COGNITIVE VULNERABILITY TO PSYCHOLOGICAL DISORDERS: OVERVIEW OF THEORY, DESIGN, AND METHODS

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Cognitive vulnerability models offer popular ways of understanding the origins and causal factors that contribute to psychological problems. Cognitive vulnerabilities are typically purported to create liabilities to psychological disorders after individuals encounter stressful events, in a vulnerability–stress interaction, and to maintain the problems after their onset. We present a conceptual framework that describes common features shared by most cognitive models, including schematic biases in information processing, developmental factors, reciprocal feedback loops, and specific vulnerabilities for specific problems or disorders. We also examine issues related to the role of theory in determining the appropriate research design, and discuss actual design options and methods used in cognitive vulnerability research, and their strengths and limitations.

Over the past 30 years, cognitive perspectives, including a focus on information–processing, have been extended to depression, anxiety, eating disorders, substance abuse, marital problems, and a variety of psychological problems (e.g., Alloy & Riskind, 2006; Mathews & MacLeod, 1994). The present article reviews some of the basic issues relating to the theory, design, and methods in research on cognitive vulnerability to psychological problems. In the sections of this article, we first discuss basic tenets of cognitive models of psychological problems, including the concept of cognitive mediation, and vulnerability–stress

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interaction. We next examine issues related to the role of theory in determining the appropriate research design, and discuss actual design options and methods used in cognitive vulnerability research, and their strengths and limitations. We conclude the article with a brief summary.

BASIC TENETS OF COGNITIVE MODELS OF PSYCHOLOGICAL PROBLEMS

Cognitive clinical models assume that cognitive phenomena mediate the relationship between events that a person experiences and subsequent emotional responses. That is, between the situation and the individual’s responses comes the all-important step of information processing and cognitive appraisal (e.g., Fridja, 1987; Lazarus, 1991; Ortony, Clore, & Collins, 1988; Roseman, Spindel, & Jose, 1990). For example, individuals who attribute failures to stable personal defects are likely to become more depressed and exhibit more helplessness. This same guiding principle applies to internal stimuli (e.g., physical sensations, unwanted thoughts or emotions) as to stimuli in the external environment (e.g., criticism from others).

The principle also applies to both “normal” and “abnormal” responses of individuals. That is, such models are also based upon the precept of a continuity of normal and abnormal cognitive processes. As Beck (1991) stated: “The [cognitive] model of psychopathology proposes that the excessive dysfunctional behavior and distressing emotions or inappropriate affect found in various psychiatric disorders are exaggerations of normal adaptive processes” (p. 370). At the same time, people can exhibit relatively habitual or stable patterns in the ways in which they appraise emotion-provoking stimuli (e.g., Abramson, Seligman, & Teasdale, 1978; Riskind et al., 2000; Weiner, 1985). For example, some individuals habitually attribute failures to personal defects. As a result of these differences, people can differ in their future risk for developing particular kinds of emotional disorders or psychological problems.

Importantly, cognitive models of vulnerability distinguish distal phenomena, which were present before the problem developed, from proximal phenomena that occur close in time to onset of its symptoms (Abramson, Metalsky, & Alloy, 1989). Distal cognitive factors are typically cognitive predispositions (e.g., dysfunctional attitudes or depressive inferential styles) to respond to stressful situations in maladaptive ways. They tend to be more trait-like aspects of personality that are more distal to future episodes than proximal cognitions, which are situationally specific thoughts or mental processes that occur very close to, or even during, the future episode of the psychological problem. Proximal cognitions are typically produced when individuals process a
stressful experience in any situation through the underlying cognitive vulnerability. The cognitive vulnerability is developed from earlier social learning history or developmental events.

COGNITIVE VULNERABILITY–STRESS PARADIGM

Most theorists adopt a vulnerability–stress paradigm in which it is recognized that psychological disorders are caused by a combination of predisposing (constitutional or learned) and precipitating (environmental) factors. That is, precipitating events (e.g., stressful life events, early childhood traumas, a marital disagreement) can trigger the development of psychological problems or emotional disorders for certain individuals (e.g., see Alloy, Abramson, Raniere, & Dyller, 1999), but the degree and even direction of response can differ enormously from one person to another. Thus, precipitating environmental events are particularly likely to produce psychological problems among individuals who have a preexisting cognitive vulnerability to the disorders.

Indeed, most individuals who are exposed to precipitating stressful events do not develop disorders. And, when disorders or problems emerge, their specific nature differs for different individuals and is not determined just by the precipitating stress alone. For example, stressful events are elevated in depression (Brown & Harris, 1978; Paykel, 1982), bipolar disorder and mania (Johnson & Roberts, 1995), anxiety disorders (Last, Barlow, & O’Brien, 1984; Roy-Byrne, Geraci, & Udde, 1986), and even schizophrenia (Zuckerman, 1999). Thus, cognitive vulnerability–stress models are offered to help account for not only who is vulnerable to developing emotional disorder (e.g., individuals with a particular cognitive style), and when (e.g., after a stress), but to which disorders (e.g., depression, eating disorder, etc.) they are vulnerable (Raskind & Alloy, 2006).

Researchers working in the cognitive paradigm often prefer the term “vulnerability” rather than “diathesis,” because this former term embraces the idea of learned and modifiable predispositions, instead of immutable genetic or biological traits (e.g. Just, Abramson, & Alloy, 2001), which is implied by the latter term. For example, Beck (1967, 1976), the earliest expounder of a cognitive vulnerability–stress interaction, proposed that an individual’s cognitive vulnerability to later difficulties is brought about by maladaptive knowledge structures or schemata formed of core beliefs, attitudes, and concepts that individuals develop from earlier experiences. The individual’s schemata influence the idiosyncratic subjective meaning that the person attaches to events and thus the emotional impact of stressful events. As such, cognitive schemata
can promote psychological and/or interpersonal maladjustment when they are poorly grounded in social reality or otherwise dysfunctional.

According to Beck's formulation, a change in the nature of information is fundamental to the onset of psychological problems. For example, once the underlying mechanism (e.g., schemata) for a relevant psychological problem (e.g., depression, anxiety, anger) is set off, the nature of self-referent cognition undergoes a profound change. For example, individuals may be biased to judge themselves as failures, which can increase vulnerability to problems of mood such as depression or anxiety. Moreover, people are thought to think differently about themselves, the world, the future, or the activities in their lives, and to remember, screen, select, and interpret information in different ways during states of depression, anxiety, etc., than during normal states.

COGNITIVE VULNERABILITY MODELS: A FRAMEWORK OF COMMON FEATURES

While cognitive vulnerability models differ in their particulars and their subject matter, most share important common features (see Riskind & Alloy, 2006). As depicted by Figure 1, most tend to assume a series of causal chains by which cognitive vulnerabilities develop from earlier life experiences (such as faulty attachment relationships or modeling). Once individuals have developed cognitive vulnerabilities, these are likely to alter their responses, and with later stressor/vulnerability combinations are converted into the emotional or psychological problems. The cognitive vulnerabilities are assumed to have schematic function (e.g., Clark, Beck, & Alford, 1999). As such, the vulnerabilities shape and schematically bias the person's selective processing, attention, and memory, and promote changes in the concomitant contents of the individual's thinking (i.e., the ideation, imagery, or "automatic" thoughts). The schematic biases associated with their cognitive vulnerabilities can guide the whole range of activities involved in information processing (e.g., selective attention, encoding, elaboration and retrieval in memory, interpretation).

An important characteristic of cognitive vulnerabilities is that they are associated with "disorder-specific" information processing biases (e.g., attention vs. memory) and in specific typical proximal cognitions (Beck & Clark, 1997). For example, the disorder-specific bias in anxiety is widely assumed to involve information relevant to threat (e.g., public humiliation), and accompanied by proximal thoughts such as "I'll look like a fool." In depression, the specific bias is for information relevant to past loss, irreversible defeat, and hopelessness. Such disorder-specific information-processing biases are presumably instigated when cogni-
tive vulnerabilities (distal factors) are engaged, but the stages of processing and subject matter that is implicated can differ with different models and psychological problems or disorders (e.g., Williams, Watts, MacLeod, & Mathews, 1988).

In this regard, some kinds of problems may involve biased information processing at multiple levels of the cognitive continuum (e.g., attention, encoding, retrieval), whereas other problems may involve processing biases concentrated at particular points on the continuum. Examples of early stage biases would be the hyper-vigilance for threat...
stimuli that occurs in people who are anxious, or an attentional bias for positive information that is likely to occur in people who are highly optimistic. Examples of later stage biases would be slanted tendencies to interpret and elaborate the meaning of stimuli in schema-congruent manners. The notion that cognitive content and biases differ in different psychological problems has been the impetus for considerable research.

A variety of cognitive vulnerability factors have been suggested (Alloy & Riskind, 2006). Beck’s cognitive model emphasized the role of faulty beliefs and dysfunctional attitudes in creating vulnerabilities to depression, anxiety, or other psychological problems (e.g., Beck, 1976; Beck & Clark, 1997). Other current models of depression focus on dysfunctional inferential (attributional) styles that create a cognitive vulnerability (Abramson, Metalsky, & Alloy, 1989; Alloy, Abramson, Safford, & Gibb, 2001). A recent model of anxiety (Riskind, Williams, Gessner, Chrosniak, & Cortina, 2000; see Riskind, Williams, & Joiner, this issue) emphasizes the roles of maladaptive mental simulations of danger involving the generation of dynamic mental scenarios of rapidly rising risk (Riskind, Williams, Gessner, Chrosniak, & Cortina, 2000).

In combination with cognitive vulnerability factors, the risk of developing psychological problems and their potential severity after exposure to stress is also influenced by protective and exacerbating factors. Even after exposure to stress, the presence of certain protective factors (e.g., social support, an intimate relation with a spouse or lover, effective coping mechanisms) may work against the development of psychological problems, or reduce the likelihood the problems will develop (e.g., Brown & Harris, 1978). Exacerbating factors are additional stresses or factors (e.g., further medical illnesses, psychological problems, or negative affect expressed by others) that impinge on the individual and act to worsen a psychological problem or disorder after its onset. As a result, a set of individuals could at any point begin with precisely the same combinations of vulnerability and stress, but still markedly differ in their future trajectories of problems because they differ in protective and exacerbating factors.

In this context, many additional factors potentially related to vulnerability to emotional disorders such as personality factors, demographic factors, developmental experiences, or interpersonal patterns can be sensibly understood with the cognitive paradigm. For example, personality characteristics such as negative affectivity or neuroticism can be seen as the consequences of the activation of underlying negative schemata (e.g., Jolly & Kramer, 1994). Factors such as the availability of social support can be understood as serving to buffer stress by helping to impart more adaptive inferences about the causes and consequences of negative life events (Dobkin, Panzarella, Fernandez, Al-
loy, & Cascardi, 2004; Panzarella, Alloy, & Whitehouse, in press). Similarly, factors such as gender can be sensibly understood within the cognitive paradigm in terms of their relationship to dysfunctional rumination patterns that affect a person’s focus of attention on negative events (e.g., Nolen-Hoeksema, Morrow, & Frederickson, 1993). When possible, it can often be of value to conceptualize cognitive mechanisms by which “noncognitive” variables can interact with the cognitive system (Riskind & Alloy, 2006).

Beyond these factors, vicious cycles involving bi-directional causal links (and feedback loops) can also contribute to the onset, maintenance, or recurrence of psychological problems (see Figure 1). For example, under the duress of the stress and intense symptoms, cognitively vulnerable individuals tend to engage in various maladaptive self-protective or compensatory behaviors. Depressed individuals often have a heightened inclination to engage in reassurance seeking from others (Joiner, Alfano, & Metalsky, 1992), which can alienate others or drive them away. Individuals who are depressed also contribute to the occurrence of self-generated life events (e.g., creating interpersonal conflicts or excessive demands) that can maintain and exaggerate their own depression (Hammen, 1991). Recent research suggests that individuals who are cognitively vulnerable to anxiety may also create their own stressful life events (Shahar, Black, & Riskind, 2004). Some individuals who are anxious engage in avoidance behaviors, some relatively subtle—such as avoiding eye contact to avoid seeing imagined rejection—that can self-generate stress and prove maladaptive.

Psychological problems are believed to persist as long as the cognitive components of the problems are present, and improve when they are altered. Further, although temporary relief is produced by changes in proximal cognitive components of the problems, durable improvement requires changes of the underlying cognitive vulnerability factors.

**ADDITIONAL ISSUES FOR COGNITIVE VULNERABILITY MODELS**

Several additional issues confront cognitive vulnerability models of psychological disorders (for a longer discussion of such issues, see Riskind & Alloy, 2006). These issues include the (1) specificity of cognitive vulnerability factors, (2) the specificity and nature of developmental antecedents, and (3) the role of cognitive vulnerability in comorbidity,

**SPECIFICITY OF COGNITIVE VULNERABILITY FACTORS.**

The first issue for cognitive models concerns the distinction between
specific and nonspecific causal factors in psychological problems/ emotional disorders. *Specific* causal factors are relatively unique or focal factors in that they influence and predict the development of a particular problem, but do not equally apply to all psychological problems or disorders in general. For example, some vulnerability factors might apply to just social anxiety, but not other forms of anxiety. In contrast, other cognitive vulnerability factors could be specific to the whole spectrum of different forms of anxiety, but not apply to depression (e.g., Riskind & Williams, 2006). Alternatively, *nonspecific* (or common) causal factors are characterized by their relatively low discriminatory power (Ingram, 1990; see also, Clark, 1997) and in this way, potentially cut across a range of different psychological problems (e.g., depression, anxiety, substance abuse disorders, marital problems). A likely instance of a nonspecific factor is prior exposure to uncontrollability (Chorpita & Barlow, 1998), which seems to play an important, but nonspecific, role in many disorders. It is, of course, important to understand nonspecific or common causal factors that apply to multiple psychological problems. At the same time, however, any adequate cognitive model of such problems also needs to identify causal factors that have discriminatory power and applies to specific problems and not others. By this view, it is necessary to identify possible factors that may play roles as specific cognitive vulnerabilities for specific problems.

COMMON AND SPECIFIC DEVELOPMENTAL FACTORS

As can be seen from the framework depicted in Figure 1, it is often assumed that people’s antecedent childhood experiences can help to mold the nature of the cognitive vulnerabilities they later develop. Recent research does, indeed, suggest that childhood experiences such as peer rejection or faulty attachment relationships may lead to cognitive vulnerabilities (e.g., Gibb, Alloy, Abramson, & Marx, 2003; Ingram, 2003; Ingram, Bailey, & Siegle, 2004; Riskind, Williams, Altman, Black, Balaban, & Gessner, 2004; Rogers, Reinecke, & Setzer, 2004; Safford, Alloy, Crossfield, Morocco, & Wang, 2004; Williams & Riskind, 2004). However, the specificity of such developmental factors to particular psychological problems is still relatively unknown. If such developmental variables were, indeed, to be found to constitute such common factors, they would be expected to play a general, but nonspecific, role in the origins of many psychological problems.

In some cases, it is perhaps not just childhood experiences alone, but factors such as variability in the particular factual details of childhood experiences may also help account for differences in the vulnerabilities that are developed. For example, children could be more prone to de-
velop depression–related cognitive patterns if they have been subjected to unremitting or relatively constant parental abuse or criticism. In contrast, they might be more prone to develop anxiety–related patterns if they have been subjected to more variable parental negative events, and/or if some positive protective factors are present. Much more work on these developmental questions is clearly warranted.

COGNITIVE VULNERABILITY AND COMORBIDITY

Another issue is how cognitive models account for the comorbidity of psychological disorders (Alloy, Kelly, Mineka, & Clements, 1990). For example, it is well documented that anxiety and depression appear in a comorbid form (e.g., Zinbarg, & Barlow, 1996). Besides this, it is known that symptoms of anxiety and depression are often more severe when they co–occur than when they occur separately—such as when case controls only have one of the disorders (see Riskind, Moore, Harman, Hohman, Beck, & Stewart, 1991). Once we recognize that cognitive vulnerability factors can vary independently of each other, we might easily imagine that individuals with compound vulnerabilities (i.e., they combine cognitive vulnerabilities to different disorders) could be expected to be far likelier to develop comorbid emotional disorders. Preliminary support for just this notion is offered by a recent study that assessed both depressive explanatory style and looming maladaptive style (Riskind et al., 2000) and examined their main effects and interactions (Riskind & Williams, 2000). As expected, individuals who had the compound vulnerability, in that they combined both depressive inferential patterns and the looming cognitive style for anxiety (see Riskind, Williams, & Joiner, this issue), exhibited more severe symptoms of anxiety and depression than would be expected from the simple summation of utterly separate effects. Thus, the study of cognitive vulnerability factors has promise in helping to illuminate the nature of psychological antecedents of comorbid emotional disorders.

THE ROLE OF THEORY IN DESIGN SELECTION:
THE INTERFACE OF THEORY AND RESEARCH METHODS

Having examined the central theoretical facets of models of cognitive vulnerability, we consider the role that such theory plays in design selection, and how it is necessary to tailor the research design selected to test our research questions or the specific logic of the cognitive vulnerability models of interest. We will begin by examining several questions related to the kinds of causal relations specified, the nature of the vulnerability–stress combination, and the role of cognitive priming; then we focus
on specific logical criteria needed to support a hypothesized vulnerability factor.

HYPOTHESIZED CAUSAL RELATIONS IN COGNITIVE VULNERABILITY–STRESS MODELS

One set of issues to consider in selecting an appropriate research design concerns the type of causal relations (e.g., distal or proximal; necessary, sufficient, contributory) posited by a given cognitive model. For example, early negative life events (e.g., attachment relationships in childhood) can be viewed as a distal cause of psychological disorder (by way of a cognitive vulnerability). In contrast, later negative life events that are proximal to the onset of the psychological problem or disorder can be viewed as the precipitating event for an episode of the disorder.

The appropriate design of a research study is also influenced by whether the hypothesized causal role is necessary, sufficient, or contributory. In these terms, a necessary cause is an etiological factor that is an essential condition (either in the present or the past) for the disorder to occur. In the absence of the etiological factor, the disorder cannot occur, although the factor by itself does not require the disorder to occur (i.e., the factor is necessary but not sufficient). A sufficient cause is an etiological factor that guarantees the occurrence of the disorder, although the factor may not necessarily cause the disorder (e.g., the factor is sufficient but not necessary). A contributory cause increases the statistical likelihood that the disorder will occur by playing a supporting causal role, but is neither necessary nor sufficient for the occurrence of the disorder. In all, the hypothesized causal relations in the model should guide the nature of the research design as well as the choice of statistical analyses.

In some cognitive models, it may be hypothesized that the cognitive vulnerability and stress combine in an additive fashion as a straightforward summation. In other models, in contrast, there is true statistical interaction (e.g., Abramson, Alloy, & Hogan, 1997; Alloy et al., 1999; Monroe & Simons, 1991) that predicts outcomes synergistically beyond the separate additive effects of the vulnerability and stress. For instance, the combination of a high level of cognitive vulnerability and high stress will be far likelier to lead to an episode of the psychological disorder in question than either factor (or their additive combination) alone. The specific manner in which the cognitive vulnerability and stress are postulated to combine as causal factors is critical in determining the appropriate statistical analysis as well as levels of the cognitive vulnerability–stress combination that must be sampled (or experimentally manipulated) to test the model (see Alloy et al., 1999).
Another key aspect of the hypothesized causal relations lies in the distinction between moderating and mediating factors (Baron & Kenny, 1986; Holmbeck, 1997). A **moderator** is a third variable that codetermines the outcomes by affecting the relationship between the independent variable (e.g., the cognitive vulnerability or stress or both) and the dependent variable (disorder). In essence, a moderator statistically interacts with the vulnerability or stress (or both) and affects the direction or strength of the relationship between the vulnerability–stress combination and disorder. For example, let’s suppose that gender is posited as a moderator of the relationship between a cognitive vulnerability–stress combination and depression (e.g., women may be more susceptible to such effects). What this means is that the direction or strength of the cognitive vulnerability–stress combination would depend on whether subjects were men or women. In contrast, a **mediator** is a third variable that is assumed to account for the relation between an independent variable (e.g., the distal cognitive vulnerability and proximal stress, or their combination) and the dependent variable (e.g., the disorder). The mediator can be seen as the intermediary process or mechanism by which the cognitive vulnerability–stress combination becomes converted into an episode of disorder. For example, as we saw before (Figure 1), biases in mental processes of memory or attention could be seen as a third variable that mediates the relation between a cognitive vulnerability and the onset of disorder. In sum, moderators specify the conditions under which a vulnerability–stress combination will lead to a disorder. In contrast, mediators specify how or why the vulnerability–stress combination leads to disorder. Baron and Kenny (1986) discuss appropriate differences in the statistical tests required to test moderation as opposed to mediation.

THE PRIMING OR ACTIVATION OF COGNITIVE VULNERABILITIES

During periods when such vulnerabilities are in an inactive or latent state, a cognitive priming task such as a relevant mood—induction or activating provocation task (Ingram, Miranda, & Segal, 1998; Riskind & Rhees, 1984) could be needed. One can make an interesting analogy between a cardiac stress test (which is used to reveal a hidden coronary dysfunction) and a cognitive priming task (which is used to reveal a hidden cognitive vulnerability). It follows that it may sometimes be necessary to bring out latent vulnerability with an appropriate priming test (for evidence, see Ingram, Miranda, & Segal, 2006). But not all cognitive vulnerabilities may require a priming procedure (Just et al., 2001). The extent to which cognitive activation and assessment of vulnerabilities requires priming in different models remains an open question.
LOGICAL CRITERIA NECESSARY TO SUPPORT A PUTATIVE COGNITIVE VULNERABILITY

There are four key logical criteria that must be satisfied to strongly empirically support a hypothesized cognitive vulnerability. First, the temporal precedence and stability of the vulnerability independent of the symptoms of the disorder must be established (e.g., Alloy, Abramson, Raniere, & Dyller, 1999; Ingram, Miranda, & Segal, 1998). That is, the putative vulnerability must temporally precede the initial onset of the psychological disorder, or, in the case of a vulnerability factor for the course of a disorder, it must precede episodes or symptom exacerbations of the disorder (i.e., it has predictive validity). Second, it must exhibit some degree of stability independent of the symptoms of the disorder. That is, the vulnerability must be shown to be more than just a transient state–manifestation or consequence of the changing symptoms of the disorder.

Third, and of equal importance, alternative explanations of results must be eliminated as plausible options. This is achieved in part by establishing that the predicted relationships are not due to potential third variables (or confounds; Cohen & Cohen, 1983). Confidence in the validity of the putative vulnerability is also increased by providing supplementary evidence that the vulnerability factor plays a causal role in the development of symptoms or onset of disorder (i.e., obtaining evidence for its construct validity). This being so, important evidence on these issues is supplied by a network of findings showing predicted differences in personality characteristics, information processing, coping patterns, etc., of individuals who are high or low in cognitive vulnerability. For example, results that can be interpreted as demonstrating the developmental antecedents or mediating processes (e.g., information processing deficits) specifically predicted by the cognitive theory (but not by a “rival” third factor), offers strong support to the system of hypotheses and the construct validity of the proposed cognitive vulnerability.

A fourth, and final, criterion for causal status is also worth highlighting. If a theory of interest claims that the vulnerability factor is restricted or specific to a certain disorder, it must further be shown that the factor is largely applicable to the disorder of interest and not to other disorders (i.e., it has discriminant validity). Evidence that a putative vulnerability has stability or temporal precedence is insufficient for establishing that it has specificity to given psychological problems (Riskind & Alloy, 2006). Thus, there is a need to directly test the specificity of the predicted outcomes.