysphoric mood, anhedonia, hopelessness, and fatigue. Likewise, prevention hypoactivation would be expected to cause and maintain symptoms characterized by hyperarousal, anxiety, and stress hormone release.
4. As episodes of depression accumulate, self-regulatory mechanisms are permanently altered. This hypothesis is intended to account for increased subsequent vulnerability to depression and for the observation that the clinical manifestations of the disorder itself appears to change over subsequent episodes. That is, irreversible changes can occur within any level

of self-regulation, which, in turn, further increases risk for subsequent episodes. The more episodes, the greater is the risk for irreversible change within each system. One noteworthy corollary of this principle is that treatment strategies for chronic, multi-episode depression will necessarily be different (and, in particular, more multifaceted) than treatments for first-episode depression—because, in the latter case, CNS-wide structural changes, as well as profound and lasting disruptions in relationships and social support, are less likely to have occurred.

There are a number of potential advantages of a self-regulation perspective on depression. First, the insights of existing theories of depression are readily translatable, and operationalizable, in self-regulation terms. This is critical because 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 et al.'s (1989) model, can be alternatively 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 processes, such as associated changes in hedonic capacity, self-representation, approach motivation, interpersonal effectiveness, and HPA axis function following an experience of failure. Third, expanding the application of brain/behavior systems theory (e.g., the BAS/BIS) 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, predictable only on the basis of the goals and beliefs an individual possesses.

It is important to acknowledge the number of theories of self-regulation with implications for understanding the etiology and treatment of depression. Outstanding examples of applying self-regulation models to depression include Brinkman & Franzen (2015), Ingram et al. (2015), and Karoly (2006). Likewise, there have been highly influential discussions of self-regulation more generally, which have substantial translational potential for the diagnosis and treatment of depression (e.g., Baumeister et al. 2007, Gollwitzer 1999, Heatherton 2011, Karoly 1993). Our focus here is on a limited subset of such theories rather than the entire domain. The concept of self-regulation, as a proximal locus for the influence of a broad range of distal biological, psychological, and social factors, represents a fertile source of novel interventions for depression.

Example: A Regulatory Focus Risk Phenotype for Depression

In the previous section, I outlined the predictions that SDT and RFT made regarding dysfunctions of self-regulation associated with depression versus anxiety. In this section, I present additional findings exploring self-regulatory mechanisms that may be dysfunctional during a depressive episode. These studies, which incorporated functional neuroimaging and behavioral genetics, were intended to challenge the model's predictions. The findings offer glimpses of how to target psychological interventions toward self-regulatory deficits in depression.

Neuroimaging evidence for self-regulatory dysfunction in depression. A series of studies using fMRI have shown that dysphoric and anxious symptoms are associated with specific neural markers of self-regulatory dysfunction. Strauman (2002) had predicted that depressed individuals would manifest an attenuated motivational response to promotion goal activation (weaker engagement, decreased eagerness), whereas anxious individuals would show an exaggerated response to prevention goal activation (stronger engagement, greater vigilance). Eddington et al. (2009) examined the neural correlates of promotion and prevention goal priming in a sample of patients meeting DSM-IV criteria for depression, with or without comorbid generalized anxiety

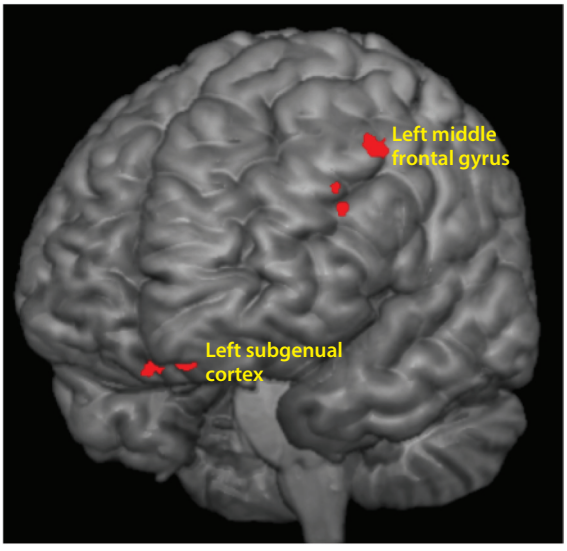

Figure 1

Group × priming condition interaction showing areas of greatest deficit in response to reward cues among depressed patients. The areas in red, left middle frontal gyrus and left subgenual cortex, show a statistically significant difference in activation between depressed patients and nondepressed controls in response to individualized promotion goal priming.

disorder (GAD), as well as a nonpsychiatric control sample. They observed a significant difference in activation between the depressed and nondepressed groups following idiographic priming with promotion goals, in which the patients showed attenuated left PFC activation compared with controls. In addition, they compared depressed patients with comorbid GAD versus those without and observed a region in the right PFC uniquely activated following prevention priming, but only for patients with comorbid anxiety.

Figure 1 summarizes a comparison of depressed patients ($N = 40$) versus matched nondepressed controls ($N = 40$) in a rapid masked goal priming fMRI paradigm that identified key brain regions that engaged following exposure to personally salient cues for reward. Depressed patients manifested attenuated left middle frontal gyrus and left subgenual cortex (Broadmann area 25) activation following subliminal promotion goal presentation, but this neuromotivational deficit was especially pronounced among a subset of the patients who met retrospectively our criteria for the risk phenotype. The figure shows the regions where the reduction in BOLD signal intensity was most significant among the depressed patients. These new findings may represent a neural signature for self-regulatory dysfunction in depression, as a general characteristic of ongoing depressive episodes and—potentially—as an index of self-regulation-based vulnerability to subsequent episodes.

***COMT* genotype and regulatory focus.** Self-regulation is critical for the control of adaptive behavior involving both bottom-up biological mechanisms and top-down social-cognitive mechanisms. On the basis of two decades of translational research examining the role of self-regulation in vulnerability to depression, my colleagues and I have hypothesized that the combination of (*a*) genetic predisposition to intense, prolonged goal pursuit (via the impact of the *COMT* gene polymorphism on mesocortical dopamine availability), (*b*) socialization history of contingent interactions with caregivers emphasizing pursuit of positive outcomes, and (*c*) perseverative responses to

goal pursuit failure is a risk phenotype for unipolar depression. Both behavioral and neuroimaging data have supported the proposed risk phenotype, and have illustrated how goal pursuit processes are impaired during a depressive episode.

Several recent studies from our lab have examined how a functional genetic polymorphism, *COMT Val158Met*, interacts with individual differences in socialization to modulate successful versus unsuccessful goal pursuit depending on the context. High levels of cognitive stability associated with the Met allele can facilitate self-regulation in certain situations (e.g., to maintain a successful strategy) but can be detrimental in others (e.g., when a change in strategy is required). The functional role of the *COMT Val158Met* polymorphism can be conceptualized as a trade-off in which the adaptiveness of the Val- or Met-like dopaminergic signaling profiles is determined by features of the environment. Our risk phenotype model postulates that individuals who are characterized by the combination of *COMT* Met/Met genotype and a history of contingent interactions with caregivers focused on positive outcomes are at risk for perseverative behavior and a downward cycle of perceived failure and associated anhedonia or hopelessness when they encounter repeated failure. A study using a well-validated reward learning task showed that the risk phenotype group had greater difficulty learning reward contingencies (Goetz et al. 2013). Specifically, both Met/Met and Val/Met participants showed impaired learning of task reward parameters, particularly when their own reinforcement history was dominated by successful pursuit of positive outcomes. **Figure 2** shows that Met/Met and Val/Met participants were unable to learn the reward contingencies efficiently, consistent with our hypothesis that *COMT*-mediated emotional vulnerability was a trade-off process involving socialization history and environmental demands. Our most recent study identified a cognitive control profile of Met/Met homozygotes when faced with repeated failure or with a change in reward contingencies (E.G. Davis, I.J. Tharp, A.R. Hariri & T.J. Strauman, submitted manuscript). Met/Met participants showed significantly greater behavioral slowing on a reward-based decision task under conditions of both frustrative nonreward and contingency change. In turn, this difference was exacerbated by a socialization

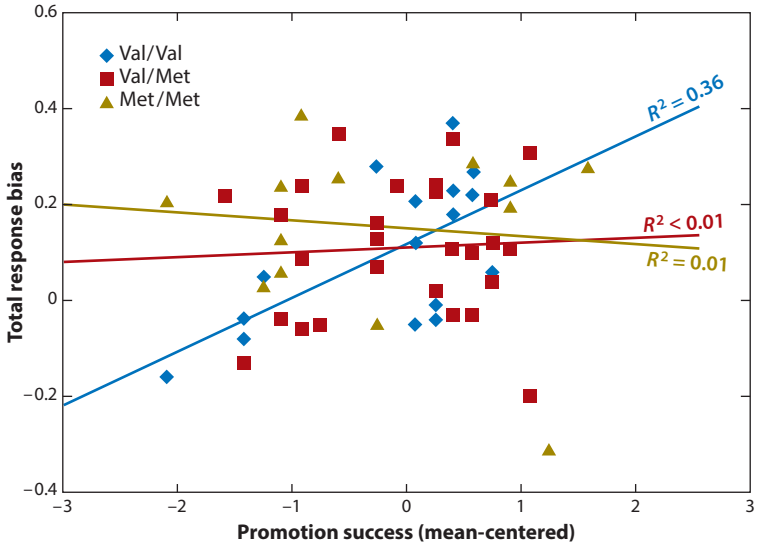

Figure 2

The correlation between positive outcome socialization history and learning of reward contingencies is moderated significantly by the *COMT* genotype.

history of positive outcome focus. Together, the two studies illustrate neurocognitive mechanisms that underlie our hypothesized risk phenotype and suggest what additional research will be required to validate the phenotype as a vulnerability pathway for major depressive disorder.

CONCLUSION: A PROPOSED RESEARCH FRAMEWORK

Although a more exhaustive review of self-regulation theories in psychopathology research undoubtedly would show a substantial variety of variables and causal pathways, I would claim that each specific theory or model shares the general principle outlined previously: Self-regulation is a critical locus for the proximal influence on motivation, cognition, emotion, and behavior of more distal factors such as genetics, temperament, socialization history, and neurophysiology. I also would claim that psychological theories of self-regulation for specific disorders are robust platforms from which to integrate the study of self-regulation across traditional disciplines and levels of analysis. In this final section, I propose a generalizable research framework that draws from our own work on depression as a disorder of self-regulation and also fits the other models reviewed briefly, as well as additional self-regulation models that space limitations precluded mentioning. The framework is cast using the language of goal pursuit but could be recast in terms of other critical self-regulatory processes (Heatherton 2011). The organization of this research framework draws heavily from the self-regulation taxonomy of Hoyle & Gallagher (2015), as well as from the work of Nolen-Hoeksema & Watkins (2011), and the reader is referred there for a discussion of developing integrative, transdiagnostic models of psychopathology.

A Self-Regulation-Based Psychopathology Research Framework

The framework begins by reiterating a working definition of self-regulation: the processes by which people initiate, maintain, and control their own thoughts, behaviors, or emotions, with the intention of producing a desired outcome or avoiding an undesired outcome. Our own research has highlighted dysfunction in goal pursuit processes, and that language will be used as an example, but the working definition is invoked here to encourage other researchers to cast their conceptual nets broadly. A thorough review of the descriptive literature for the disorder of interest should highlight several potential avenues for a self-regulation approach. Having done so, the researcher can then follow a series of steps intended to elucidate the distal and proximal self-regulatory processes potentially implicated in the disorder, as follows:

1. Description: Determine whether dysfunction of self-regulation is a sufficiently fundamental characteristic of the disorder to merit a more extensive examination of underlying processes. In what ways are affected individuals having difficulties producing important desired outcomes or preventing undesired outcomes? From a trade-off perspective, what self-regulatory costs are being incurred that outweigh or negate the self-regulatory benefits? Or, to use different conceptual language, in what ways are the normal patterns of learning (in which behavior with negative consequences becomes less frequent over time) disrupted by self-regulatory failure?
2. Identification: Label the key aspects of self-regulation relevant to the disorder. From among the range of available theories, what particular process (or processes) best captures the difficulties that were described in the previous step? As Nolen-Hoeksema & Watkins (2011) noted, such processes also may be either mediators or moderators within the overall model of disorder being postulated.
3. Specification: State the hypothetical precursors, mechanism(s), and consequences of dysfunction in that self-regulatory process. Our experience has been that precursors need not

be dysfunctional or even represent deficits, in and of themselves. Gene by environment (G × E) interactions, and similar dynamic models of disorder, suggest that in many cases a specific combination of individual characteristics and environmental circumstances is required for the dysfunction to arise.

4. Phenotypic history: Imagine critical points in development at which the dysfunction might appear. Again, using the example of G × E interactions, what critical events, periods, or trends in the individual's developmental history might shed light on the origins of the self-regulatory dysfunction being implicated? As above, these events or trends need not themselves be pathological or dysfunctional to contribute to ultimate risk for a specific disorder.
5. From risk phenotype to disorder: Describe a pathway by which the dysfunction leads to the onset of a dysregulated state (i.e., the disorder itself). The sequence of events between the emergence of a full-fledged risk phenotype to the onset of a clinically diagnosable disorder may or may not involve the same developmental time scale as the emergence of the risk phenotype. Likewise, the pathway leading from phenotype to disorder may be similar or distinct from those contributing to the establishment of the risk phenotype.

Example: A Flowchart for Self-Regulatory Dysfunction in Depression

Figure 3 presents our self-regulation risk phenotype model for depression in the form of a flowchart. Our intent, and our challenge, in presenting the model this way is to be as specific as possible with regard to each step outlined above. Using a flowchart also helps to bring into relief which aspects of the model have empirical support and which remain to be tested. Note that the example flowchart necessarily conflates and distorts developmental time, with the hypothesized contributory antecedents of the risk phenotype (*COMT* Met/Met genotype, a socialization history dominated by an emphasis on promotion, and a history of success in pursuing promotion goals that creates an expectancy of future success) compressed into a single level in the chart. In turn, the flowchart proposes that those three antecedents converge on the individual's goal pursuit experiences, which are presumed to increase in complexity and difficulty moving from childhood to adolescence to adulthood (Higgins 1989). The time frame of the flowchart then shifts to momentary challenges in promotion of goal pursuit and hypothesizes the immediate and eventual consequences of repeated failure feedback and perseverative goal pursuit efforts. We also have listed one potential moderator, rumination, and linked it with several of the predicted consequences of promotion goal failure feedback.

The flowchart suggests a series of questions, which, if the model itself is useful, should be amenable to empirical inquiry. For instance, What are the specific characteristics of the failure feedback that engenders perseverative goal pursuit efforts? Which moderators, in addition to rumination, might exaggerate or diminish the downstream consequences of perceived failure? What is the role of individual differences in emotion regulation within this hypothesized pathway to an episode of depression? Should there be looping arrows from promotion system hypoactivation [meant to resemble the "functional derangement of the diencephalic mechanisms of reinforcement" hypothesized by Akiskal & McKinney (1973, p. 26)] back to earlier processes depicted in the flowchart, as well as the self-regulatory processes that contribute to both the maintenance of the episode and the increased risk for subsequent episodes? And with regard to treatment, can the flowchart help to identify depressed individuals who might benefit optimally from a psychotherapy focusing specifically on self-regulation (Strauman et al. 2006)?

In closing with this example, my intent is simply to illustrate how a program of research that examines self-regulatory dysfunction in psychopathology might be structured. If nothing else, it

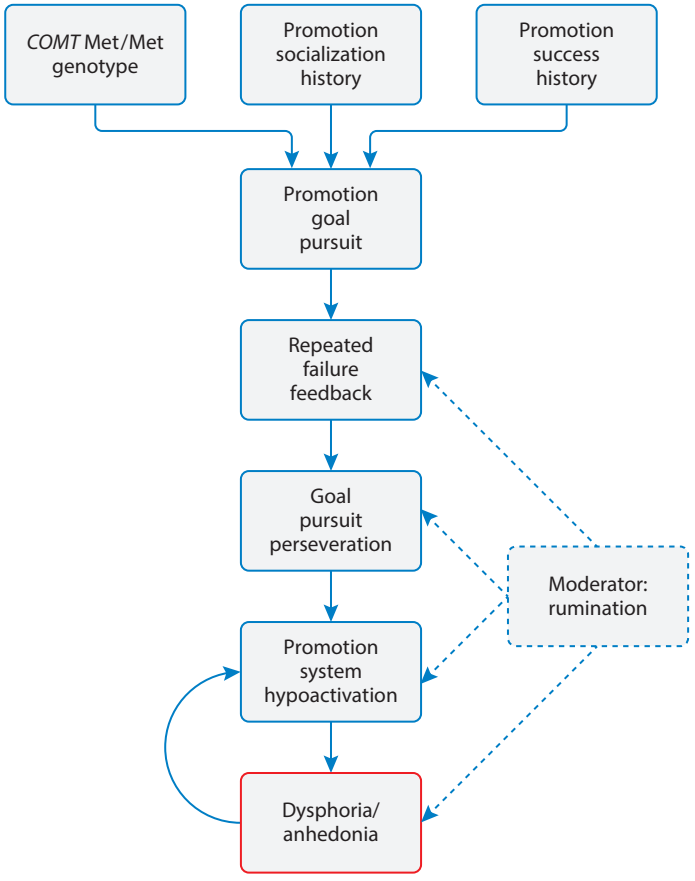

Figure 3

A flowchart describing our model for self-regulatory dysfunction as a contributory causal factor for depression, based on the hypothesized self-regulation risk phenotype. Dysphoria/anhedonia represents the dysfunctional state corresponding to depression. The dashed box and arrows signify the potential moderating influence of ruminative thought at different points in the sequence of events.

reminds us that many questions remain to be answered before we can confidently assert that the proposed risk phenotype is a valid construct. Hopefully, the model building process can serve the purpose of guiding translational research in a proactive manner so that new approaches to diagnosis, treatment, and prevention can be generated and tested. The challenge to create such models, across development, levels of analysis, and disorders, is daunting. But psychology and neuroscience have an enormous amount to offer, and this translational process has the potential to advance our understanding of psychopathology and our ability to remediate and prevent it.

SUMMARY POINTS

- The term self-regulation refers to the ongoing process of managing personal goal pursuit in the face of internal, interpersonal, and environmental forces that could interfere with that goal pursuit.

- Self-regulation represents a critical locus for the proximal influence on motivation, cognition, emotion, and behavior on more distal factors such as temperament, socialization history, physiology, and the physical and social environment. As such, theories of self-regulation are highly relevant to understanding psychopathology.
- In addition to other well-validated theories of self-regulation, self-discrepancy theory and regulatory focus theory offer useful insights into the etiology and treatment of depression.
- A general yet flexible self-regulation research framework may be of value for understanding the diagnosis, treatment, and prevention of a broad range of psychological disorders.

FUTURE ISSUES

- What are the implications of the proposed self-regulation research framework for enhancing diagnosis of mental disorders? Could such a mechanism-focused framework offer guidance regarding development of new diagnostic and assessment methods?
- How can the self-regulation research framework inform treatment development? Using self-system therapy (Strauman et al. 2006) as an example, can models of self-regulation be translated with appropriate fidelity to efficacious therapeutic interventions for depression and other disorders?
- By combining knowledge of the psychological and neural processes associated with self-regulation with a broad neurodevelopmental perspective, can more efficacious and precisely targeted preventive interventions emerge from this research framework?
- How might this and similar research models be integrated effectively into the recently developed Research Domain Criteria for psychopathology research?

DISCLOSURE STATEMENT

The author is not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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