Depression in Adolescence

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Adolescence is an important developmental period for understanding the nature, course, and treatment of depression. Recent research concerned with depressive mood, syndromes, and disorders during adolescence is reviewed, including investigations of the prevalence, course, risk factors, and prevention and treatment programs for each of these three levels of depressive phenomena in adolescence. A broad biopsychosocial perspective on adolescent depression is recommended, and possible directions for future integrative research are proposed. Based on current research and knowledge, implications for research, program, and national policy are considered.

The image of adolescence as a time of storm and stress, intense moodiness, and preoccupation with the self has permeated both professional and lay perspectives on this developmental period. The belief that significant difficulties, including depression, during adolescence represent normal development has had two major effects on research and practice: (a) Difficulties during adolescence were not considered as an important developmental variation, and (b) adolescent problems were often not treated because of the belief that the adolescent would grow out of them.

Although this view of adolescence is the one commonly reflected in the media and many professional descriptions of adolescence, it is not supported by research on this period (Petersen, 1988a). In the late 1960s, there were reports showing that many adolescents traverse this period of life without significant psychological difficulties (Douvan & Adelson, 1966; Offer, 1969). It is now known that the majority of adolescents of both genders successfully negotiate this developmental period without any major psychological or emotional disorder, develop a positive sense of personal identity, and manage to forge adaptive peer relationships at the same time they maintain close relationships with their families (Powers, Hauser, & Kilner, 1989). Conversely, research in the 1970s focusing on those youth with problems demonstrated that psychological difficulties in adolescence frequently developed into serious psychiatric disorder in adulthood (Rutter, Graham, Chadwick, & Yule, 1976; Weiner & DelGaudio, 1976). These and other studies demonstrated the inappropriateness of the belief that difficulties such as depression were normal manifestations of adolescence and pointed toward the need for assessment, diagnosis, prevention, and treatment at this age. These studies also highlighted the need for more research on the development of depression in adolescence.

The dramatic increase in knowledge about adolescence has had clear effects on the direction and emphasis of recent research. Now that depression, depressive symptoms, and depressed mood have been recognized as appropriate subjects of investigation in adolescents, the number of studies on these topics has exploded. For example, in our search of PsycLIT using the key words *adolescent* and *depression* for entries published from 1987 through 1991, more than 2,000 entries were found. There now is a critical mass of knowledge that lends itself to a synthesis such as the present one.

The goals of this article are to provide an overview of the primary issues in research, intervention, and policy related to depression during adolescence. We first consider the conceptualization and measurement of depressive phenomena during this developmental period, followed by a discussion of other disorders and conditions that cooccur with depression. Next we consider the important risk and protective factors that have been found to be related to adolescent depression, as well as programs that have been developed to prevent and treat this problem. Finally, we outline the policy implications of the results of research on adolescent depression.

What Is Adolescent Depression?

Three approaches to the assessment and classification of adolescent psychopathology have been reflected in the literature on adolescent depression: (a) depressed mood, (b) depressive syndromes, and (c) clinical depression. Each approach reflects different assumptions about the nature of psychopathology, serves different purposes, and reflects

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a different level of depressive phenomena (e.g., Angold, 1988; Cantwell & Baker, 1991; Compas, Ey, & Grant, 1992; Kazdin, 1988; Kovacs, 1989). For example, the study of depressed mood during adolescence has emerged from developmental research in which depressive emotions are studied along with other features of adolescent development. The depressive syndrome approach assumes that depression and other syndromes reflect the co-occurrence of behaviors and emotions as quantitative deviations from the norm. The clinical approach is based on assumptions of a disease or disorder model of psychopathology.

Depressed Mood

Everyone experiences periods of sadness or unhappy mood at various points in his or her life. These periods of depressed mood may occur in response to many situations, such as the loss of a significant relationship or failure on an important task. They may last for a brief or an extended period of time; they may be associated with no other problems or many problems. Research on depressed mood has been concerned with depression as a symptom and refers to the presence of sadness, unhappiness, or blue feelings for an unspecified period of time. No assumptions are made about the presence or absence of other symptoms. Depressed mood is typically measured through adolescents' self-reports of their emotions, either through measures specifically concerned with mood (e.g., Petersen, Schulenberg, Abramowitz, Offer, & Jarcho, 1984) or through items included in checklists of depressive symptoms (e.g., Kovacs, 1980). Sad or depressed mood is usually experienced with other negative emotions, such as fear, guilt, anger, contempt, or disgust (Watson & Kendall, 1989) and is frequently present during adolescence when any of these other negative emotions are present (Saylor, Finch, Spirito, & Bennett, 1984). Depressed mood is also likely to be linked with other problems, such as anxiety and social withdrawal. Although anxiety and depressed mood frequently occur at the same time, anxiety may or may not be related to positive moods; in contrast, depressed mood is not present when one feels happy, or, conversely, happy mood does not occur at the same time as depressed mood (Watson & Clark, 1984; Watson & Kendall, 1989). Moreover, the presence of depressed mood, based on parents' or adolescents' reports, has been found to be the single most powerful symptom in differentiating clinically referred and nonreferred youth (Achenbach, 1991b, 1991d).

Depressive Syndromes

Multivariate empirical approaches to the assessment of adolescent psychopathology, including depression, have shown that aspects of depression are associated with many other problems (Achenbach, 1991a). Depression is viewed as a constellation of behaviors and emotions that have been found statistically to occur together in an interpretable pattern at a rate that exceeds chance, without implying any particular model for the nature or cause of these associated symptoms. This approach has identified a syndrome of complaints that include both anxiety and depression and is based on symptoms such as feels lonely; cries; fears doing bad things; feels the need to be perfect; feels unloved; believes others are out to get him or her; feels worthless, nervous, fearful, guilty, self-conscious, suspicious, or sad; and worries (Achenbach, 1991a, 1991b, 1991c). This constellation of symptoms has been reliably identified in reports of adolescents, their parents, and their teachers. Scores on this syndrome are strongly related (average $r = .51$) to seven other problem syndromes identified by this approach: withdrawn, somatic complaints, social problems, thought problems, attention problems, delinquent behavior, self-destructive, and aggressive behavior (Achenbach, 1991a).

Clinical Depression

There are two major diagnostic models typically used to diagnose clinical depression: the categorization of mental disorders developed by the American Psychiatric Association (1987) and the method developed by the World Health Organization (1990). The American Psychiatric Association method is the one most widely used in the United States and abroad (Maser, Kaelber, & Weise, 1991). It bases the diagnosis of disorders on a review of the presence, duration, and severity of sets of symptoms. This approach not only assumes that depression includes the presence of an identifiable syndrome of associated symptoms but also assumes that these symptoms are associated with significant levels of current distress or disability and with increased risk for impairment in the individual's current functioning. Under depressive disorders, adolescents may be diagnosed as experiencing major depressive disorder (MDD) or dysthymic disorder, or both. To meet the criteria for MDD, the adolescent must have experienced five or more of the following symptoms for at least a two-week period at a level that differs from previous functioning: (a) depressed mood or irritable mood most of the day, (b) decreased interest in pleasurable activities, (c) changes in weight or perhaps failure to make necessary weight gains in adolescence, (d) sleep problems, (e) psychomotor agitation or retardation, (f) fatigue or loss of energy, (g) feelings of worthlessness or abnormal amounts of guilt, (h) reduced concentration and decision-making ability, and (i) repeated suicidal ideation, attempts, or plans of suicide.

A dysthymic disorder is diagnosed when the adolescent has had a period of at least one year in which he or she has shown depressed or irritable mood every day without more than two symptom-free months. In addition, dysthymic disorder requires the presence of at least two of the following symptoms: (a) eating problems, (b) sleeping problems, (c) lack of energy, (d) low self-esteem, (e) reduced concentration or decision-making ability, and (f) feelings of hopelessness. There cannot be an episode of MDD during the first year of dysthymic disorder. Primary dysthymia (DY) is unrelated to non-mood disorders such as eating disorders, substance abuse disorders, or anxiety disorders; any of these would classify the disorder as a secondary DY. The occurrence of an episode of MDD

following the onset of DY is referred to as *double depression.*

Integration of Approaches

Although considerable overlap exists in the classification of depressed mood and depressive syndrome, only depressed mood and low self-esteem or feelings of worthlessness link the first two approaches with a clinical diagnosis of depression. Four other symptoms are common to two of the approaches: loneliness, guilt, suicidal ideation, and emotional sensitivity. A clinical diagnosis of depression is unique in that it also requires somatic problems such as sleep and appetite problems, psychomotor problems, and fatigue.

Empirical studies of the correspondence among the three approaches have used diagnostic interviews to assign *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed; *DSM-III*) diagnoses and questionnaires or checklists to assess depressive mood or syndromes (Edelbrock & Costello, 1988; Garrison, Addy, Jackson, McKeown, & Waller, 1991; Rey & Morris-Yates, 1991; Roberts, Lewinsohn, & Seeley, 1991; Weinstein, Noam, Grimes, Stone, & Schwab-Stone, 1990). These studies provide evidence for the convergence of *DSM-III* diagnoses with adolescents' and parents' reports of depressed mood and syndromes. However, measures of depressed mood and syndromes identify large numbers of adolescents who do not meet diagnostic criteria for a diagnosis of MDD or DY. Thus, adolescents with clinical depressive disorders may represent a subgroup of a larger population of adolescents who are experiencing a depressive mood or syndrome (Compas, Ey, & Grant, 1992).

Considerable confusion has arisen in the literature from the use of the term *depression* to refer to all three levels of depressive phenomena. Greater clarity can be achieved by specifying whether a depressed mood, syndrome, or disorder has been measured.

Who Becomes Depressed in Adolescence?

There are no nationally representative epidemiological studies of depression in adolescents. As a result, little is known about the incidence of depression in various ethnic groups and social classes. At the same time, there are increasing numbers of studies using the three approaches just described that provide some information on rates of depressive mood, syndromes, and disorders in some samples of adolescents.

Depressed Mood

Most of the studies conducted more recently assess what we have called *depressed mood* (see Petersen, Compas, & Brooks-Gunn, 1991, for a review). The 30 studies we identified are based entirely on nonclinical samples. For example, Achenbach (1991a, 1991b, 1991c) has reported the frequency of sad, unhappy, depressed mood based on a single item. On the basis of parents' reports, 10%–20% of nonreferred boys and 15%–20% of nonreferred girls experienced depressed mood in the previous six months; on the basis of adolescents' self-reports, 20%–35% of boys and 25%–40% of girls experienced depressed mood. Although studies of depressed mood typically examine the phenomenon in relation to other problems or aspects of adolescent development, some investigators of depressed mood have identified a threshold above which a score is thought to be predictive of clinical depression. Using such scores, the median rate of depression in 14 studies was 35%, a much higher rate than that reported in studies examining clinical depression (e.g., Kandel & Davies, 1982; Roberts et al., 1991).

All but 3 of the 16 studies examining gender effects found differences, in all cases with girls reporting more depressed affect than boys. Of 10 studies examining age effects, most found no such effects; however, only 2 of these studies were actually longitudinal, following the same subjects over time. In two longitudinal studies, depressed affect decreased with age for boys but remained level across time for girls (J. Block, 1991; Petersen, White, & Stemmler, 1991). A different pattern of developmental change has also been reported: Radloff (1991) found dramatic increases in depressed moods between the ages of 13 and 15 years, a peak at approximately 17–18 years, and a subsequent decline to adult levels. This pattern has also been found with a measure of depressive episodes in which the midadolescence peak was higher for girls than for boys (Petersen, White, & Stemmler, 1991). Elevated rates of depressed mood in adolescence relative to adulthood have been reported in other studies as well (e.g., Allgood-Merten, Lewinsohn, & Hops, 1990; Larsson & Melin, 1990).

Depressive Syndrome

Research examining depressive syndromes has utilized a cutoff score corresponding to the 95th percentile in a nationally representative sample of adolescents to identify a clinical range (Achenbach, 1991a, 1991b, 1991c). This score was identified as having the optimal power for discriminating between clinically referred and nonreferred samples with the lowest rates of false positives and false negatives. Thus, this approach to measuring depressive syndromes has established an empirically based rate of 5% of the normal population in the clinical range on a depressive syndrome at any given time.

Clinical Depression

Considering all published studies of clinical depression in adolescence (Petersen, Compas, & Brooks-Gunn, 1991), the percentage of adolescents receiving a diagnosis of depression varied from near zero in a large nonclinical sample of children aged from 10 to 11 years (Rutter, Tizard, & Whitmore, 1970) to 57% in a clinical sample of children 8 to 13 years old (Kovacs, Feinberg, Crouse-Novak, Paulauskas, Pollack, & Finkelstein, 1984). Studies based on clinical samples naturally tended to yield higher depression rates (averaging 42%, median 48%, across six studies). Fourteen studies of nonclinical samples reported an average of 7% clinically depressed. These studies generally reported minimal information on the characteristics

of the samples. Two studies of community samples provided examples of base rates of depressive disorders in the population, with Kashani et al. (1987) reporting 8% of the population with MDD and Rohde, Lewinsohn, and Seeley (1991) reporting approximately 3% of adolescents with MDD.

Although there do not appear to be age variations in community samples within the adolescent decade, depressive diagnoses as well as depressed mood appear to increase dramatically in adolescence compared with childhood (Fleming & Offord, 1990; Rutter et al., 1976; Rutter, 1986). Preadolescent or adolescent onset of clinical depression is considered to be a serious risk factor for adult depression and perhaps other major mental disorders as well (Harrington, Fudge, Rutter, & Pickles, 1990; Kovacs, Feinberg, Crouse-Novak, Paulauskas, & Finkelstein, 1984; Kovacs, Feinberg, Crouse-Novak, Paulauskas, Pollack, & Finkelstein, 1984).

Variations by Gender, Ethnic Group, and Cohort

All the evidence suggests that increases in depressive disorders and mood are greater for girls than for boys during adolescence (e.g., Kandel & Davies, 1982; Kashani et al., 1987; Petersen, Kennedy, & Sullivan, 1991). The gender difference that emerges by age 14–15 years appears to persist into adulthood. Many scholars have considered whether the gender difference is a true difference in depression or whether it can be explained by artifacts such as different styles of responding to questions and differences in openness. These examinations have concluded that the gender difference appears to be a true difference in the experience of depression (Gove & Tudor, 1973; Nolen-Hoeksema, 1987; Nolen-Hoeksema, Girgus, & Seligman, 1991; Weissman & Klerman, 1977). Men and women may have different response styles in which men distract themselves, whereas women ruminate on their depressed mood and therefore amplify it (Nolen-Hoeksema, 1987). Sex role socialization in early adolescence, related to the biological changes of puberty that heighten an identity with one's gender, is thought to produce the observed change in these gender differences by midadolescence. Another explanation for increased experience of depression among girls is that girls experience more challenges in early adolescence (Petersen, Sarigiani, & Kennedy, 1991). For example, girls are more likely than boys to go through puberty before or during the transition to secondary school (Petersen, Kennedy, & Sullivan, 1991; Simmons & Blyth, 1987). In addition, several studies have reported that parental divorce is more likely for girls than for boys in early adolescence (e.g., J. H. Block, Block, & Gjerde, 1986; Petersen, Sarigiani, & Kennedy, 1991). Both less effective coping styles and more challenges may increase the likelihood of depression among girls. Studies are needed that simultaneously test these and other hypotheses.

Rates of depression and depressed mood may be higher among adolescents in some ethnic groups or other subgroups. For example, in a review of community studies of adolescent depression, Fleming and Offord (1990) reported that in two of five studies where race was examined, African-American adolescents had higher rates of depression and depressed mood than Whites. On the other hand, Nettles and Pleck (in press) reviewed several studies and concluded that although African-American youth are at greater risk for many negative behavioral and health outcomes, rates of depressive symptoms in African-American samples are typically lower than in Caucasian youth. In a study of one of the largest multiethnic samples of adolescents, Dornbusch, Mont-Reynand, Ritter, Chen, and Steinberg (1991) reported that Caucasian and Asian-American youth reported more depressive symptoms than African-American or Hispanic-American adolescents, even after controlling for levels of stressful life events. Given other findings (e.g., Fitzpatrick et al., 1990), it is probably wise to note Hammen's (1991) conclusion that there is no evidence for Black–White differences in depression among adults. Rates among Native-American adolescents appear to be elevated (Beiser & Attneave, 1982; May, 1983); high rates have been reported especially among Native Americans in boarding schools (Kleinfeld & Bloom, 1977; Kursh, Bjork, Sindell, & Nelle, 1966; Manson, Ackerson, Dick, Baron, & Fleming, 1990). Furthermore, adolescents living in rural areas may be at a greater risk for depression compared with those in urban or suburban areas (Sarigiani, Wilson, Petersen, & Vicary, 1990; Petersen, 1991; Petersen, Bingham, Stemmler, & Crockett, 1991), although Hammen (1991) concluded that there are no urbanicity variations among adults. Gay and lesbian youth have a two- to threefold risk of suicide (Gibson, 1989), and they are probably at greater risk for depression.

The National Institute of Mental Health Epidemiological Catchment Area studies have suggested historical increases in depression (Weissman, Leaf, Holzer, Myers, & Tischler, 1984). Rates of depression have increased significantly since World War II (Klerman, 1988). These historical changes in rates of depression may have had an especially strong impact on the adolescent population. This was supported by a recent study by Ryan et al., (1992), who found similar increases in depressive disorders in more recently born cohorts of prepubertal siblings of depressive probands. Although it has been speculated that these increases were baby boom effects (Klerman, 1988), recent cohorts continue to show higher rates of most of these problems, suggesting that it is not simply due to a larger cohort of youth (Gans, Blyth, Elsby, & Gaveras, 1990). In summary, although much more work is needed on the epidemiology of depression in adolescence, existing evidence suggests increased risk of depression in recent decades. Girls and other groups—such as Native Americans and homosexual youth—may have increased risk of depression, but too few studies have considered subgroup variations, with the exception of gender, to permit inferences about depression in subgroups of adolescents.

Comorbidity and Co-Occurrence of Other Disorders

One of the most well-documented facts about depressive mood, syndromes, and disorders is that they often co-occur with other symptoms and disorders (Compas & Hammen, in press). In examining the comorbidity of disorders and the co-occurrence of symptoms and syndromes, one must distinguish between studies with community as opposed to clinical samples. Estimates of true comorbidity and co-occurrence can be obtained only from community samples, as clinical groups will be affected by a number of sources of bias (Caron & Rutter, 1991).

In a large community-based sample, 42% of the adolescents who had experienced a depressive disorder had a comorbid disorder, a rate that was significantly higher than expected from the base rates of the disorders and higher than the rate for adults with depression (Rohde et al., 1991). The co-occurrence of disorders was similar for boys and girls, except that boys were more likely to have both disruptive disorders and depression, whereas girls were likely to have eating disorders and depression. Comorbidity with depression in adults was found primarily for substance abuse, whereas depression was associated with more disorders in adolescence.

A wide range of disorders show high rates of comorbidity with depressive disorders. For example, the comorbidity of depression and anxiety disorders is estimated at 30%–70% (Kovacs, 1990). Twenty-one percent of the Rohde et al. (1991) sample were diagnosed as having an anxiety disorder. The overlap between depression and conduct disorders is also high, estimated at 10%–35% in adolescents (Kashani, Reid, & Rosenberg, 1989; Kovacs, Paulaskas, Gatsonis, & Richards, 1988; Rohde et al., 1991). Furthermore, a high incidence of personality disorders has been reported among depressed adolescent patients (Clarkin, Friedman, Hurt, Corn, & Aronoff, 1984; Yanchyshyn, Kutcher, & Cohen, 1986).

A high proportion of suicide attempters are depressed, at least after the attempt (Rotheram-Borus & Troutman, 1988; Spirito, Overholser, Ashworth, Morgan, & Benedict-Drew, 1989). Depressed mood appears to be a strong predictor of suicidal ideation (Kandel, Raveis, & Davies, 1991; Lester & Miller, 1990). Depressed mood, drug use, and suicidal ideation are strongly related (J. Block & Gjerde, 1990; Kandel et al., 1991; Levy & Deykin, 1989). Further examination of the process linking substance use, depression, and suicide is needed to test hypotheses differentiating intent to self-medicate from suicidal intent.

Eating disorders and substance abuse also frequently co-occur with depression (Attie, Brooks-Gunn, & Petersen, 1990; Katon, Kleinman, & Rosen, 1982; Rivinus et al., 1984). Similarly, extreme weight and eating concerns covary with depressed mood (Post & Crowther, 1985; Richards, Boxer, Petersen, & Albrecht, 1990; Rosen, Gross, & Vara, 1987). At least in girls, poor body image may lead to eating disorders and then to depression. Several studies have shown elevated depression with medical illness (Cavanaugh, 1986; Fitzpatrick et al., 1990). This relationship is usually interpreted as stress and anxiety of the medical illness causing the depression. It also seems possible that depression may make one vulnerable to medical illness. Depression may cause other problems through its effects on interpersonal functioning (Hammen, 1991). Deficits in interpersonal functioning are thought to produce poor relationships between parent and child as well as between romantic partners. This explanation is hypothesized to explain the threefold elevation of pregnancy among school-aged girls with depressive symptoms (Horwitz, Klerman, Sungkuo, & Jekel, 1991).

In summary, depressive mood, syndromes, and disorders are related to a broad spectrum of other disorders and problems in adolescence. Whether depression causes these problems or the other disorders cause depression remains to be clarified. It appears that there may be some risk factors that contribute to a wide range of negative developmental outcomes during adolescence, including depression, whereas other risk factors may be specific markers of increased vulnerability to depression (e.g., Downey & Coyne, 1990).

Developmental Processes: Risk and Protective Factors

Reviews of research document the challenges of adolescent development, (e.g., Feldman & Elliott, 1990; Petersen, 1988a). Adolescence is a phase of life characterized by change in every aspect of individual development as well as in every major social context (e.g., Petersen, Kennedy, & Sullivan, 1991). The biological changes of puberty as well as the social changes related to the move from elementary to secondary school may be considered primary, with other changes derived from one or both of these. For example, puberty affects body and self-image as well as how the adolescent is seen by others. Similarly, the move to a larger secondary school affects the peer group and friendships. Like other phases of the life course, adolescence also includes experience of stressful life events, with some likely to be more frequent or stressful at this age (Camarena, Sarigiani, & Petersen, 1990). For example, school changes are more frequent in adolescence, and parental divorce may have a stronger impact on some aspects of adolescent development (e.g., romantic relationships). Thus, the extent of potentially difficult changes in adolescence predicts increased psychological difficulty. Depression stands out among the psychological problems of adolescence, both for its impact on adjustment during the adolescent years and its long-term effects on adult psychological functioning.

Biological Processes

Among adults, there is evidence that biological dysregulation occurs with depressive episodes (Akiskal & McKinney, 1973; Shelton, Hollon, Purdon, & Loosen, 1991). The search for clear biological markers of depression in adolescence is in its early stages, and the findings

to date have been inconclusive. For example, although adults tend to secrete more cortisol when depressed (Asnis et al., 1985; Sachar, Puig-Antich, & Ryan, 1985), some studies have found that depressed children and adolescents tend to secrete less cortisol (Dahl et al., 1991; Kutcher & Marton, 1989). On the other hand, it appears that the sleep onset mechanism is impaired in some depressed adolescents, as evidenced by decreasing growth hormone secretion, increasing cortisol, and increasing the time until sleep in suicidal depressed adolescents (e.g., Dahl et al., 1992). Further attention needs to be given to differences among subgroups of depressed adolescents and to the timing of biological measures (e.g., sleep onset vs. 24-hour averages).

Genetic factors are also implicated with depression. Affective disorders tend to run in families (Andreasen, Endicott, Spitzer, & Winokur, 1977; Gershon et al., 1982; Weissman et al., 1984). Identical twins are four to five times more likely than fraternal twins to show concordance for MDD (Kendler, Heath, Martin, & Eaves, 1986; Wender et al., 1986). The genetic loading for childhood and adolescent depression may be higher than that for depression when onset occurs in adulthood, although additional research is needed to examine genetic and environmental contributions (Puig-Antich, 1987; Strober et al., 1988). It also appears that earlier onset of depression is predictive of more frequent and severe depressive episodes (Kovacs, Feinberg, Crouse-Novak, Paulauskas, & Finkelstein, 1984; Strober, 1983). Most family studies have failed, however, to disentangle genetic and environmental processes that may predispose offspring of depressed parents to depressive outcomes.

Psychological Factors

As mentioned earlier, negative body image is thought to lead to depression and eating disorders (Attie & Brooks-Gunn, 1992; Post & Crowther, 1985). Low self-esteem may also lead to depression (Harter, 1990; Renouf & Harter, 1990). Anxiety typically precedes depression, suggesting a causal role (Finch, Lipovsky, & Casat, 1989; Kovacs, 1990; Suomi, 1991). The psychological processes related to depression may be different for boys and girls; in one study, boys who were depressed at 18 years of age were aggressive, self-aggrandizing, and undercontrolled in preschool, whereas depressed 18-year-old girls were overcontrolled in preschool (J. Block, 1991).

Cognitive Factors

Adolescents show dramatic increases in cognitive ability and reasoning capacity (Graber & Petersen, 1991; Keating, 1990). Their increased capacity to reflect on the developing self and the future is thought to play a role in the possibility of experiencing depressed mood. Hammen (1990) outlined three general approaches to research on cognitive vulnerability to child–adolescent depression: information-processing models, depressive attributional style, and self-control cognitions. There is evidence of differences between depressed and nondepressed adolescents in each of these aspects of cognition; however, the role of cognitive processes as causal factors in depression is far from clear. For example, children and adolescents who attribute negative events to internal, stable, and global causes are more likely to be depressed (Kaslow, Rehm, & Siegel, 1984). A recent longitudinal study indicated that attributions may emerge as an important correlate or predictor of depressive symptoms in later childhood and early adolescence but are unrelated to depressive symptoms in childhood (Nolen-Hoeksema, Girgus, & Seligman, 1992). Further longitudinal research is needed to clarify the temporal relations between cognitive processes and depression.

Family Factors

Having a depressed parent is a major risk factor for depression in childhood (see reviews by Downey & Coyne, 1990; Hammen, 1991). Offspring of depressed parents are more likely than children of healthy parents to experience perinatal complications, cognitive impairments in infancy, school problems, peer problems, and high rates of depressive disorders as well as other psychiatric disorders and problem behaviors. Both genetic and psychosocial processes are likely to be involved with these outcomes.

Multiple mechanisms appear to be involved in the transmission of depressive disorders from parents to their children. These include genetic predisposition (Weissman, 1990), emotional unavailability of parents (Lee & Gotlib, 1991), dysfunctional parent–child interactions (Burge & Hammen, 1991), and marital conflict (Downey & Coyne, 1990). Low family cohesion and expression (e.g., Friedrich, Reams, & Jacobs, 1982; Reinherz, Stewart-Berghauer, Pakiz, Frost, & Moeykens, 1989) as well as family conflict (e.g., Carlton-Ford, Paikoff, & Brooks-Gunn, 1991) are associated with depressive symptoms in children. Parental divorce also appears to amplify behavioral disturbances and depression in adolescents (J. H. Block et al., 1986; Cherlin et al., 1991; Sarigiani, 1990). Marital discord and economic hardship lead to higher incidence of depression in adolescents (Asarmov & Horton, 1990; Lempers & Clark-Lempers, 1990).

Peers

Low peer popularity is related to depression and depressive symptoms (Jacobsen, Lahey, & Strauss, 1983). Among young adolescents, less closeness with a best friend, less contact with friends, and more experiences of rejection contributed to increases over time in depressive affect (Vernberg, 1990). Conversely, being depressed appears to contribute to poor relationships. Whereas poor peer relationships constitute a risk factor for depression in early adolescence, good peer relationships at this age do not appear to provide a protective influence; later in adolescence, close peer relationships do appear to be protective, particularly when parent relationships are impaired in some way (Petersen, Sarigiani, & Kennedy, 1991; Sarigiani, 1990). Poor peer relationships in adolescence are among the strongest predictors of adult disorder (Sroufe & Rutter, 1984).

School Factors

The academic grades of both boys and girls appear to decrease over adolescence (Schulenberg, Asp, & Petersen, 1984; Simmons & Blyth, 1987); although part of this decline is due to increasingly difficult grading practices as students move from elementary to secondary school, depression also may play a role. In one study (Ebata & Petersen, 1992), boys who were depressed and who engaged in minor delinquent activities had sharp grade declines relative to those who were only depressed or only delinquent. Boys with no depressive episodes and no delinquent activities showed stable achievement over the course of adolescence. Interestingly, there was no relationship between depression and achievement for girls.

Daily Stress and Stressful Life Events

The experience of difficult changes or challenges in adolescence appears to predict increased depressed affect (Compas, Grant, & Ey, in press). The effects of parental divorce were mentioned earlier. To give another example, both boys and girls who went through puberty before or at the same time they moved from elementary to secondary school reported more depressed affect than adolescents who went through puberty after their school transition. Interestingly, the effects of becoming pubertal before changing schools increased over the course of adolescence so that the differences in depressed affect were more pronounced by the end of high school (Petersen, Sarigiani, & Kennedy, 1991).

Studies show that daily stressors (e.g., responsibilities at home, arguments with peers, parental restrictions) mediate the association between major stressful events and psychological symptoms (Wagner, Compas, & Howell, 1988). Major life events appear to exert their effects on psychological well-being at least in part by creating daily stress and possibly by weakening personal and social resources for coping with stress (Compas, Howell, Phares, Williams, & Ledoux, 1989). Depressed adolescents report more acute and chronic stressors than youth with conduct disorder or with rheumatic disease or than healthy youth with a number of life stressors. In addition, depressed adolescents report fewer social resources, including fewer supportive social relationships (Daniels & Moos, 1990).

Summary: Risk Factors and Developmental Trajectories

At present, several developmental pathways appear plausible for depression during adolescence. For example, environmental events may trigger biological dysregulation through disruption in the social fabric of an individual's life (Ehlers, Frank, & Kupfer, 1988). Alternatively, depression might result from a series of events and processes, including genetic susceptibility, biological insults, temperament and other individual characteristics, environmental events, developmental changes, and coping responses available to the individual. Biological changes might serve to heighten and maintain the psychological distress experienced by a depressed individual (Whybrow, Akiskal, & McKinney, 1984). A third causal pathway for depression might be chronic or extreme stresses that become difficult and overwhelming for the individual. The death of a loved one or growing up with a depressed parent both constitute strong risk factors for depression. Some dysfunctional processes involve the perpetuation of interpersonal deficits and depression across generations (Hammen, 1991).

Once on a depressed trajectory in development, an individual becomes more likely to stay on this course because of the tendency to both alienate and withdraw from the very social supports that can minimize negative effects. These effects are likely to be especially devastating to a developing adolescent. Imagine the 13-year-old, hospitalized for depression following the death of a parent. The hospitalization removes the adolescent from the peer group and school; family members are likely to visit, but the context is certainly not the same as home. This adolescent not only experiences unusual and perhaps stigmatizing treatment but is likely to miss important developmental experiences, especially at school and with peers. The divergence from the normal developmental pathway in adolescence during treatment for depression may cause, or perhaps reinforce, a more isolated, less socially competent, depressed approach to life. Research is needed that examines the effects of treatment, explores whether there are negative effects, and devises ways of minimizing them if any are found. Developmentally supportive treatments are needed and should be one aim of future research.

Good relationships with parents, and by mid-adolescence with peers, appear to buffer negative effects of stressful life events. Children of depressed parents may not experience these protective relationships, thus exacerbating any genetic vulnerability that may exist. Again, intervention with these children appears to be important. Risk factors for depression and other disorders may be intercorrelated, contributing to the co-occurrence of other problems along with depression (Downey & Coyne, 1990). Research that identifies factors that predispose adolescents to depression is very much needed, as opposed to the wider range of negative developmental outcomes.

Approaches to Intervention: Treatment and Prevention of Adolescent Depression

Treatment

Pharmacotherapy. Building on research with adults, several varieties of psychoactive medications have been evaluated as treatments for adolescent depression. The results of the initial controlled studies of the efficacy of pharmacotherapy for adolescent depression have failed to support the efficacy of pharmacotherapy for adolescent depression (Petersen, Compas, & Brooks-Gunn, 1991; Strober et al., 1988). For example, two double-blind placebo studies of tricyclic antidepressants with adolescents found no differences between treated and control subjects

(Boulos et al., 1991; Geller, Cooper, Graham, Marsteller, & Bryant, 1990), and open trial studies report improvement rates of much less than 50% (e.g., Strober, Freeman, & Rigali, 1990). Given the efficacy of these treatments with adults, researchers have speculated on the reasons for the lack of results and perhaps negative effects. The possibilities range from the different biological substrate of adolescents to the possibility that adolescent depressives have more serious forms of depressive disorders because of earlier onset and higher rates of comorbid disorders such as anxiety.

Psychosocial and psychotherapeutic treatments. Psychosocial and psychotherapeutic interventions in the treatment of adolescent depression include cognitive–behavioral therapy (Lewinsohn, Clarke, Hops, & Andrews, 1990), psychodynamically oriented therapy (Bemporad, 1988), family therapy (Lantz, 1986), social skills training (Fine, Forth, Gilbert, & Haley, 1991), and supportive group therapy (Fine et al., 1991). The three studies that included random assignment to treatment and no-treatment control groups all provide some confirmation that depressed mood, or, in one case, depressive disorders, can be significantly reduced through treatment (Kahn, Kehle, Jenson, & Clark, 1990; Lewinsohn et al., 1990; Reynolds & Coats, 1986). Improvement was found on self-report measures of depressive symptoms, with effect sizes of one standard deviation on the Beck Depression Inventory (BDI) and Center for Epidemiologic Studies of Depression scale (Lewinsohn et al., 1990), two standard deviations on the BDI (Reynolds & Coats, 1986), and three standard deviations on the Children's Depression Inventory (Kahn et al., 1990). Furthermore, Lewinsohn et al. found a 50% reduction in the rate of MDD in the treated versus untreated groups in their study. There was also some evidence that reductions in depressive symptoms and disorders were maintained over longer periods of time ranging from five weeks to six months posttreatment. These studies also suggest that treatment effects are not limited to depressive symptoms; significant reductions have also been found in anxiety symptoms (Lewinsohn et al.; Reynolds & Coats). Studies comparing the efficacy of different types of psychosocial interventions have been rare and the findings inconclusive.

No research has yet compared or integrated the effects of pharmacotherapy and psychotherapy for depressed adolescents. Attention to individual differences in response to treatments as well as to the overall effectiveness rates of these interventions appears to be a more productive avenue for research. It is important to discover whether there are subgroups who show different responsiveness to pharmacologic versus psychosocial treatment.

Prevention of Adolescent Depression

On the basis of the high rates of depressed mood, depressive syndromes, and depressive disorders that occur during adolescence, it is clear that treatment efforts will never be sufficient to meet the full needs of the population. Professional resources are inadequate to meet these needs, both in terms of the limited number of trained professionals who are available and in terms of barriers to access to treatment that are confronted by adolescents, especially adolescents living in poverty. Therefore, prevention of the entire spectrum of depressive problems experienced by adolescents is of paramount importance if the needs of the largest number of adolescents are to be met.

Prevention of adolescent depression can take two forms. First, preventive services can be delivered to entire populations of adolescents on the basis of the assumption that all adolescents are at some risk of experiencing at least depressed mood, if not for the development of depressive disorders. The assumption behind such programs is that all adolescents are exposed to factors that place them at risk for depression and that enhancing their ability to respond adaptively to these risk factors will reduce the incidence of depression in the population. This approach further assumes that depressive symptoms exist on a continuum and that subclinical levels of depressed mood or negative affect may serve as markers of increased risk for the development of depressive disorders. By reducing depressed mood in the population, the overall risk for depressive disorders may also be reduced. Second, adolescents who have been exposed to an identifiable risk factor can be selectively targeted for preventive efforts. For example, adolescents whose parents are clinically depressed are known to be at increased risk of suffering from depressive disorders, as well as other problems, compared with the population at large. Services can be delivered selectively to those at greatest risk and in greatest need.

Populationwide prevention programs. In spite of the promise that broad-based preventive intervention could contribute to reduction in the incidence of depression, no controlled evaluations of such programs have been published. The ongoing research of Petersen and colleagues (cf. Peterson, 1988b, 1991) promises to provide the first such data concerning the effects of a preventive intervention targeting depression for early adolescents.

Broadly focused programs designed to promote social competence and teach problem-solving skills also have relevance for the prevention of depressive mood and symptoms at the population level (e.g., Weissberg, Caplan, & Harwood, 1991). This general family of interventions has been designed to teach the social competencies and life skills needed by adolescents to foster positive social, emotional, and academic development. Although problem-solving interventions have been found to influence a wide variety of outcomes, measures of depressed mood, syndromes, or disorders have not been included in evaluations of these programs. Assessment of depression will be an important criterion to include in future evaluations of social competence promotion programs.

Prevention programs for adolescents at risk for depression. Although populationwide interventions may provide some level of protection against sources of risk for depression for many adolescents, others may be exposed to conditions and circumstances that present a more profound risk for the development of depressive syndromes and disorders. The basic skills that can be

taught in broadly focused programs such as those described above may not be sufficient for individuals faced with heightened risk for depression. Secondary preventive services can be directed to those groups who are identified on the basis of a marker of their increased risk.

Compelling evidence has been summarized above indicating that the single most powerful source of risk for adolescent depression is the presence of a depressive disorder in a parent (Downey & Coyne, 1990; Fendrich, Warner, & Weissman, 1990; Phares & Compas, 1992). The diagnosis of depression in parents serves as a distinct marker of the need for either a prevention- or treatment-oriented intervention for the offspring of these individuals. It would not be an overstatement to say that the need for services for children of depressed parents as closely approximates a *prescriptive* recommendation as can be found in the mental health professions. Furthermore, the incidence of MDD in offspring of depressed parents increases substantially during adolescence, indicating that young adolescents whose parents suffer from depression are a group in particularly high need of services (Beardslee, 1990).

Initial steps have been taken in the development of preventive interventions for depressed parents and their families by Beardslee and colleagues (Beardslee, 1990; Beardslee et al., 1992; Beardslee & Podorefsky, 1988). The intervention is guided by research on the characteristics of adolescents who have displayed resilience in the face of depressive disorders in their parents. Parents and children participate in a psychoeducational intervention that focuses on providing family members the information needed to enhance their understanding of and ways of coping with the parent's depression.

Although there are few prevention programs targeting depression specifically, a number of preventive interventions have been developed to address stressors and problems that may be linked to depression. For example, several programs have been developed to assist adolescents in coping with the stress of divorce (see Grych & Fincham, 1992, for a review). Although the effects of these programs have been reported with regard to anxiety symptoms and other internalizing problems, measures of depressed affect or symptoms have not been, but should be, included in reports of their effectiveness.

Integrating Treatment and Prevention Efforts

It is an unfortunate reality that the resources to support mental health services in the United States fall far short of the economic and human resources needed to address mental health problems. It is even more unfortunate that limits on these resources have contributed to conflict among mental health professionals about the best way to expend these limited resources. In particular, battle lines have been drawn between proponents of prevention and those who favor treatment (e.g., Albee, 1982, 1990).

With regard to adolescent depression, the battle between proponents of prevention and treatment is one that we cannot afford to fight. Services aimed at alleviating the pain and misfortune associated with depression during adolescence must reflect a broad effort to provide coordinated prevention and treatment programs. This position is based on the assumption that no one approach to providing services for adolescent depression will be sufficient to address the problem. Although we fully expect that primary prevention programs will be able to reduce levels of depressed mood and perhaps deter the development of some forms of depressive syndromes and disorders, they will not provide a sufficient dose to interrupt the development of depression in a substantial portion of the population.

The addition of secondary prevention programs aimed at high-risk groups should provide a second net to catch and help many adolescents who, because of exposure to high-risk circumstances such as parental depression, conflict, or divorce, were not helped by the resources they gained through primary preventive efforts. Finally, treatment programs will remain a necessity because many adolescents who are exposed to conditions of profound risk will not be able to gain access to secondary prevention services in order to prevent the onset of more serious depressive disorders. Moreover, the majority of adolescents may have insufficient access to treatment services (Keller, Lavori, Beardslee, Wunder, & Ryan, 1991).

Primary prevention programs at the population level, secondary prevention services for high-risk groups, and treatment for adolescents who manifest severe forms of depressive disorders must be delivered in a coordinated, sequential fashion (Compas, in press). Evidence is accumulating to indicate that interventions at all three of these levels need to address dysfunctional cognitive processes, skills for coping with acute and chronic stress, and strategies to deal with interpersonal relationships and problems. If these themes were addressed in primary and secondary prevention programs as well as in treatment, they would contribute to the development of a core set of competencies for all adolescents. The groundwork for these skills would be laid in a preventive intervention delivered at the population level (Petersen, 1988b). Further development of these competencies could then be pursued in secondary prevention programs for adolescents whose parents are suffering from clinical depression or other sources of risk (Beardslee, 1990). Finally, for those adolescents who warrant further intervention, these same skills would continue to be the target in psychotherapeutic interventions (Lewinsohn et al., 1990). Continuity of this type across interventions could enhance the effects of our efforts to alleviate adolescent depression at the levels of both prevention and treatment.

Implications for Research, Program, and National Policy

Research Policy Implications

Several inferences follow from the nature of the research that we discovered in our review. First, most of the studies classified under *adolescent depression* were based on clinical populations and used diagnostic measures. Second,

a high percentage of studies published since 1987 have focused on children of depressed parents. Third, most of these studies considered depression as an outcome of other conditions or influences, whereas a few examined depression as a cause, for example, of suicide or poor school achievement. Fourth, we could find few studies that examined protective factors; interestingly, individual factors were more likely to be considered only as risk factors, whereas social influences provided most of the instances of protective factors (but were still more likely to be construed as risk factors). Finally, the current literature provides little basis for differentiating processes leading to depressive disorder compared with depressed mood, self-esteem, or other problems such as drug abuse or conduct disorders.

Although the large quantity of research that now exists on adolescent depression may suggest to some that no further efforts are needed to stimulate work in this area, the nature of research that has been conducted is quite lopsided. Most notably lacking are studies that integrate biological and social processes in the etiology of depression at adolescence. (And, as many have argued, understanding the etiology of depression at adolescence would be tantamount to understanding the etiology of depression.) All of the biological studies use clinical samples; studies of social processes tend to use nonclinical samples. As this review demonstrates, enough research exists now to formulate clear hypotheses. The nature of the hypothesis should determine which samples are needed to test it.

Similar observations result from current intervention research. Although the studies of biological treatments rigorously pursue the specific neurobiological processes involved, other treatment studies are less clearly based in theories about process. Intervention studies could illuminate understanding of etiology if well conceptualized and designed.

Having depressed parents is the most consistent risk factor for adolescent depression. Although growing up with a depressed parent presents an enormous risk for the development of depression and other disorders in offspring, not all children of depressed parents display dysfunctional patterns of development. Therefore, comparison between siblings within families of depressed parents is an important avenue for future research. Comparisons among siblings whose parents are depressed may offer clues to the nonshared characteristics of family environments and genetic factors that either increase the risk for disorder or protect children from disorder in these families. Furthermore, data of this type should be useful in constructing preventive interventions for families of depressed parents, as they could provide information about both risk and protective factors for adolescent depression.

Program Policy Implications

A major implication emerging clearly from the existing research on adolescent depression is that children in families of depressed parents are at significant risk for developing depression and other psychopathological problems. Treatment programs for depressed parents must consider treatment for their families. Without such an approach, programs treating adult depression are treating only the parent component of a problematic family dynamic and ignoring the significant effects on offspring.

The possibility that pharmacological treatments may work differently with adolescents than adults suggests that the nature of the processes involved in depression change across the life course in depression. It is important that information about the initial findings of lower rates of effectiveness in pharmacological treatment of adolescent versus adult depression be communicated to clinicians.

If, like diabetes, depression is triggered by situational factors and exacerbated by biological vulnerability, significant opportunity exists for multiple levels of preventive intervention. For example, life skills training programs show promise of providing coping skills to permit young people to deal with situations that might precipitate depression. Such programs would be especially important if the challenges of adolescence have increased relative to earlier historical times. Any effects that reduce morbidity among youth would surely be important to pursue as a policy matter.

Another major implication for programs is the comorbidity of disorders and covariation among adolescent problems. These results suggest that categorical and single problem-focused interventions may be limited in their potential effects. Instead, multivariate, person-focused, comprehensive approaches are likely to be more effective with adolescent depression. Furthermore, interventions targeting single problems have typically not measured program effects' multiple outcomes. It is possible that interventions targeting problems that co-occur with depression (e.g., substance abuse) may affect depression as well.

Implications for National Policy

At the national level, consideration should be given to the reorganization of research funding. The Office of Technology Assessment report (U.S. Congress, 1991) on adolescent health highlighted the low national priority accorded to this area at the same time that it documented the significant challenges confronting adolescents. The three major options recommended in that report would also benefit issues related to adolescent depression: (a) Improve U.S. adolescents' access to health services, (b) restructure and invigorate federal efforts to improve adolescent health, and (c) improve adolescents' environments. Specific strategies are identified in each of these areas. Furthermore, the report provides specific options related to mental health problems.

It is clear that depression is a major, pervasive, and perhaps increasing problem for youth. There now exists a sufficient knowledge base to guide action. Significant morbidity of adolescence and adulthood could be ameliorated by attention to adolescent depression.

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