

Cognitive Vulnerability to Depression: Exploring Risk and Resilience

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The concepts of risk and resilience are often described as different sides of the same coin. Risk refers to the heightened probability of negative outcome among individuals possessing certain vulnerabilities or sharing exposure to certain conditions. Resilience is a dynamic process encompassing the manifestation of positive functioning despite possessing vulnerabilities or the presence of high risk. Thus, risk factors increase the likelihood of developing a particular disorder, whereas resilience factors decrease the risk of developing that disorder. Although risk and resilience are inexorably linked to one another, most studies have focused on risk. There is a vast body of literature on environmental, genetic, biological, interpersonal, and cognitive risk factors for psychopathology. This work has increased our understanding of the causes of mental illness and our ability to identify at-risk populations. In principle, understanding the causes of psychopathology should lead to the generation and testing of interventions that decrease risk factors and, in turn, bolster resilience. However, results from risk research have not always led to advancements in resiliency research. Indeed, the resilience literature is much younger and smaller in volume than the body of research on risk factors.

Why has the transition from risk to resilience progressed slowly? One possible explanation is that researchers are more dedicated to testing theories of risk than they are to studying resilience. A more plausible explanation is that many “theories” of risk do not easily translate into resilience. For

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research on risk to inform resilience interventions, there must be a certain level of theoretical sophistication. It is not good enough simply to identify risk factors. For example, one of the most established findings in clinical research is that stressful life events confer risk for future psychopathology. A naive reinterpretation of this finding made in the context of bridging the domains of risk and resilience might state that if stressful life events constitute the risk, then eliminating these events should foster resilience. Unfortunately, it is not feasible to create an intervention that can eliminate all of life's stressors. And, even if stressful life events could be eliminated, it is unclear who should receive this intervention. Should stressful life events be eliminated from everyone's life or just the lives of a select group of people who are at risk? Not everyone who experiences a stressful life event develops psychopathology; in fact, stressful life events might lead to long-term positive changes for some individuals. Along these same lines, particular types of stressful life events may be more likely to lead to psychopathology than others. The point of this example is that risk factors cannot stand alone as "main" causes of psychopathology. Without more information about the relationship between stressful life events, their interactions with individual-based resilience factors, and resulting psychopathology outcomes, it is difficult to determine how to intervene.

It is critical for the field to build stronger theories of psychopathology. For risk research to inform resilience research, theories need to be more specific (see Ref. [1] for a discussion of theory construction). A good theory should specify an etiologic chain that elucidates the relationship between the risk factor and the disorder. It must also specify the elements of this chain for which issues of resilience are particularly important. In the example of stressful life events, it is necessary to understand how life stress leads to psychopathology. Without an understanding of the specific role of risk factors in psychopathology, it is not possible to create a theoretically driven intervention or to identify which people are most likely to benefit from it.

The purpose of this article is twofold. First, we show how a well constructed and well tested theory of risk can provide a framework for creating resilience interventions. We use the cognitive theories of depression as an example of how to create and enhance resilience in dealing with depression. Second, we expound on this example to sketch a more general structure of possible connections between risk and resilience. We also attempt to generalize some of these ideas to the context of cultural and cross-cultural research. In doing so, we explain why examples from such research might enrich our general causal theories of psychopathology.

Theory construction and evaluation

Depression is one of the most common and potentially lethal (through suicide) forms of psychopathology. By 2020, it is projected to be the second leading cause of disability worldwide [2]. Depression is also a substantial

financial burden. The total economic cost of depression is estimated at more than \$83 billion a year in the United States [3]. Understanding the factors involved in risk and resilience for depression is vital to our society.

Over the past 30 years, there has been an explosion of research on depression, with many investigators emphasizing the importance of cognitive factors in the etiology, maintenance, and treatment of depression [4,5]. According to these cognitive theories of depression, some individuals possess a “cognitive vulnerability” that interacts with stress to produce depression. Specifically, people are at risk for depression because they tend to generate interpretations of stressful life events that have negative implications for their future and their self-worth. People who generate these negative interpretations develop hopelessness, which is a proximal and sufficient cause of depression.

The cognitive theories of depression are an excellent example of strong theory construction (Fig. 1). These theories lay out a testable etiologic chain that specifies (1) distal and proximal causes, (2) contributory and sufficient causes, and (3) moderating and mediating risk factors. This type of specification in the etiologic chain makes it easy to subject these theories to “grave danger of refutation” [6]. It is critical to construct theories that are falsifiable because many philosophers of science argue that scientific progress occurs through the refutation of theories [1,6]. To place the cognitive theories in danger of refutation, researchers must test whether individuals with cognitive vulnerability

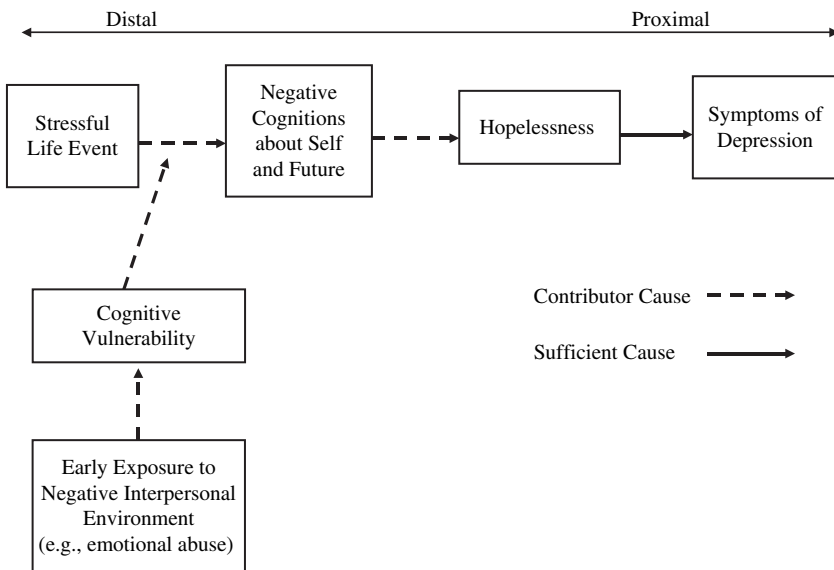


Fig. 1. A cognitive theory of depression. (Data from Abramson LY, Alloy LB, Hankin BL, et al. Cognitive vulnerability-stress models of depression in a self-regulatory and psychobiological context. In: Gotlib IH, Hammen CL, eds. Handbook of depression. New York: Guilford; 2002. p. 268–94.)

are at high risk for depression when stressful life events occur. If this central hypothesis does not hold up, this would be grounds for refutation.

A powerful method for testing the central hypothesis of the cognitive theories of depression is the “behavioral high-risk design” [7]. The behavioral high-risk design involves studying participants who do not have the disorder of interest but who are hypothesized to be at high or low risk for developing it. To test the cognitive vulnerability hypothesis of depression, one would select nondepressed people who are at high versus low risk for depression based on the presence or absence of the hypothesized depressogenic cognitive styles. One would then compare these cognitively high- and low-risk groups on their likelihood of exhibiting depression in the future.

Behavioral high-risk designs and approximations to it have provided strong support for the cognitive theories. Studies using these designs consistently find that adults and adolescents who have a cognitive vulnerability are more likely to develop depressive symptoms and disorders when they experience negative life events than are individuals who do not show this vulnerability [8]. Perhaps the most impressive results come from the Temple–Wisconsin Cognitive Vulnerability to Depression Project [8,9]. In the Cognitive Vulnerability to Depression Project, participants (college freshmen) with high cognitive vulnerability were significantly more likely than those with low cognitive vulnerability to develop DSM-IV major depressive disorder during the 2.5-year prospective follow-up. Taken together, these studies establish temporal precedence and suggest that cognitive vulnerability may be a causal contributor to the development of depressive symptoms and depressive disorders. Moreover, the results support the etiologic chain featured in the cognitive theories.¹ Given this empiric success, the cognitive theories are ripe for translation into preventions and interventions.

Using theory to inform studies of resilience

According to the cognitive theories of depression, negative interpretations of stressful life events create risk for future depression. To create or amplify resilience, an intervention should target one or both of the risk factors specified in the theory—stressful life events or cognitive vulnerability. It may not be feasible to eliminate stressful life events, but research suggests that it is possible to change an individual’s cognitive vulnerability. The cognitive theories indicate three main time points for a cognitive intervention: (1) before cognitive vulnerability develops (precognitive vulnerability), (2) after cognitive vulnerability develops but before the onset of depression, and (3) after the onset of depression.

¹ In addition to supporting the cognitive-vulnerability stress hypothesis, recent research supports the mediating role of hopelessness in the etiologic chain. The cognitive-vulnerability hypothesis holds for first onsets and recurrences of depression (see Ref. 8 for review).

Precognitive vulnerability

If cognitive vulnerability causes depression, then one strategy for creating or amplifying resilience is to prevent cognitive vulnerability from developing. This idea of creating or strengthening resilience for a risk factor (cognitive vulnerability) rather than the disorder itself (depression) is relatively new and untested. For most theories of risk, a prevulnerability intervention is not possible to realize because they do not specify the antecedents of the risk factor. In contrast, the cognitive theories of depression specify a number of empirically validated factors involved in the development of cognitive vulnerability.

Recent studies converge on the idea that early exposure to negative interpersonal contexts is an important antecedent of cognitive vulnerability (see Fig. 1). Specifically, children may acquire cognitive vulnerability by receiving direct inferential feedback from significant others or as a result of negative parenting practices [10–12]. The association between direct inferential feedback and children's own inferential style seems to be particularly robust [10,13–15]. For example, Cole and colleagues [13] conducted a 4-year longitudinal study and found that elementary school children's self-appraisals of competence in five developmentally important domains are significantly predicted by parents', teachers', and peers' appraisals of their competence in the same domain.

Children's cognitive vulnerability may also be influenced by the quality of their relationships with their parents [16,17]. The few studies that have examined the ability of parenting behavior to predict offspring's cognitions prospectively have provided consistent evidence supporting this hypothesis [10,18,19]. For example, Garber and Flynn [10] reported that level of maternal acceptance significantly and uniquely predicted children's self-worth 1 year later even after controlling for prior level of self-worth and mothers' history of depression.

On the far end of the negative parenting continuum is maltreatment. Studies have provided strong support for a link between a history of childhood maltreatment and the presence of cognitive vulnerability in adults [20]. Gibb and colleagues [21] provided evidence that particular types of maltreatment are more likely than others to be related to cognitive vulnerability. Specifically, their results indicated that childhood emotional, but not physical or sexual, maltreatment was related to cognitive vulnerability to depression [21]. Rose and Abramson [22] hypothesize that emotional maltreatment may be an especially potent contributor to cognitive vulnerability to depression because, unlike in physical or sexual abuse, the abuser, by definition, supplies negative cognitions to the victim.

In short, recent research suggests that early social environments may be an important determinant of whether or not a person develops cognitive vulnerability. These results lay the groundwork for a theoretically driven intervention targeting early child–parent relationships. For example, researchers

could identify parents who tend to generate negative interpretations for their children's behaviors and then intervene with cognitive training to help them generate more adaptive inferences about their child's behavior. Because the intervention targets parents' cognitions and behaviors, it can be administered before the child is born. A prenatal intervention may be effective for mitigating cognitive risk because the child would have minimal exposure to negative cognitions or a negative parent-child relationship. In one of the only prevulnerability interventions to date, Munoz and colleagues [23] reported administering a depression intervention to pregnant mothers who had a history of depression. The results of this ongoing project have not yet been published.

The idea of targeting children's early social environments with cognitive interventions is consistent with research in developmental psychology. For example, recent studies suggest that young children may not have the cognitive capacities necessary for developing a stable cognitive vulnerability factor (ie, they do not have a reliable style of interpreting events). Supporting this view, longitudinal and cross-sectional analyses by age indicate that the vulnerability-stress interaction may only predict depression in children older than 11 years of age [24,25] (but see Ref. [26] for exception). This work suggests it may be easiest to alter a child's developing cognitive vulnerability before age 11 because it has yet to stabilize. It is also important to target preadolescents because rates of depression skyrocket during middle to late adolescence [27]. Thus, reaching children before they develop cognitive vulnerability and before depression rates begin to rise may be an optimal time to create resilience.

Precognitive vulnerability interventions should focus on girls. Significant gender differences in depression exist among adults, with twice as many women experiencing depression as men [28]. Consistent with this gender difference in depression, women show elevations on two of the factors featured in the causal chain of the cognitive theories: stressful life events and cognitive vulnerability. Holding all else equal, girls should be targeted for early interventions more often than should boys.

Prevulnerability interventions are an innovative, and potentially effective, strategy for preventing psychopathology. The greatest strength of this type of intervention is that it builds or enhances resilience two steps out from the development of the disorder. In other words, it targets individuals who are vulnerable for developing risk for the disorder. However, further research is needed to determine if pre-vulnerability interventions are effective.

Cognitive vulnerability, predepression

The next time point for an intervention is after individuals develop a cognitive vulnerability but before they experience an episode of depression. An intervention at this time point is called a "prevention" intervention because

the goal is to prevent a high-risk person from developing psychopathology. A prevention intervention, unlike a prevulnerability intervention, targets individuals who are at imminent risk for developing depression. According to the cognitive theories, depression can be prevented if cognitive vulnerability is decreased. A prevention intervention based on this theory would identify a group of cognitively vulnerable individuals and administer an intervention that decreases their level of cognitive vulnerability. Prevention interventions are particularly important for disorders that are recurrent, such as depression. Once a person has an episode of depression, they are at high risk for developing another episode [29]. Thus, it is critical to try to prevent the onset of a first episode of depression.

Cognitively based prevention interventions have been tested in a variety of populations from children to college freshman. The interventions, based on cognitive behavioral treatments for depression, teach people to identify, evaluate, and change their negative cognitions. Many of these studies have not specifically targeted cognitively vulnerable individuals. Rather, these studies have selected participants based on other risk factors for depression or have used a universal approach (ie, using an entire population regardless of risk status). Results of studies using the universal approach have not been supportive. Most universal interventions do not significantly reduce risk for depression [30]. These results may not be surprising given the unselected sample, and they suggest that prevention interventions should target at-risk populations. Thus, building and enhancing resilience in the absence of risk factors for psychopathology might not bear immediate results for preventive efforts with regard to that specific psychopathology.

In contrast to universal prevention programs, results from the few prevention studies specifically targeting at-risk samples have been promising. For example, Clarke and colleagues [31] administered a cognitive behavioral intervention to high school students who reported high levels of depressive symptoms. Results showed that at-risk participants who received the intervention were significantly less likely to develop depressive disorders during a 1-year follow-up period than were participants in the control condition. Clarke and colleagues [32] found similar results with a sample of adolescents who had depressed parents. Jaycox and colleagues [33] also found support for cognitive behavioral prevention programs for at-risk school-aged children. In this study, children were selected based on levels of depressive symptoms or family conflict. Results showed that children who participated in the cognitive behavioral intervention had fewer depressive symptoms than did children in the control conditions immediately after treatment and at the 2-year follow-up assessment. These results suggest that cognitive behavioral interventions can effectively prevent future depression even in samples not specifically chosen for cognitive vulnerability.

Only one study has specifically targeted cognitively vulnerable individuals. Seligman and colleagues [34] randomized cognitively vulnerable college students to an 8-week cognitive behavioral intervention program or an

assessment-only control group. Students were followed for 3 years. Results showed that the students who completed the workshop had significantly fewer depressive and anxious symptoms than did the control group and showed a trend for fewer depressive episodes. Changes in cognitive vulnerability mediated the prevention effect. These results suggest that interventions derived directly from the etiologic chain featured in the cognitive theories are feasible and effective.

After depression

The final point for intervention is after a person has developed depression. Most mental health resources are dedicated to this point of intervention. According to the cognitive theories, depression can be treated by reducing hopelessness (proximal and sufficient cause) or by changing the negative cognitions that lead to hopelessness (distal contributory cause).

If hopelessness causes depression, then creating hopefulness should treat depression. Consistent with this hypothesis, research on hopefulness [35] and the placebo effect [36] suggests that positive expectations for improvement can lead to improvements in depressed mood. Similar to a self-fulfilling prophecy, generating hopefulness for improvement seems to lead to real improvement. Snyder [37] argues that hopefulness is an active ingredient of therapeutic outcomes, regardless of psychotherapy orientation. With regard to the cognitive theories, Taylor and colleagues [38] suggest that cognitive behavioral interventions may be especially effective at instilling hopefulness because they have a clearly articulated treatment rationale and teach patients to set manageable goals.

Increasing hopefulness may help treat depression, but it does not solve the problem of recurrence. After depression remits, distal risk factors (eg, cognitive vulnerability) continue to confer risk for future depression. Treatment interventions need to treat the disorder and the risk factors that may have caused the disorder. According to the cognitive theories, both of these goals can be accomplished by changing a person's cognitions. This hypothesis is the basis for cognitive behavioral therapy.

Cognitive behavioral interventions are among the most effective interventions for depression. A central goal of these interventions is to change the way that people interpret life events. People are taught to identify negative cognitions, evaluate them, and then generate more adaptive cognitions. Research indicates that cognitive behavioral interventions are as effective as medication and have no side effects [39]. The most exciting results show that cognitive behavioral interventions may have a preventative effect [39]. These results are consistent with the etiologic chain featured in the cognitive theories, which indicate that changing cognitive vulnerability should treat depression and create or enhance resilience for future depression.

With regard to the interactions between risk and resilience, an especially interesting question is why there are such significant individual differences in

response to cognitive interventions of depression that are considered to be effective. One possible relevant hypothesis is that people who possess genetic factors for resilience are predisposed to respond better to cognitive intervention (which itself can be viewed as an environmental source of resilience enhancement). For example, genes associated with cognitive control (eg, *COMT*, the gene coding for catechol-o-methyltransferase) may influence which individuals are most likely to successfully master the cognitive techniques taught in therapy (ie, altering negative cognitions).

In summary, the cognitive theories of depression are an example of how a well constructed theory can inform research on risk and resilience. Strong theories of psychopathology can be used to develop and apply prevulnerability, prevention, and treatment interventions. Although further research is needed, results of studies using cognitive interventions are promising. It also is important for future research to develop alternative strategies for changing cognitive risk factors. Changing cognitive vulnerability may not require a cognitive intervention. For example, the strategies used in meditation [40] and mindfulness therapy [41] may mitigate cognitive vulnerability and, in turn, increase resilience for depression.

Risk and resilience: a general framework

Relationship between risk and resilience

There are vast bodies of literature attesting to the importance of the concepts of risk and resilience for studies of psychopathology [42]. As the field now attests, for a point of etiology, risk and resilience factors can be broadly classified as environmental and genetic [43]. There is no clear common position that clarifies the relationships between these two constructs. In fact, the positions range from stating that these concepts are flip sides of the same coin to asserting an etiologic and phenomenologic differentiation between these two concepts. Fig. 2 delineates possible connections between risk and resilience factors. Specifically, the presence or absence of risk and resilience factors can be assumed. In turn, each of the systems of factors (risk and resilience) can be of environmental or genetic etiology. When both systems of factors are present, the psychopathology outcome is determined by main effects and interactions between these factors (the left top quadrant of the figure). When either of the systems is in play (the off-diagonal quadrants of the figure), the psychopathology outcomes are determined by the mechanisms (environmental or genetic, or both in interplay) of risk or resilience systems. Finally, reflective of complex systems of which the development of psychopathology is an example, the lower diagonal quadrant captures the importance of random factors in the development of psychopathology.

Fig. 2 is a simplification. It assumes that risk and resilience are two interactive factors that might have different, overlapping, or common causal

		<i>Resilience</i>			
		<i>Present</i>		<i>Absent</i>	
		E	G		
<i>Risk</i>	<i>Present</i>	E			
		G			
	<i>Absent</i>				

Note:
E = Environmental; G = Genetic

Fig. 2. Theoretical relationships between risk and resilience factors. E, environmental; G, genetic.

mechanisms and might act as independent, correlated, or joint factors. There are two important considerations. First, conceptually, three types of situations might be encountered: (1) risk and resilience factors are present, and the psychopathology (or lack of such) outcome is determined by a balance or imbalance of both forces; (2) risk or resilience factors are present, and the psychopathology (or lack of such) outcome is determined by one of these factors; or (3) risk and resilience factors are absent, and the psychopathology outcomes are attributable to other or random factors. Second, it is assumed that risk and resilience can arise as an outcome of independent, interacting, or overlapping environmental or genetic factors. Thus, the delineation of risk and resilience factors in Fig. 2 permits a discussion of tactics for the prevention and remediation of psychopathology.

How can the cognitive theories of depression be mapped onto the typology presented in Fig. 2? First, these theories are directly related to the quadrants of the figure that assume the presence of risk and resilience factors. It is well accepted that risk factors are of environmental and genetic nature. The occurrence of stressful life events is viewed and classified as an environmental factor. Yet, during the last decade, there have been many studies suggesting that genetic factors may influence risk for depression and cognitive vulnerability [44,45]. Similar statements can be made about resilience factors. Specifically, particular characteristics of cognitive processing can be influenced by environmental effects (eg, positive feedback and positive

cognitions even in the face of failure). Similarly, the existence of protective alleles (eg, similar to those in the *APOE* [apolipoprotein E] gene contributing to risk and resilience to dementia) can be assumed with regard to depression. For example, genes associated with cognitive control (eg, *COMT*) may influence the degree to which individuals can alter their negative cognitions. Thus, all four groups of factors can interact, resulting in the formation of cognitive vulnerability.

Relevance of cultural and cross-cultural research

Much of the research on risk and resilience for psychopathology uses White American samples. Thus, it remains unclear whether the results of these studies apply to other cultures and populations. It is critical to test theories of risk and resilience in other cultures for a number of reasons.

First, as suggested in Fig. 2, risk and resilience factors might have environmental and genetic etiologies. With regard to genetic etiology, it is important to realize that because the development of complex human traits (eg, risk or resilience) is governed by many genes, it is likely that overlapping, but possibly not completely identical, sets of genes will be identified as genetic factors for these traits in different populations. Thus, studying different populations (ie, different ethnic and racial groups) is important for elucidating genetic mechanisms of risk and resilience. Second, even if the same gene is implemented in a particular risk factor (eg, cognitive vulnerability), it is possible that its different alleles² are of importance in different populations. Finally, even if the same alleles are important for the development of a particular trait, their frequencies might vary drastically in different ethnicities, heightening or lowering the prevalence and possibly the magnitude of particular risk or resilience factors in this population. In summary, conducting cross-cultural, cultural, and population research is important for understanding general and specific mechanisms of genetic machinery influencing the development and manifestation of different risk and resilience factors.

Second, two and a half centuries of the existence of psychology as a scientific discipline have resulted in the accumulation of an unequivocal body of evidence attesting to one simple fact: Nothing (no theories and no phenomena) in psychology is general enough to be applied to all cultures without a correction for cultural specificity. Thus, description and content of any risk or resilience factor must be verified, confirmed, and filled with culturally meaningful content when transported from one culture to another. Although the realization of the importance of these studies is not new [46],

² There is a vast amount of literature indicating the involvement of various polymorphisms in a number of genes involved in the function of the neurotransmitter systems such as GABA, glutamate, serotonin, and dopamine.

there is a limited number of them in the field. This consideration is especially important for exploring risk and resilience factors. For example, harsh discipline in one culture (eg, American culture of European origin) might be considered as a risk factor for depression, whereas it might be considered a protective factor in another culture (eg, rural Africa). Similarly, although schooling might be considered a source of resilience in developed countries, it might be associated with a source of disagreement and turmoil in rural African families.

Third, of special interest are considerations regarding manifestations of psychopathology itself. There is a need to calibrate behavioral manifestations of risky and resilient behaviors, examples of negative cognitions, and indicators of depression in different cultures. It has been stated in the literature that the prevalence of depression is distributed unevenly across different countries and different ethnic groups. Behind these uneven distributions might lay genetic and environmental factors.

Summary

Most psychopathology research focuses on understanding the factors that contribute to the onset of mental illness. The general idea is that understanding the causes of psychopathology should lead to better prevention and treatment interventions. Yet, the process of bridging risk and resilience has been slow. In this article, we argue that the transition from risk to resilience could be facilitated by stronger conceptualization of the association between risk and resilience and theory building. Specifically, eliciting typologies of risk and resilience and constructing theories with testable and well defined etiologic chains can be a cornerstone for prevention and intervention for psychopathology. Strong theories of psychopathology can specify how to identify at-risk populations, the time points for interventions, and strategies for creating resilience.

References

- [1] Meehl PE. Theoretical risks and tabular asterisks: Sir Karl, Sir Ronald, and the slow progress of soft psychology. *J Consult Clin Psychol* 1978;46:806–34.
- [2] Murray CJL, Lopez AD. The global burden of disease. Cambridge (MA): Harvard University Press; 1996.
- [3] Greenberg PE, Kessler RC, Birnbaum HG, et al. The economic burden of depression in the United States: how did it change between 1990 and 2000? *J Clin Psychiatry* 2003;62:1465–75.
- [4] Beck AT. *Depression*. New York: Harper and Row; 1967.
- [5] Abramson LY, Metalsky GI, Alloy LB. Hopelessness depression: a theory-based subtype of depression. *Psychol Rev* 1989;96:358–72.
- [6] Popper KR. *The logic of scientific discovery*. London: Hutchinson of London; 1959.
- [7] Depue RA, Slater J, Wolfstetter-Kausch H, et al. A behavioral paradigm for identifying persons at risk for bipolar depressive disorder: a conceptual framework and five validation studies. *J Abnorm Psychol* 1981;90:381–437.

- [8] Abramson LY, Alloy LB, Hankin BL, et al. Cognitive vulnerability-stress models of depression in a self-regulatory and psychobiological context. In: Gotlib IH, Hammen CL, editors. *Handbook of depression*. New York: Guilford; 2002. p. 268–94.
- [9] Alloy LB, Abramson LY, Whitehouse WG, et al. Prospective incidence of first onsets and recurrences of depression in individuals at high and low risk cognitive risk for depression. *J Abnorm Psychol* 2006;115:145–56.
- [10] Garber J, Flynn C. Predictors of depressive cognitions in young adolescents. *Cognit Ther Res* 2001;25:353–76.
- [11] Goodman SH, Gotlib IH. Risk for psychopathology in the children of depressed mothers: a developmental model for understanding mechanisms of transmission. *Psychol Rev* 1999; 106:458–90.
- [12] Gibb BE. Childhood maltreatment and negative cognitive styles: a quantitative and qualitative review. *Clin Psychol Rev* 2002;22:223–46.
- [13] Cole DA, Jacquez FM, Maschman TL. Social origins of depressive cognitions: a longitudinal study of self-perceived competence in children. *Cognit Ther Res* 2001;25:377–95.
- [14] Dweck CS, Davidson W, Nelson S, et al. Sex differences in learned helplessness: II. The contingencies of evaluative feedback in the classroom and III. An experimental analysis. *Dev Psychol* 1978;14:268–76.
- [15] Turk F, Bry BH. Adolescents' and parents' explanatory styles and parents' causal explanations about their adolescents. *Cognit Ther Res* 1992;16:349–57.
- [16] Beck AT, Young JE. Depression. In: Barlow DH, editor. *Clinical handbook of psychological disorders: a step-by-step treatment manual*. New York: Guilford Press; 1985. p. 206–44.
- [17] Bowlby J. Developmental psychiatry comes of age. *Am J Psychiatry* 1988;145:1–10.
- [18] Alloy LB, Abramson LY, Gibb BE, et al. Developmental antecedents of cognitive vulnerability to depression: review of findings from the cognitive vulnerability to depression project. *Cognit Ther Res* 2004;18:115–33.
- [19] Koestner R, Zuroff DC, Powers TA. Family origins of adolescent self-criticism and its continuity into adulthood. *J Abnorm Psychol* 1991;100:191–7.
- [20] Feiring C, Taska L, Lewis M. The role of shame and attributional style in children's and adolescents' adaptation to sexual abuse. *Child Maltreat* 1998;3:129–42.
- [21] Gibb BE, Alloy LB, Abramson LY, et al. History of childhood maltreatment, negative cognitive styles, and episodes of depression in adulthood. *Cognit Ther Res* 2001;25:425–46.
- [22] Rose DT, Abramson LY. Developmental predictors of depressive cognitive style: research and theory. In: Toth DCSL, editor. *Rochester symposium on developmental psychopathology*, vol. 4. Hillsdale (NJ): Erlbaum; 1992. p. 323–49.
- [23] Munoz RF, Le HN, Clarke G, et al. Preventing the onset of major depression. In: Gotlib IH, Hammen CL, editors. *Handbook of depression*. New York: Guilford; 2002. p. 343–59.
- [24] Nolen-Hoeksema S, Girgus JS, Seligman MEP. Predictors and consequences of childhood depressive symptoms: a 5 year longitudinal study. *J Abnorm Psychol* 1992;101:405–22.
- [25] Turner J, Cole DA. Developmental differences in cognitive diatheses for child depression. *J Abnorm Child Psychol* 1994;22:15–32.
- [26] Abela JRZ. The hopelessness theory of depression: a test of the diathesis-stress and causal mediation components in third and seventh grade children. *J Abnorm Child Psychol* 2001; 29:241–54.
- [27] Hankin BL, Abramson LY, Moffitt TE, et al. Development of depression from preadolescence to young adult: emerging gender differences in a 10-year longitudinal study. *J Abnorm Psychol* 1998;107:128–40.
- [28] Nolen-Hoeksema S. *Sex differences in depression*. Stanford (CA): Stanford University Press; 1990.
- [29] Kessler RC. Epidemiology of depression. In: Gotlib IH, Hammen CL, editors. *Handbook of depression*. New York: Guilford; 2002. p. 22–37.
- [30] Gillham JE, Shatte AJ, Freres DR. Preventing depression: a review of cognitive behavioral and family interventions. *Appl Prev Psychol* 2002;9:63–88.

- [31] Clarke GN, Hawkins W, Murphy M, et al. Targeted prevention of unipolar depressive disorder in an at-risk sample of high school adolescents: a randomized trial of group cognitive intervention. *J Am Acad Child Adolesc Psychiatry* 1995;34:312–21.
- [32] Clarke GN, Hornbrook M, Lynch F, et al. A randomized trial of a group cognitive intervention for preventing depression in adolescent offspring of depressed parents. *Arch Gen Psychiatry* 2001;58:1127–34.
- [33] Jaycox LH, Reivich KJ, Gillham J, et al. Prevention of depressive symptoms in school children. *Behav Res Ther* 1994;32:801–16.
- [34] Seligman MEP, Schulman P, DeRubeis RJ, et al. The prevention of depression and anxiety. *Prevention & Treatment* 1999;2.
- [35] Kuyken W. Cognitive therapy outcome: the effects of hopelessness in a naturalistic outcome study. *Behav Res Ther* 2004;42:631–46.
- [36] Kirsch I, Sapirstein G. Listening to Prozac but hearing placebo: a meta-analysis of antidepressant medications. *Prevention and Treatment*, 1 1998;1.
- [37] Snyder CR. Hope theory: rainbows in the mind. *Psychol Inq* 2002;13:249–75.
- [38] Taylor JD, Feldman DB, Saunders RS, et al. Hope theory and cognitive behavioral therapies. In: Snyder CR, editor. *Handbook of hope: theory, measures, and applications*. San Diego (CA): Academic Press; 2000. p. 109–22.
- [39] Hollon SD, Thase ME, Markowitz JC. Treatment and prevention of depression. *Psychological Science in the Public Interest* 2002;3:39–77.
- [40] Davidson RJ, Kabat-Zinn J, Schumacher J, et al. Alterations in brain and immune function produced by mindfulness meditation. *Psychosom Med* 2003;65:564–70.
- [41] Segal ZV, Teasdale JD, Williams MG. Mindfulness based cognitive therapy: theoretical rationale and empirical status. In: Hayes S, Follette V, Linehan M, editors. *Mindfulness and acceptance: expanding the cognitive behavioral tradition*. New York: Guilford Press; 2004. p. 45–65.
- [42] Luthar SS. Resilience in development: a synthesis of research across five decades. In: Cicchetti D, Cohen DJ, editors. *Developmental psychopathology*. vol. 3: risk, disorder, and adaptation. 2nd edition. Hoboken (NJ): John Wiley & Sons, Inc; 2006. p. 739–95.
- [43] Luthar SS, editor. *Resilience and vulnerability: adaptation in the context of childhood adversities*. New York: Cambridge University Press; 2003.
- [44] Egan MF, Goldberg TE, Kolachana BS, et al. Effect of COMT Val 108/158 Met genotype on frontal lobe function and risk for schizophrenia. *Proc Natl Acad Sci U S A* 2001;98:6917–22.
- [45] Berton O, McClung CA, Dileone RJ, et al. Essential role of BDNF in the mesolimbic dopamine pathway in social defeat stress. *Science* 2006;311:864–8.
- [46] Hunter AJ. A cross-cultural comparison of resilience in adolescents. *J Pediatr Nurs* 2001;16: 172–9.