Depression in Children and Adolescents
Linking Risk Research and Prevention

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Abstract: The National Institute of Mental Health has called for translational research linking basic knowledge about vulnerabilities that underlie mood disorders to the development of effective preventive interventions. This paper highlights research about risk factors for depression in children and adolescents and links it to current knowledge about interventions aimed at preventing depression in youth. Basic epidemiologic and clinical research indicates that increased risk for depression is associated with being female; a family history of depression, particularly in a parent; subclinical depressive symptoms; anxiety; stressful life events; neurobiological dysregulation; temperament/personality (e.g., neuroticism); negative cognitions; problems in self-regulation and coping; and interpersonal dysfunction. These vulnerabilities both increase individuals' chances of encountering stress and decrease their ability to deal with the stress once it occurs. Although several existing depression-prevention studies have targeted one or more of these risk factors, the efficacy of these various prevention programs for youth with different combinations of these risk factors needs to be investigated further. Most existing depression-prevention programs in youth have used cognitive–behavioral techniques, with some success. Other depression-prevention strategies have included training in coping, social problem solving, social skills, communication skills, and parenting. A comprehensive prevention program is recommended that includes multiple intervention components, each of which addresses risk and protective factors across different domains and levels of analysis.
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Introduction

The "road map" outlined by the National Institutes of Health[1] calls for translational research that enhances bi-directional communication between basic science and clinical application. New knowledge discovered by basic researchers then should be used to develop interventions that can prevent or reduce the suffering of individuals with mental disorders. In turn, clinical trials testing the efficacy of these interventions can be used to provide further insights into the mechanisms underlying and maintaining the disorders.

Regarding depression in particular, the National Institute of Mental Health (NIMH) Strategic Plan for Mood Disorders Research[2] stated that research that provides a "detailed account of how cognitive, behavioral, and affective vulnerabilities influence the onset and prolongation of mood disorders can contribute to the development of effective preventive interventions." In addition, prevention trials can "provide an opportunity to test theories regarding the mechanisms that lead to onset and the strategies that avert it." Thus, developmental theory and basic research should guide the design of prevention trials, which then should generate results that can be used to inform and revise the theory.[3]

The distinction between risk factors and causal risk mechanisms[4–6] is relevant to the present discussion. Risk factors are antecedents that increase the probability of an outcome over the population base rate. They do not, however, explain the processes through which these factors influence the likelihood of the condition. In contrast, risk mechanisms describe the intervening paths that link the risk factor to the outcome of interest. Altering these mechanisms will affect the likelihood of the condition. Indeed, these are the processes that interventions aim to affect. In addition, fixed markers are risk factors that are not considered changeable (e.g., gender, genotype), although they may influence more proximal risk factors that can be altered (e.g., responses to stress, levels of neurotransmitters). Variable markers change (e.g., age) or can be changed.[5] With regard to depression in particular, potential risk factors include such fixed markers as gender[7,8] and genes,[9,10] and such variable markers as parental depression,[11,12] anxiety,[13,14] subsyndromal levels of depressive symptoms,[15,16] neurobiological dysregulation,[17,18] temperament/personality,[19,20] negative

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cognitions,[21,22] problems in self-regulation and coping,[23] stressful life events,[24,25] and interpersonal difficulties.[26,27] These variables have been particularly linked with subsequent depression, although this list is not exhaustive.

Depression has a complex, multifactorial causal structure. Therefore, it is unlikely that any one risk factor will explain its development, nor will reducing the chances of the occurrence of a single risk factor be sufficient to prevent depression. Rather, it is more likely that the accumulation[28,29] and/or interaction among multiple risk factors[30,31] will lead to depression.

The present paper highlights basic research findings on depression in youth to identify who is at risk and therefore should be targeted for prevention, and notes potential mechanisms of risk, which can inform the content of such programs. Studies testing the efficacy of depression-prevention programs have varied with regard to which, if any, of these risk factors they have targeted. Appendix A outlines several risk factors, basic findings, and relevant prevention programs. Appendix B provides descriptive information about these prevention programs categorized by the populations to whom the interventions were directed.[32] Whereas universal preventive interventions are administered to all members of a target population, selective programs are provided to a subsample who are at above average risk, and indicated prevention is given to individuals who manifest subclinical signs or symptoms of the disorder. (For a review of depression-prevention studies in children and adolescents, see a recent meta-analysis.[33])

Gender

Being female is significantly associated with depression in adolescents and adults. Prior to adolescence, the rate of depressive disorders is about equal in girls and boys,[34,35] or even higher among boys[36,37]; during early to middle adolescence, the rate of depressive symptoms and disorders in girls rises to two to three times that of boys.[7,38] Explanations for this gender difference have included hormonal changes, increased stress, differences in interpersonal orientation, tendencies toward rumination and other maladaptive responses to stress, and different socialization experiences.[8,39] A better understanding of the reasons underlying gender differences in rates of depression is needed to guide the specific content of programs aimed at preventing it.

An important distinction needs to be made here between gender differences in risk versus response to interventions. Although female adolescents are at greater risk than their male peers, this does not necessarily mean that males and females will respond differently to preventive interventions. Some of the apparent gender effects in prevention studies, however, could be due to the fact that the rate of depression in a no-intervention control group will be greater for females than males, and therefore the effect of the intervention may appear to be stronger for females. Thus, the higher rate of depression in females is relevant to prevention research in so far as the likelihood of depression in the no-intervention group will be higher for females than males. Significant group by gender interactions could be due to either within-gender effects of the intervention or within-intervention effects for gender. For example, girls and boys may respond similarly to an intervention, but girls in the control group may show an increase in depression (Figure 1). Alternatively, boys could do better than girls in the intervention, and there could be no difference within girls as a function of condition (Figure 2).

What have prevention studies found with regard to gender differences? Whereas some programs have re-

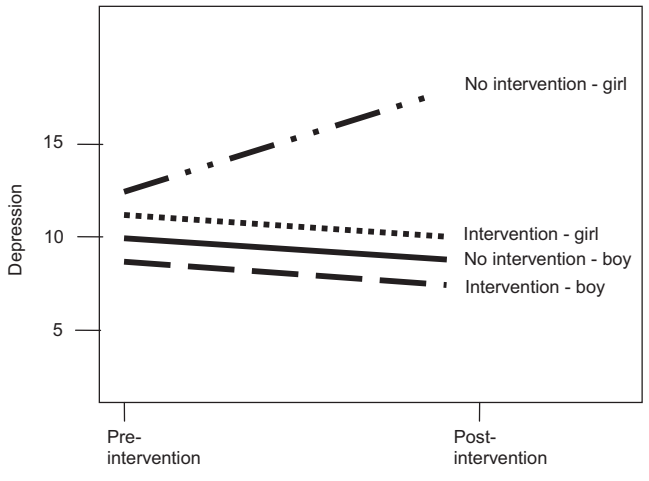

Figure 1. Within-gender effects of the intervention: girls and boys in the intervention group were not significantly different, but girls in the control group had significantly higher post-intervention depression scores than girls in the intervention group.

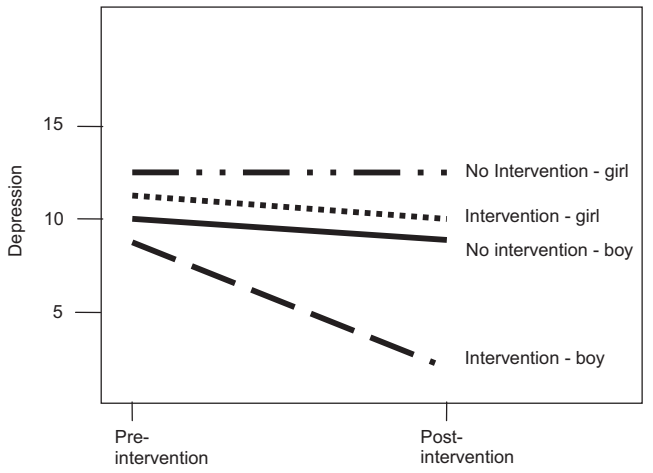

Figure 2. Within-intervention effects for gender: boys in the intervention group had significantly lower depression scores than girls in the intervention; there were no significant difference for girls as a function of condition.

ported that girls respond better than boys, others have found the reverse, and still others have found no gender differences. Using a behaviorally based program, Clarke et al.[40] found short-term improvement in depressive symptoms for boys, but not for girls. They suggested that such a mechanistic approach may have been more in line with the natural coping style of boys than girls. Similarly, two school-based universal interventions designed to prevent depression by improving school achievement and mastery learning were found to be more effective for boys than for girls.[41,42]

Researchers at the University of Pennsylvania examined gender differences in the effectiveness of the Penn Optimism Program (POP) and the Penn Enhancement Program (PEP), and found that POP was effective for boys but not girls, and PEP was more effective for girls than boys.[43,44] POP is a cognitive–behavioral, skills training program that involves a logical and systematic approach to dealing with negative emotions, and may appeal more to boys than girls. PEP, on the other hand, is a more social approach in which participants are encouraged to share feelings and experiences, which may better suit girls, who tend to value the sense of interpersonal connection and social support.[39]

Consistent with this view, Forsyth[45] tested the efficacy of a prevention program that was based on interpersonal theory, and found a substantial effect size for the program at post-treatment and at 3-month follow-up. Because Forsyth's[45] sample was almost entirely female, however, it is not possible to know from this study whether this interpersonal program would have worked equally as well for males. Nevertheless, these findings show that an interpersonal approach was effective for females.

Other studies using cognitive–behavioral approaches, however, have found different gender effects. The Penn State Adolescent Study[46] showed improvement among girls but increased symptoms among boys at post-treatment, although these effects were no longer present at the 6-month follow-up. In a primary care setting, girls in the Penn Resiliency Program had significantly reduced depressive symptoms compared to girls in the usual care control condition, whereas there was no such difference for boys as a function of condition.[47] Similarly, in a college student sample, Seligman et al.[48] found that females benefited more than males in their cognitive–behavioral program, although this difference could have been due to a floor effect for the male sample.

Gender differences also were found in a study of the Family Bereavement Program, which taught children positive coping, stress appraisals, control beliefs, and self-esteem, while the surviving parent learned discipline skills and how to improve the quality of the parent–child relationship.[49] They reported that the rate of recovery was significantly better for girls in the intervention compared to the control group. For boys in both conditions, there was a significant decrease in symptoms over time, particularly for those receiving the intervention.

Thus, results of studies comparing the outcome of prevention programs for girls versus boys are inconsistent. Important questions about gender difference remain regarding both the content and structure of interventions for preventing depression. Specifically, do girls benefit more from an interpersonal approach, whereas boys respond better to a cognitive–behavioral intervention? A recent prevention study that directly compared a cognitive–behavioral versus a program based on interpersonal psychotherapy (IPT) found that both programs were significantly more effective than a no intervention control group in reducing depressive symptoms, but no significant effects were found for gender.[50] Cognitive interpersonal models of depression[27] would suggest that programs that incorporate and integrate both approaches should be effective for females and males alike, with greater emphasis placed on that which best suits the particular individuals in the group and culture.

What are the implications of these findings for the design and structure of depression-prevention programs? Should only girls be targeted for intervention? Certainly if the goal is to maximize limited resources, then girls clearly are at greater risk. However, boys do get depressed as well, and if depression serves as a precursor for other kinds of problems (e.g., substance use, oppositional defiant disorder),[51] then boys also should have access to depression-prevention programs, particularly when there is evidence that they benefit from them.[40–42]

Should same-sex groups be used? Whereas girls may feel more comfortable in female-only groups, boys actually may benefit more from the presence of girls, because boys may be more inclined to behave appropriately around them; boys also may learn by modeling girls' pro-social and empathic behavior. Preliminary evidence indicates that girl-only groups were better than co-ed groups in reducing girls' hopelessness, but were similar to co-ed groups in reducing depressive symptoms.[52] Co-ed groups were found to decrease depressive symptoms in both girls and boys. Moreover, the possibility of iatrogenic effects of male-only groups as has been suggested for conduct-disordered boys[53] needs to be examined with regard to depression.

Thus, females are at greater risk for depression than males, but there is no conclusive evidence that one gender consistently responds better to depression-prevention programs than the other. Although several explanations for the greater risk in females have been suggested,[8,35,39] depression-prevention programs have not yet been designed to address specifically these speculations. The translation of basic knowledge about the processes that account for gender differences in rates of depression to the actual content of prevention

programs remains an important direction for future studies.

Genes

Depression clearly is familial,[11,54] but family data alone cannot distinguish environmental from genetic causes of the transmission of depression across relatives. Family, twin, and adoption studies have provided evidence of both genetic and environmental effects for unipolar depression.[9,10] Estimates of heritability of depressive symptoms tend to be moderate, although these estimates vary as a function of informant, age, and severity of depressive symptoms.[55,56] Nonshared environmental effects, that is, experiences that are unique to individuals within a family, also tend to be moderate.[56] Twin studies indicate that depressive symptoms in general population samples of twins are significantly heritable. That is, the proportion of variance explained by additive genetic factors has been reported to be between 40% and 70%.[57,58]

Genetic influences have been found to vary with age, with some studies[59–61] reporting a decrease in heritability with increasing age, and several other studies finding the opposite.[62–66] Indeed, Scourfield et al.[64] asserted that "[t]he weight of evidence . . . supports the importance of early shared environmental influences on depression scores in younger children and adolescents, with these influences being replaced by new genetic and unique environmental influences as children grow older."

If the relative influences of environmental and genetic factors on depression are different in children and adolescents, then the types of interventions used to prevent depression at different ages likely will need to vary. More basic research aimed at identifying the developmental processes underlying these age differences is needed to inform the construction of age-appropriate preventive interventions. In particular, **what** is inherited that places individuals at risk at different ages, and through what processes does this occur?

Prevention programs may affect gene products or expression,[67] or may interrupt gene–environment interplays.[68,69] Genes likely contribute to neurobiology, personality, and self-regulation, which then interact with the environment to produce symptoms. For example, genes may indirectly affect depression through influencing children's sensitivity to negative life events.[69] Thus, when confronted with stress, an individual's inherited level of reactivity likely will contribute to the extent of their distress. Caspi et al.[70] showed that a functional polymorphism in the promoter region of the serotonin transporter (5-HTT) gene moderated the effect of stress on depression. Depression was significantly more likely to occur after experiencing stressful life events for individuals with one or two copies of the short allele of the 5-HTT promoter polymorphism compared to those who were homozygous for the long allele. Prevention programs could utilize information about familial transmission to help family members better understand the condition and its consequences,[71] as well as teach strategies for managing the stressors that interact with this potential liability.

Offspring of Depressed Parents

Parental depression is one of the strongest risk factors for depression in children, which likely is the result of both genetic and environmental influences.[11,12] Compared to children of nondepressed parents, offspring of depressed parents are about three to four times more likely to develop a mood disorder,[72] and are at increased risk for high levels of medical utilization, other internalizing disorders, behavior and school problems, suicide attempts, substance abuse disorders, and lower overall functioning.[73,74] Thus, children of parents with affective disorders are a logical and critical population to target for prevention.

Several selective programs have been developed to prevent depression in the offspring of depressed parents. Beardslee et al.[71] targeted nonsymptomatic children of parents with an affective disorder. Their "control" intervention consisted of two 1-hour lectures educating groups of families about depression. Their "active" clinician-facilitated program used psychoeducational techniques focusing on increased understanding within the family, education about mood disorders, and promotion of resilience-related behaviors and attitudes in the children through enhanced parental and family functioning. Participants in the clinician-facilitated program reported greater understanding of parental affective disorder and had better adaptive functioning compared to those in the control condition. The programs did not differ, however, regarding children's depressive symptoms assessed at the 18-month follow-up or internalizing symptoms at the 2.5-year follow-up.[75]

Another prevention study that specifically targeted offspring of depressed parents was conducted by Clarke et al.[76] This was an indicated as well as a selective sample such that adolescents themselves had subclinical levels of depressive symptoms. This 15-session prevention program focused on cognitive restructuring techniques to challenge unrealistic and/or negative thoughts, and was modified specifically from an earlier program[77] to address beliefs related to having a depressed parent. Clarke et al.[76] found that adolescents in the prevention group had significantly fewer depressive diagnoses at a 12-month follow-up compared to the control group. This effect persisted at a diminished level at 18- and 24-month follow-up assessments.

Two other randomized controlled depression-prevention trials currently are being conducted. One mul-

tisite investigation (NIMH grants R01MH064735, R01MH064541, R01MH064503, R01MH064717) is a replication and extension of the study by Clarke et al.[76] involving a revised configuration of the intervention (i.e., eight weekly acute sessions and six monthly continuation sessions) in order to try to obtain a more enduring effect. In the second prevention study (NIMH grant R01MH069940), families with a depressed parent participate in a multifamily-focused cognitive intervention that teaches coping to the children and parenting skills to the adults.

Finally, a more-distal prevention strategy has involved the reduction of depression in parents with the expectation that the negative effects of their condition on their children will diminish with remission, and thus the benefits will "trickle down" to their children. The premise underlying this approach is that if parental depression contributes to children's psychopathology, at least partially through parents' behavior while depressed, then reducing their depression should diminish its negative effects on their offspring. Preliminary evidence consistent with this hypothesis recently has been reported in three different samples.[78–80] Garber et al.,[78] for example, showed that remission of parental depression predicted a significant decrease in children's depressive symptoms and was associated with improvements in family functioning.

Thus, targeting offspring of depressed parents is a logical and promising direction for prevention programs. Some of the factors that have been found to increase high risk children's vulnerability for depression, such as dysfunctional parent–child relationships[81,82] and negative cognitions,[83–85] have been the focus of the preventive interventions. Programs that combine several of these risk factors are likely to be particularly effective.

Subsyndromal Depression

Subsyndromal levels of depressive symptoms significantly increase risk of having a full major depressive episode in adults,[15,16] adolescents,[86,87] and children.[88] For example, in a prospective study, Pine et al.[87] showed that a difference of two standard deviations from the mean in depressive symptoms predicted a two- to three-fold greater risk of an episode of major depression in adulthood; the symptoms of anhedonia and thoughts of death were particularly predictive of subsequent depressive episodes. Subsyndromal levels of depression also can have a negative impact on academic/occupational and interpersonal functioning.[89] Thus, targeting individuals with subsyndromal depression for preventive intervention may be justified not only as a means of reducing risk of future major depressive disorder (MDD), but also as a way to ameliorate existing levels of distress and dysfunction.[90]

Several prevention studies have targeted such indicated samples of children and adolescents with subsyndromal depressive symptoms. Clarke et al.[76] found evidence of the efficacy of their cognitive–behavioral program in a sample of adolescents with subsyndromal depressive symptoms who also were offspring of depressed parents. In an earlier study involving 9th- and 10th-grade students with depressive symptoms, Clarke et al.[77] examined the efficacy of their cognitive–behavioral program that met three times a week for 5 weeks, and found lower levels of depressive symptoms at post-treatment, although not at the 6- or 12-month follow-up.

The Penn Prevention Programs also have been evaluated with subclinically depressed youth. Children in the intervention condition had significantly lower levels of depressive symptoms compared to controls at post-treatment and at 6-month follow-up assessments over 2 years.[91,92] Although targeting youth who are already showing subclinical levels of depressive symptoms straddles the line between prevention and treatment,[93] given the seriousness of the dysfunction often experienced by these children, they are an important group for whom intervention should be provided.

Anxiety

Anxiety is the most common comorbid disorder with depression, with estimates ranging from 30% to 75% in preadolescents and between 25% and 50% in adolescents.[13,51,94] There also is increasing evidence that anxiety precedes the onset of mood disorders and thus might be a risk factor for depression.[94–97] Therefore, children with anxiety also should be targeted for depression-prevention programs.

Two studies have explicitly examined anxiety as a potential moderator of the effects of programs for preventing depression. Lowry-Webster et al.[98] tested a universal cognitive–behavioral program known as FRIENDS, which was designed to prevent anxiety as well as depressive symptoms. Although they found no overall effect on the rates of depressive disorder, they did find a reduction in depressive symptoms for those children who were clinically anxious at pre-test. Similarly, Hains and Ellmann[99] tested the efficacy of a stress inoculation training program for children with different levels of emotional arousal at baseline. The program significantly lowered depressive symptoms for children with high emotional arousal, but not for those with low emotional arousal. Consistent with these prevention findings are the results of a treatment study showing that depressed adolescents with comorbid anxiety had greater decreases in depressive symptoms in response to a cognitive–behavioral treatment than did depressed adolescents without comorbid anxiety.[100] In all of these studies, however, the positive results for the subgroup with anxiety may have been

attributable to higher levels of depressive symptoms that occur in the context of anxiety, rather than the effect of anxiety symptoms per se. Future prevention studies should explore whether reducing anxiety in children with different baseline levels of depressive symptoms actually decreases the risk of future depression. Given the high degree of comorbidity of anxiety and depression, basic research should continue identifying risk factors that these conditions have in common, and prevention programs should aim to reduce both anxious and depressive symptoms by specifically targeting their shared risk processes. The strategy of preventing depression by explicitly reducing anxiety in children and adolescents has yet to be adequately tested.

Neurobiology

Psychobiological studies of depression in youth have focused on dysregulation in neuroendocrine and neurochemical systems, sensitization of biological stress mechanisms, and disturbances in sleep architecture.[17,101,102] Studies of growth hormone, prolactin, and cortisol levels after pharmacologic stimulation in currently depressed, remitted, and at-risk youth have shown in all three groups abnormalities in the secretion of these hormones, such as blunted growth-hormone secretion after the administration of growth-hormone–releasing hormone.[101,103,104] Thus, alteration in certain hormonal systems may be trait markers for MDD.[105]

There also is preliminary evidence of functional and anatomic brain differences in depressed and at risk children compared to normal controls.[106] Studies of brain activity asymmetry, for example, have found left frontal hypoactivation in infant[107,108] and adolescent[109] offspring of depressed versus nondepressed mothers. Decreased left frontal activation presumably reflects an under-activation of the approach system and reduced positive emotionality, which also may be a vulnerability marker for depression.[110]

Results of studies of other biological systems (e.g., the hypothalamic–pituitary axis, sleep electroencephalogram) are inconsistent and vary as a function of age, gender, maturation, severity, psychiatric family history, and exposure to stress.[17,101] The extent to which these biological factors predict the onset of MDD alone or in interaction with psychosocial risk factors needs to be explored.

What are the implications of this neurobiological research for prevention? Identifying markers of risk can be important for targeting the most vulnerable children for intervention. In addition, studies that track neurobiological changes as a function of intervention can be used to understand the processes underlying improvement in the disorder. Thus, assessing neurobiological risk factors before, during, and after preventive interventions, and examining the temporal relations among changes in biology, cognitions, affect, and behavior may be informative about how these different levels of analysis interact to produce and maintain depression.

An intriguing example of the integration of basic brain science and clinical interventions relevant to prevention is the recent finding by Davidson et al.[111] that adults who completed a short program in mindfulness meditation compared to a wait-list control group had significant increases in left-sided anterior activation, which is presumably associated with positive affect. Davidson et al.[111] concluded that prefrontal activation asymmetries are plastic and can be shaped by training. Moreover, Davidson et al.[112,113] have argued that left-sided anterior activation is associated with more adaptive responding to negative and/or stressful events, and individuals with greater left-sided anterior activation tend to show faster recovery after a negative provocation. Is it possible that interventions such as mindfulness meditation could be used preventively to alter high-risk adolescents' left-sided anterior activation and associated responses to stress?

Although most depression-prevention programs likely do not directly aim to produce neurobiological changes, it is becoming increasingly feasible and important to assess neurophysiology, and therefore future prevention trials should include such measures in their assessment batteries. In so doing, prevention studies not only can have practical benefits of reducing depression, but also can be used to obtain basic knowledge about some of the biological processes underlying the disorder.

Temperament/Personality

Temperament is thought to have a genetic/biological basis, although experience and learning, particularly within the social context, also can influence its development and expression,[114] and therefore interventions may be able to affect trait expression. Negative emotionality, the propensity to experience negative emotions, is conceptually related to negative affectivity,[115,116] neuroticism,[117] the behavioral inhibition system,[118] stress reactivity,[119] "difficult temperament,"[120] behavioral inhibition,[121] and harm avoidance.[122] Negative emotionality reflects a sensitivity to negative stimuli, increased wariness, vigilance, physiologic arousal, and emotional distress. In contrast, positive emotionality is characterized by sensitivity to reward cues, approach, energy, involvement, sociability, and adventurousness, and is associated with positive affectivity,[116] extraversion,[117] the behavioral activation system,[118] activity and approach,[120] and novelty seeking.[122]

According to the tripartite model of anxiety and depression, depression is characterized by high levels of negative affectivity and low levels of positive affectivity.[115,116] Support for this model has been found in

children.[123–126] Longitudinal studies have shown that children with inhibited, socially reticent, and easily upset temperament at age 3 were significantly more likely to have elevated rates of depressive disorders at age 21 than were those without such a temperament.[127] In addition, another childhood temperament, apathy (i.e., lack of alertness) as rated by physicians when children were aged 6, 7, and 11 years, has been reported to predict adolescent mood disorders and chronic depression in middle adulthood.[128] "Difficult temperament, " which is characterized by inflexibility, low positive mood, withdrawal, and poor concentration also has been found to correlate with depressive symptoms both concurrently and prospectively in adolescents.[129]

How are temperament/personality and depression related? Potential mediators of the association between neuroticism and depression, particularly under condition of stress, have included appraisals, expectations, and coping.[23,130,131] Negative affectivity leads to greater emotional arousal, negative appraisals, difficulty modulating emotional reactivity to stress, a greater likelihood of using avoidance coping, and an increased probability of experiencing stress.[20,132] A common third variable (e.g., genes, neurobiological dysregulation, stress) also may partially explain the association between negative affectivity and depression.

Depression-prevention programs for youth generally have not focused on negative and positive emotionality or emotional regulation in particular. Given the burgeoning interest and research on emotion regulation in neuroscience,[112,133] developmental,[134,135] and clinical science,[136,137] more work needs to be done linking such basic research on emotion regulation to interventions for preventing depression. At what age can children begin to learn how to actively and deliberately regulate their emotions, and what are the most developmentally appropriate and effective methods for teaching children such skills?

Therapeutic interventions for adults that have emotion regulation as a central feature include mindfulness meditation[111,137] and dialectical behavior therapy (DBT).[136] DBT has been modified for use with depressed adults,[138] suicidal adolescents,[139] and incarcerated female juvenile offenders.[140] Group DBT for adult depression focuses on mindfulness, interpersonal effectiveness skills, and distress-tolerance skills to endure suicidal ideation, strong negative emotions, distressing memories, and stressful situations, rather than engaging in impulsive or harmful behaviors. Emotion-regulation skills, in particular, include identifying and labeling emotions, understanding their functions, differentiating between when to accept an emotion and when to attempt to change it, and strategies for changing emotions. Lynch et al.[138] found that medication plus DBT was significantly better than medications alone in reducing self-reported depressive symptoms for older adults. The extent to which a DBT approach can be used to prevent depression, particularly with adolescents is worth exploring.

Negative Cognitions

Cognitive theories of depression assert that when confronted with stressful life events, individuals who have negative beliefs about the self, world, and future, and make global, stable, and internal attributions for negative events will appraise stressors and their consequences negatively, and hence are more likely to become depressed than are individuals who do not have such cognitive styles.[21,22] A growing convergence of evidence from correlational, predictive, and offspring studies supports the idea that negative cognitions may be a vulnerability to depression.[141–143]

Prospective studies in children and adolescents have shown that a range of negative cognitions including low global self-worth, perceived incompetence, and negative explanatory and inferential style predict increases in depressive symptoms, often in interaction with negative life events.[144–146] Developmental theorists[147,148] have suggested that negative cognitions emerge over time and their relation with depression increases with development. Indeed, the association between negative cognitions and depressive symptoms has not been found to be as strong in young children as it is in older children and adolescents.[144,149] Thus, even though there is a concurrent and predictive relation between negative cognitions and depression in children and adolescents, some have questioned whether negative cognitions are a concomitant or consequence of depression rather than part of the causal chain.[150,151]

Therapy approaches based on Beck's[22] cognitive model of depression generally have found that cognitive–behavioral therapy (CBT) in adults[152] and youth[153,154] can reduce depressive symptoms. Randomized controlled treatment trials conducted with depressed youth have shown that CBT is significantly better than no intervention, but is sometimes comparable to other psychosocial treatments (e.g., interpersonal psychotherapy, relaxation training).[155] A recently completed multisite treatment trial with depressed adolescents found that fluoxetine plus CBT was superior to medication alone, which was better than CBT and placebo, which did not differ significantly.[156] Concerns about the overly structured CBT manual and the inexperience of some study therapists have led some to question the initial CBT results in the Treatment for Adolescents with Depression Study (TADS),[157] however.

Given these findings from both basic research and clinical interventions, the majority of depression-prevention programs have made cognitive restructuring a central component of the intervention. A recent meta-analysis[33] of investigations of depression-preventive interventions for children and adolescents showed that

19 of the 30 studies had cognitive–behavioral techniques such as cognitive restructuring, problem solving, assertiveness training, and cognitive coping strategies as primary features of their prevention programs. Eight of these studies tested some form of the Penn Prevention Program,[91] which teaches children about the links between thoughts and feelings, how to generate possible explanations for negative events, and how to use evidence to choose the most plausible explanations for these events. In general, studies using the Penn cognitive–behavioral intervention have shown significantly lower levels of depressive symptoms in the intervention group both at post-treatment and at 6-month follow-up assessments through 2 years.[91,92] Similarly, studies by Clarke et al.[76,77] of cognitive–behavioral programs for preventing depression in adolescents have shown positive results. Indeed, Clarke et al.[76] is one of the few prevention studies to show a significantly lower rate of clinician-assessed MDD and not just a change in self-reported depressive symptoms.

Thus, research on cognitive–behavioral preventive interventions is far enough along that it is now time to address additional questions, although other approaches might need further development and testing before considering the following issues:

1. What are the most important and active ingredients in cognitive–behavioral preventive interventions? Some treatment studies of depressed adults have found that the behavioral and cognitive components were equally effective by themselves.[158,159] Can the behavioral activation intervention be developed into an effective prevention program, and can it be implemented successfully with adolescents?
2. Although cognitive–behavioral interventions presumably "work" because they teach participants new ways of thinking, what is the evidence that this really is the mechanism of change? Few prevention trials in youth have measured whether the interventions actually affected the hypothesized mediators, and therefore it is premature to conclude that cognitive restructuring was the process responsible for the preventive effect. Are the targets of the intervention changed by it, and if so, do these changes lead to a decreased likelihood of the disorder? Such findings do not necessarily tell us about the processes that cause depression, but they can demonstrate mechanisms that may reduce the probability of onset. It also is possible, however, that a program can affect the hypothesized mediator, without appearing to influence depression. The effect of these mediators may take more time to carry over to depressive symptoms, and therefore, a longer follow-up might be needed. Finally, another alternative is that the putative mediator does not have an effect on the outcome at all.
3. For whom are cognitive–behavioral interventions most effective? Studies need to examine whether selecting adolescents with particularly negative cognitive beliefs for cognitive–behavioral interventions enhances or diminishes the success of the cognitive–behavioral program. There is some evidence that matching intervention techniques with related individual characteristics leads to better outcomes,[160,161] although other studies have found the reverse to be true.[162,163] Sotsky et al.,[163] for example, found that contrary to expectation, low social dysfunction predicted superior response to interpersonal psychotherapy, and low levels of negative cognitions predicted superior response to cognitive–behavior therapy. Thus, those individuals who were the least characterized by the kinds of problems targeted in the interventions did better in those particular treatments. This "matching" question should be explored with respect to preventive interventions in adolescents.
4. How does development affect these interventions? At what point in development are children most likely to benefit from a cognitive restructuring approach? How do current cognitive–behavioral programs need to be modified to make them developmentally appropriate in terms of content and structure? Basic findings that negative cognitions emerge over time and become increasingly associated with depression with development[144,149] can be used to inform the extent to which various cognitive strategies can be used successfully with children at different ages.
5. How can cognitive–behavioral prevention programs be enhanced or supplemented to increase the duration of their effects? This can be divided further into a parametric and a substantive question. What is the ideal number of sessions, session length, and intervention duration needed to maximize the likelihood that the skills will become permanent? What is the best number and timing of booster sessions? Who are the best people to deliver the intervention (e.g., teachers, clinicians)? Second, are there additional strategies that can be included in the prevention program that complement the cognitive–behavioral skills and address other important risk factors such as interpersonal communication and emotion regulation?

Stress

Considerable empirical evidence exists of a link between stressful life events and depression in children and adolescents.[25] The stress exposure model posits that individuals who have experienced stress will be more likely to become depressed than those who have not.[164] Support for this model has been provided by prospective studies showing that stress temporally precedes increases in depressive symptoms,[165,166] and the

onset of depressive disorders in youth.[167,168] Interestingly, the association between stress and depression grows stronger in adolescence, particularly in girls.[165,169,170]

The stress-generation model suggests that depressed individuals contribute to negative events through their own behavior.[171,172] Longitudinal studies have shown support for this model, particularly with regard to interpersonal relationships.[173,174] Factors that might contribute to the generation of stress include personality,[175,176] lack of interpersonal competence,[173,177] and comorbid psychopathology.[172,174]

The reciprocal model essentially puts the two previous models together and highlights the "vicious cycle" that can occur between depression and stress. Depressive symptoms at one time predict stressors at a later time, and similarly, previous stressors lead to subsequent symptoms. Support for this reciprocal model has been found in a few studies of adolescents.[178,179]

Although no one specific type of stressful event invariably leads to depression, certain negative life events consistently have been found to be associated with depression including personal disappointments, failures, and losses,[164,180,181] child abuse/maltreatment, especially for women,[182–184] poverty,[185,186] and interpersonal problems, particularly those of their own making.[172,187] Thus, stressful life events clearly are linked with depression, although there are important individual differences in both genetic and psychological vulnerabilities that contribute to differential reactions to stress.

Despite the central role of stress in producing depression, particularly among those with biological and/or psychological diatheses, prevention programs for youth generally have not made stress management, per se, a primary focus. In one small trial of a preventive intervention modeled after a stress inoculation training program, high school students were taught to identify stressful events, their negative cognitions in response to these events, and their physiologic responses to stress.[99] Then they were instructed in cognitive restructuring, anxiety management, and problem-solving skills. The intervention had a significant effect on depressive symptoms at post-intervention, although not at follow-up 2 months later.

Other programs have included stress management as a part of the larger intervention (see Responses to Stress section for a discussion of these programs). Besides the Family Bereavement Program,[188] studies of prevention programs have not examined actual decreases in levels of stress as a function of the intervention. Thus, despite the prominent role of stress in the onset and exacerbation of depressive symptoms, most prevention programs have not made stress reduction, per se, a central feature. Cognitive–behavioral interventions teach strategies for changing cognitions in reaction to stress and for coping with negative events, as well as encourage engaging in pleasant activities to distract attention from the negative thoughts about the stress. No depression-prevention program in youth, however, has explicitly addressed the role of a person's own behavior in the generation of stress. Although clinicians need to be cautious not to "blame" adolescents for the stressors they encounter, therapists should help teens examine their potential role in generating negative events and provide them with strategies for minimizing this in the future. Given the evidence of individual differences in the extent to which adolescents make "mature" decisions,[189] however, some teens might not be cognitively ready to learn how to avoid producing stress.

Responses to Stress

How individuals respond to stress can significantly affect their future adjustment and psychopathology. Several perspectives on coping with stress in children and adolescents have been suggested.[135,190,191] Earlier theories differentiated between problem-focused and emotion-focused coping.[192] Whereas problem-focused coping involves responses that act on the source of stress, emotion-focused coping involves attempts to palliate negative emotions that arise from a stressful event through such responses as expressing one's emotions, seeking solace and support from others, and trying to avoid the source of stress. This dimension has been widely criticized, however, because it is overly broad and includes some forms of coping that have been found to be associated with dysfunction.[23,193]

More recently, Compas et al.[191] proposed that responses to stress can be distinguished as voluntary or involuntary and engaged versus disengaged. Involuntary or automatic reactions are in part a reflection of individual differences in temperament, particularly stress reactivity. Coping is a subset of broader self-regulatory processes that involves volitional and intentional responses to stress. Engagement coping includes problem solving, cognitive restructuring, positive reappraisal, and distraction, whereas disengagement responses are avoidance, self-blame, emotional discharge, and rumination.

In their review, Compas et al.[23] concluded that the majority of studies have found that higher levels of engagement coping and problem-focused coping are associated with lower levels of internalizing symptoms. In contrast, disengagement coping, involuntary engagement coping, and emotion-focused coping tend to be related to higher levels of internalizing symptoms. In particular, responses to stress that have been associated with worse adjustment include cognitive and behavioral avoidance, social withdrawal, resigned acceptance, emotional arousal, ventilation or discharge, wishful thinking, self-blame or self-criticism, intrusive thoughts, and rumination.

Most of this research has been cross-sectional, however, thus limiting our ability to draw conclusions about the direction of the coping–depression relation. Although coping can lead to reductions in emotional distress, it also is plausible that various levels of emotional distress can lead to different kinds of coping responses. That is, children who are less depressed and possibly more competent may be better at generating solutions to problems and maintaining a positive outlook when faced with stress. Few prospective studies of the association of coping and depression after controlling for previous depression have been conducted. Laboratory studies that experimentally manipulate coping in a controlled setting could further our understanding of basic coping processes. In addition, randomized intervention trials designed specifically to enhance coping skills can be particularly informative about the role of coping in reducing depression.

One intervention trial[194] with mild to moderately depressed children tested an eight-session coping enhancement program based on a two-process model of control beliefs.[190] The intervention focused on primary control (changing objective conditions through activity selection and goal attainment) and secondary control (changing one's thoughts to buffer the impact of objective conditions). The treatment group had greater reductions than the control group on self- and clinician-rated depressive symptoms at post-treatment and 9 months later.

Several prevention programs that involve a significant coping component have been developed and tested. To some extent, programs that train cognitive restructuring and problem-solving implicitly teach various forms of coping. Programs that have explicitly included a coping component are: (1) the Resourceful Adolescent Program,[195] which promoted self-management and calming skills in response to stress as well as cognitive restructuring and problem-solving skills; (2) the Penn State Adolescent Study[46] taught emotional, cognitive, and behavioral strategies for responding adaptively to stress including emotion-focused coping, generation of alternative solutions to problems, relaxation, anticipating consequences, and problem solving; (3) the Penn Prevention Programs[43,91] used cognitive–behavioral techniques to teach coping strategies including goal setting, generating alternatives for action, and decision-making and social problem-solving methods such as distancing from stress, distraction, and relaxation to cope with family conflict; and (4) the FRIENDS family CBT program[196] taught children physiologic, cognitive, and behavioral coping strategies.

Other prevention programs taught coping with specific kinds of stressors including parental divorce,[197,198] parental alcoholism,[199] and bereavement.[188] The advantage of these programs is that they can tailor the intervention to the specific needs and concerns relevant to the particular stressor. In a program for children of alcoholics, Roosa et al.[199] taught children situation-specific appraisals and emotion-focused coping strategies, as well as educated them about alcohol and alcohol abuse, problem-solving techniques, and self-esteem enhancement. A limitation, however, is that although these interventions might have several beneficial effects on children's adaptation, they have not always prevented depressive symptoms per se. For example, the Family Bereavement Program[188] targeted parental warmth through communication skills, increasing positive events through fun activities, and decreasing negative events through emotion-focused and problem-focused coping. They found increased warmth in the parent–child relationship, fewer negative events, more positive events, and better family coping and cohesion for the intervention group compared to controls, but no significant effect for children's depression. Thus, individuals' responses to stress are a logical target for prevention programs, but more basic, prospective, and experimental research needs to be conducted on the nature of the relation between coping and depression. In addition, possible iatrogenic effects of prevention programs that train children to use emotion-focused coping should be evaluated. If some forms of emotion-focused coping (e.g., avoidance, emotional expression) are linked with more internalizing problems,[200–203] then teaching such coping strategies in depression-prevention programs may be contraindicated.

Interpersonal Relationships

Two important findings emerge regarding the link between interpersonal vulnerability and depression. First, families with a depressed member tend to be characterized by problems with attachment, communication, cohesion, social support, childrearing practices, chronic criticism, harsh discipline, and inappropriately peer-like relationships.[204–206] Moreover, low levels of parental warmth, high levels of maternal hostility, and escalating parent–adolescent conflict significantly predict increases in adolescents' internalizing symptoms.[207,208] In addition, perceived rejection by peers, family, and teachers predicts increases in depressive symptoms in children and adolescents.[209,210] Thus, depression in youth is associated with high levels of interpersonal conflict and rejection from various members in their social domain.

Second, depressed individuals are themselves more interpersonally difficult, which results in greater problems in their social network. For example, depressed children have poorer communication and problem-solving skills, and are less supportive and assertive than nondepressed children.[211,212] Depressed parents have more conflict with their spouses and children; are more hostile, less involved with, and affectionate toward their children; and communicate more poorly than nondepressed parents.[12,82] Moreover, negative reciprocal in-

teraction patterns tend to develop between depressed mothers and their children.[80]

Thus, the relation between interpersonal difficulties and depression is likely to be reciprocal and transactional.[27,213] Longitudinal studies examining the contribution of family dysfunction, parent–child conflict, peer difficulties, and interpersonal rejection to increases in and maintenance of depressive symptoms in children have shown that social problems temporally precede depression, and that depression contributes to interpersonal difficulties. Moreover, interpersonal problems appear to persist after depressive symptoms have remitted.[214]

Programs for preventing depression in children and adolescents have addressed these interpersonal difficulties in several ways. An IPT prevention program that is based on the interpersonal theory and therapy for depression[215] has been tested in college students.[45] This intervention addressed role transitions, role disputes, interpersonal difficulties, and communication skills. At both post-treatment and the 3-month follow-up, students in the intervention group reported lower depression scores than those in the control group. Preliminary results of a study currently being conducted at Columbia University (NIMH grant K23MH071320) testing an interpersonal psychotherapy–adolescent skills training (IPT-AST) group prevention program[216] in a predominantly female sample of middle-school children have been positive.[217]

Several depression-prevention programs have included a social skills training or a social problem-solving component in the overall curriculum, although it was not necessarily the main focus of the intervention. In addition to teaching strategies for erasing negative thoughts and monitoring mood, Cecchini[218] instructed children in social skills such as appropriate interactions, conversation skills, politeness with adults, accepting "no" for an answer, asking appropriate questions, giving compliments, making eye contact, and introductions. This study found increases in social skills for the intervention group, but no significant effect on self-reported depressive symptoms. The Penn State Adolescent Study program[46] similarly focused on both social skills and coping. Although they found improvement in the skills taught in the intervention group, there were no group differences on depressive symptoms.

The study of the Resourceful Adolescent Program (RAP), which combined cognitive–behavioral and interpersonal approaches, found that the intervention group reported lower levels of depressive symptoms than controls.[195] The interpersonal component promoted family harmony and building and accessing psychological support networks. Finally, the Penn Prevention Programs,[43,91] which have been found to reduce levels of depressive symptoms, also included both cognitive–behavioral and interpersonal skills. The social problem-solving component taught perspective taking, information gathering, generating alternatives, decision making, and strategies for coping with family conflict. In a separate study, the Penn Optimism Program (POP) was compared to the Penn Enhancement Program (PEP), which targeted various risk factors for depression, including peer pressure, trust, friendship, and families, as well as study skills, setting goals, self-esteem, and body image.[43] They found that levels of depressive symptoms for children in both conditions were lower compared to controls. Thus, there is some evidence that prevention programs that include an interpersonal component, particularly when combined with strong cognitive–behavioral training may be particularly effective. The independent contribution of each component needs to be examined.

Given the evidence from basic descriptive studies of dysfunctional relationships in families with a depressed parent or child, interventions for preventing depression in youth should attempt to enhance the family environment. Existing prevention programs that have included parents have focused on improving the quality of the parent–child relationship,[188,198] teaching better child-management skills,[42,98] training parents in cognitive and coping skills,[219] providing parents with information about normative development,[195] and giving other family members information about the effects of mood disorders.[71] Many of these programs showed benefits in family relationship variables, although most did not affect children's depressive symptoms. Thus, existing evidence is inconclusive about the benefits of including parents in depression-prevention programs, what particular parenting components have the greatest preventive effect, and at what ages a parent component is likely to have the greatest impact. An important next step in the development of depression-prevention programs would be to explicitly target parenting behaviors that are most likely to contribute to depression in children (e.g., harsh, inconsistent discipline; criticism and rejection; withdrawal, intrusiveness). This then could supplement the child-focused components of the intervention that more directly address children's cognitions and coping strategies.

Summary and Future Directions

In summary, basic epidemiologic and clinical research has helped to identify who is at risk for developing depression, and thus who should be the target of prevention programs. Risk factors particularly associated with an increased likelihood of depression include being female, anxious, offspring of depressed parents, having subclinical levels of depressive symptoms, and being exposed to stress or trauma. The specific number, combination, or weighting of these or other risk factors for predicting depression is not yet known, however. No one risk factor is either necessary or

sufficient; rather the number or interaction among several risk factors may be what increases the likelihood of depression.[29] Some have asserted that the simple accumulation of risk factors leads to depression,[220,221] whereas others have suggested integrated multilevel models, which posit that individuals with particular diatheses are at increased risk for depression when confronted with stressful life events.[21,22,222,223] Moreover, these individual vulnerabilities both increase their chances of encountering stress[172,174] and decrease their ability to deal with the stress once it occurs.[23]

Thus, given that the causes of depression likely involve numerous risk and protective factors across multiple levels of analysis, a multilevel integrated prevention program may be needed that targets these factors either simultaneously or sequentially. Effective prevention requires coordinated action in each domain of functioning and system of influence implicated in the risk model upon which the prevention approach is based. As we learn more about the relations among the various risk factors and processes, particularly regarding the timing of the links between distal and proximal causal variables, then it will become possible to target the more essential elements in the chain. In the absence of this knowledge, however, it makes sense to cast the net broadly and intervene across the multiple levels. Therefore, depression-prevention programs should initially include a variety of intervention components, each of which addresses risk and protective factors in different domains. Once a comprehensive program is shown to successfully prevent depression, then the process of systematically dismantling it to identify the crucial ingredients can begin. The challenge is to strike a balance between creating the most complete and effective prevention program, while at the same time making it feasible and cost effective for widespread dissemination.

Several depression-prevention studies have targeted one or more risk groups, but no one study has included all of them, nor has any study compared the efficacy of depression-prevention programs for persons with different kinds of risk or number of risk variables (e.g., two, three, four, or more). Whereas targeting high-risk individuals ensures that the intervention is provided to those with the greatest need, the recruitment and screening processes often are time consuming and expensive. In light of the transactional and contextual aspects of development, the optimal plan may be to combine universal and targeted approaches. Multitiered prevention programs that provide general skills to universal samples and then increasingly narrow their focus to different selective and indicated high-risk groups may provide the best "bang for your buck." Most children likely would benefit from learning problem solving, coping, conflict resolution, and interpersonal communication. In addition, universal interventions can alter the environment in which high-risk individuals live by improving general social interactions and reducing stigma. Then, using a targeted approach, more specific skills could be provided to certain high-risk groups depending on their particular area of need.

The ultimate targeted prevention program would identify those at greatest risk based on their family history, individual characteristics (e.g., neuroticism, cognitive style), or exposure to particular stressors (e.g., parental divorce, parental psychopathology). The intervention should teach (1) cognitive and behavioral strategies for responding to and managing stress (e.g., cognitive restructuring, behavioral activation, problem solving), (2) engagement rather than disengagement coping, (3) interpersonal communication skills to improve relationships with others and help reduce some of the social stressors that they generate, (4) recognition of the way one's own behaviors may contribute to the generation of stress, and (5) emotion-regulation strategies that reduce labile reactions to stress and distress. Finally, given that several risk factors are common predictors of various disorders,[3] teaching some of these general skills likely will reduce the occurrence of other conditions that share several of these generic risk factors.

Of course, the interventions need to be delivered in a manner consistent with the children's level of cognitive, emotional, and social development. In addition, parents should be taught about age-appropriate parenting skills, as well as the same communication, coping, and cognitive strategies that their children are taught. Field trials of the preventive intervention then should yield further insights about the developmental processes that contribute to the underlying risk for depression.

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Appendix A. Risk factors, basic findings, and related prevention programs

Risk factors	General findings	Prevention strategies	Relevant program study (year)
Gender	Rates of depression are greater in females than males	Use interpersonal approaches with girls? Penn Resiliency Programs Same gender groups?	Penn Prevention Programs[1,2] Forsyth (2001)[3] Gillham (2006)[4] Chaplin (2006)[5]
Genes	Depression is heritable, but not inevitable	Psycho-education about genetic risk	Beardslee (1997)[6]
Parental depression	Offspring of depressed parents are at greater risk for depression compared to offspring of nondepressed parents	Psycho-education Cognitive-behavioral Cope with parent's depression Treat parental depression	Beardslee (1997)[6] Clarke (2001)[7] Compas (2004)[8] Garber (2003)[9], Riley (2003)[10], Weissman (2006)[11]
Subsyndromal depressive symptoms	Subsyndromal depressive symptoms predict later MDD	Reduce current symptoms and prevent them from getting worse	Penn Prevention Programs[1,2] Clarke (1995)[12], Clarke (2001)[7]
Anxiety	Anxiety tends to precede and predict depression	Specifically target reducing anxiety as a means of preventing depression	Lowry-Webster (2001)[13], Hains (1994)[14]
Temperament/personality	Negative emotionality predicts depression, and may mediate the link between genes and depression	Emotion regulation Dialectical behavior therapy[a] Mindfulness[a]	Linehan (2001)[15] Teasdale (2000)[16]
Biology	Depression is characterized by neurobiological dysregulation and left frontal hypoactivation	Mindfulness meditation[a]	Davidson (2003)[17]

Risk factors	General findings	Prevention strategies	Relevant program study (year)
Negative cognitions	Negative cognitions predict depression, particularly under conditions of stress	Cognitive restructuring, reappraisal, problem solving, assertiveness training, cognitive coping strategies	Clarke (1995)[12], Clarke (2001)[7] Penn Prevention Program[1,2] Hains (1994)[14] Petersen (1997)[18] Pössel (2004)[19] Seligman (1999)[20] Shochet (2001)[21] Spence (2003)[22], Spence (2005)[23]
Stress and trauma	Recent stress and early adversity predict depression Depressed persons generate stress	Stress inoculation Reduce stress Reduce their generation of stress	Hains (1994)[14]
Responses to stress	Maladaptive responses (e.g., rumination, avoidance) mediate and moderate the relation between stress and depression	Primary and secondary control coping skills Coping skills Self-management, calming Behavioral activation vs avoidance[a] Coping with specific stressors Parental divorce Parenting skills Death/bereavement Parental alcoholism	Weisz (1997)[24] Petersen (1997)[18], Penn Prevention Programs[1,2] Shochet (2001)[21] Dimidjian (2006)[25] Gwynn (1987)[26] Wolchik (1993)[27] Sandler (1992)[28] Roosa (1989)[29]
Interpersonal relationships	Problems in interpersonal relationships (e.g., dysfunctional families, peer rejection) are associated with depression in children and adolescents		Forsyth (2001)[3], Young (2005)[30]
Parents		Family harmony, access social networks Parental child-management skills Parent-child relationship	Shochet (2001)[21] Lowry-Webster (2001)[13] Wolchik (1993)[27] Sandler (1992)[28]
Peers & Romantic		Social skills Social problem solving	Cecchini (1997)[31] Penn Prevention Programs[1,2]

[a]Study was with adults.
MDD, Major Depressive Disorder.

Appendix B. Description of depression prevention studies in children and adolescents

Study (year)[ref]	Universal samples	Age	*n* (% female)	Intervention type and length
Clarke (1993)[32] Study 1	9th & 10th graders	M = 15.4	662 (42.2%)	Two educational lectures and 1 videotape describing symptoms, causes, and treatments of depression Three 50-minute sessions in consecutive health classes
Clarke (1993)[32] Study 2	9th & 10th graders	M = 15.1	380 (46%)	Depression education and behavioral training: increase pleasant activities; show relation between mood and activities Five 50-minute sessions in consecutive health classes
Kellam (1994)[33]	1st graders	4.7–9.4 M = 6.3	575 (49%)	Mastery learning program to improve reading competence: group-based approach to mastery; more flexible corrective process Continual implementation of curricular alterations over school year
Hains (1994)[14]	9th–12th graders	NR	21 (76%)	Stress inoculation training using cognitive restructuring, problem solving, anxiety management Four group and 9 individual 50-minute sessions
Cecchini (1997)[31] Johnson (2000)[34]	5th graders	NR	110 (NR)	Improve interpersonal relationships, social skills, strategies for reducing negative thoughts, mood monitoring Eight 50-minute group sessions 2 times a week
Petersen (1997)[18]	6th–9th graders	NR	335 (NR)	Teach adaptive emotional, cognitive, and behavioral stress responses Sixteen 40-minute group sessions
Ialongo (1999)[35]	1st graders	5–7 M = 6.2	678 (47%)	Classroom-centered program: curriculum changes; improve behavior management strategies; family-school partnership training for teachers and parents. Continual implementation of curricular alterations over school year
Pattison (2001)[36]	5th & 6th graders	9–12 M = 10.4	66 (52%)	Penn Prevention Program: one group with cognitive component first, one with social component first Ten weekly 2-hour group sessions
Lowry-Webster (2001)[13]	5th–7th grade Australia	10–13	594 (53%)	A family-based group CB program targeting anxiety. Teaches physiological, cognitive, and behavioral coping; teaches parents child management, discipline skills Ten weekly 1-hour group sessions
Shochet (2001)[21]	Year 9 Australia	12–15 M = 13.5	260 (53%)	Resourceful Adolescent Program (RAP): school-based resilience program with cognitive-behavioral and interpersonal approaches. Family program includes parallel parent education RAP-Adolescent: 11 weekly 40–50-minute group sessions RAP-Family: +3 parent sessions
Spence (2003)[22] Spence (2005)[23]	Grade 8 Australia	12–14 M = 12.9	1500 (52%)	Problem Solving for Life Program: school-based program teaching cognitive restructuring and problem-solving skills Eight weekly 45-minute sessions
Merry (2004)[37]	Years 9 and 10 New Zealand	13–14 M = 14.2	364 (52%)	Adaptation of Resourceful Adolescent Program Eleven sessions conducted in school
Pössel (2004)[19]	Grade 8 Germany	13–14 M = 14.0	324 (48%)	Cognitive-behavioral: changing negative to more realistic thoughts, assertiveness training, social competence training Ten weekly 90-minute sessions conducted in schools

Study (year)[ref]	Selective samples	Age	*n* (% female)	Intervention type and length
Gwynn (1987)[26]	Children of divorced parents	9–11	60 (50%)	Educational support group: divorce education, encourage emotional expression, problem-solving skills training Eight weekly group sessions
Roosa (1989)[29]	Children of alcoholic parents	9–13 M = 10.3	81 (50%)	Educate about alcoholism, activities to improve self-esteem, and emotion-focused coping strategies Eight weekly group sessions
Sandler (1992)[28]	Children whose parent died <2 years ago	7–17 M = 12.4	72 (49%)	Family Bereavement Program: grief workshop, family advisement program targeting parental demoralization, parental warmth, stable positive events, stress management 9 family and 6 parent-only sessions
Wolchik (1993)[27]	Children of divorced parents	8–15 M = 10.6	94 (39%)	Parent-only intervention: improve the mother-child relationship, teach discipline skills, schedule positive activities, improve child's contact with father Two individual and 10 weekly group sessions
Beardslee (1997)[6]	Children of parents with a mood disorder	8–15 M = 11.5	52 (40%)	Cognitive-education program: increase understanding within family, educate about mood disorders Six to ten meetings with parents, child, or both Control condition received two 1-hour lectures
Seligman (1999)[20]	College freshmen with low ASQ scores	NR	235 (52%)	Cognitive restructuring, empirical hypothesis testing, behavioral activation, interpersonal skills training Eight weekly 2-hour group sessions and 6 individual sessions over next 2 years
Quayle (2001)[38]	7th & 8th grade Australian	11–12	47 (100%)	Adaptation of Penn Prevention Program for Australian children Eight 80-minute weekly sessions
Cardemil (2002)[39] Study 1	Low-income Latino	M = 11.3	49 (45%)	Modified Penn Resiliency Program: changed ethnicity of children in examples, focused on problems specific to low-income families, single-parent homes, and managing interpersonal conflict Twelve weekly 90-minute group sessions
Cardemil (2002)[39] Study 2	Low-income African American	M = 10.9	106 (55%)	Modified Penn Resiliency Program: changed ethnicity of children in examples, focused on problems specific to low-income families, single-parent homes, and managing interpersonal conflict Twelve weekly 90-minute group sessions

Study (year)[ref]	Indicated samples	Age	*n* (% female)	Intervention type and length
Jaycox (1994)[2] Gillham (1995)[40]	Children with depressive symptoms and/or family conflict	10–13 M = 11.4	143 (46%)	Penn Prevention Program (PPP): cognitive component teaches link between thoughts and feelings; social problem solving component teaches goal setting, perspective taking, decision making, generating action alternatives Twelve weekly 90-minute group sessions
Clarke (1995)[12]	Children with depressive symptoms	M = 15.3	150 (70%)	Cognitive-behavioral: identify and challenge automatic negative thoughts, develop effective coping strategies Fifteen 45-minute group sessions 3 times a week
Reivich (1996)[1] Shatté (1996)[41]	Children with depressive symptoms	12–14 M = 12.7	152 (47%)	Penn Optimism Program: identical to PPP; Penn Enhancement Program: affect focused with an emphasis on emotional expression Twelve weekly 2-hour group sessions
Lamb (1998)[42]	Rural high school students	14–19 M = 15.8	41 (56%)	Cognitive, coping, problem-solving, and communication skills Eight weekly sessions
Forsyth (2001)[3]	Students with depressive symptoms	18–25 M = 19.4	59 (97%)	Interpersonal program: role transitions, role disputes, and emotional expression Four group sessions

Study (year)[ref]	Indicated samples	Age	*n* (% female)	Intervention type and length
Clarke (2001)[7]	Children with depressive symptoms	13–18 M = 14.6	94 (60%)	Cognitive-behavioral program: cognitive restructuring, specifically targeting parent-related beliefs Fifteen 1-hour group sessions
Yu (2002)[43]	Chinese youth with depressive symptoms or family conflict	8–15 M = 11.8	220 (45%)	Modified Penn Optimism Program: adapted for use with Chinese children Ten weekly 2-hour group sessions
Gillham (2006)[4]	Children with depressive symptoms, in primary care	11–12	271 (53%)	Penn Resiliency Program: same as PPP Twelve 2-hour group sessions
Gillham (2006)[44]	6th & 7th graders with depressive symptoms	11–12	74 (36%)	Shortened Penn Resiliency Program with all the same components for children. Parents were taught the core skills that their children were learning but at an adult level Eight 2-hour group sessions for children; six 90-minute sessions for parents

ASQ, Attributional Style Questionnaire; CB, cognitive–behavioral; NR, not reported.

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