The Development of Depression in Children and Adolescents

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In recent decades, research on child and adolescent depression has proliferated. Currently, attention in the field is directed toward examining the epidemiology, causes, course, sequelae, and treatment response of children at risk for developing or presently experiencing depressive disorders. In this article, a developmental psychopathology approach is used to elucidate the development of depressive disorders, the diverse pathways that evolve, and the processes that contribute to varied outcomes. The developmental psychopathology perspective underscores the importance of moving beyond the identification of isolated aberrations in psychological and biological components of depressive presentations to the understanding of how those components have evolved and how they are integrated within and across biological, psychological, and social systems. Implications for prevention and intervention are addressed as is the importance of increasing the public awareness of depressive disorders and reducing the social stigma that interfere with the attainment of treatment for depressed persons.

In this article, we present a developmental psychopathology conceptualization of the depressive disorders of childhood and adolescence. Such an approach espouses the viewpoint that to comprehend human development, it is essential to understand the integration of developmental processes at multiple levels of biological, psychological, and social complexity within individuals over the life course. Thus, multidisciplinary efforts to unify and integrate the advances that have taken place in the fields of developmental psychology, clinical psychology, psychiatry, epidemiology, sociology, neurobiology, genetics, and the neurosciences within a developmental psychopathology perspective are essential to address the critical issues involved in the development of depressive disorders.

Depressive disorders are conceived as heterogeneous conditions that are likely to eventuate through a variety of developmental pathways. Single risk factors can rarely be conceived as resulting in depressive outcomes. Rather, the organization of biological, psychological, and social systems as they have been structured over development must be fully examined. We consider a depressotypic developmental organization to be a potential precursor to depressive illness, and we argue that the concept of this organization has much heuristic value in guiding thinking about the diverse processes that underlie symptom expression and depressive outcomes. The developmental position challenges us to move beyond identifying isolated aberrations in cognitive, affective, interpersonal, and biological components of depressive presentations, to understand how these components have evolved developmentally, and to understand how they are integrated within and across biological and psychological systems of the individual embedded within a multilevel social ecology.

We begin by discussing the nature of depressive illness and then examine epidemiological data on and clinical characteristics of depression in children and adolescents. We then articulate concepts from the field of developmental psychopathology and present a model that addresses the development of depression and its manifestation in children and adolescents. As there is a paucity of longitudinal investigations on the development of depression, we draw on research that includes studies that used epidemiological (e.g., representative of the community under investigation), high-risk (e.g., children with depressed parents), and clinical (e.g., hospitalized clinically depressed youngsters or clinically referred children) samples. Of course, our model is necessarily speculative because studies have not examined the emergence and evolution of a depressotypic organization over time. We focus on unipolar depression because the majority of research that has occurred on mood disorders and their development has been conducted with either the offspring of mothers with unipolar depression or on depressed children and adolescents. Accordingly, we examine the emerging insights that a developmental psychopathology perspective provides for comprehending the etiology, course, and sequelae of depressive disorders in childhood and adolescence. We also discuss the treatment and social policy implications derived from this perspective.
Definitional Parameters and Nature of Impairment

Over the past several decades, remarkable scientific progress has occurred in our understanding of the mental disorders of childhood, adolescence, and adulthood (Institute of Medicine [IOM], 1985, 1989). Despite these advances, mental illness continues to challenge millions of individuals and families as well as to place major stress on the service delivery system and on the research community that strive to better comprehend psychopathology and thereby contribute to improved treatment and prevention efforts (IOM, 1985, 1989, 1994).

Among adults, depressive disorders are quite common and tend to co-occur with other serious mental disorders, including substance abuse, anxiety disorders, and schizophrenia. Perhaps most significantly, they are strongly associated with suicide, one of the leading causes of death in adolescents and adults. Depressive disorders also are associated with far-reaching impairments in functioning not only for the depressed individual but also for family members.

With respect to the financial burden to society accompanying the treatment of depression as well as the costs associated with the suffering experienced by those who have or are confronted with the illness, depressive disorders are among the most serious psychopathological disorders. The annual cost of depressive disorders in the United States has been estimated at $43 billion, 85% of which is attributed to expenditures for major depressive disorder (MDD), including costs of treatment as well as absenteeism from work, losses of productivity, and premature death (Hirschfeld et al., 1997). Additionally, because of its chronicity, depression can remain burden-some for a significant period of time. Given the substantial economic drain on individuals, families, and society, and the long-term suffering, the risk for suicide, and the impairment in occupational, interpersonal, and family relationships, it is not at all surprising that, since antiquity, depression and its impact on the human condition have constituted a prominent area of inquiry for philosophers, physicians, psychiatrists, and psychologists alike (Jackson, 1986). Children with clinical depression, similar to adult depressives (Hirschfeld et al., 1997; Robins & Regier, 1991), are undertreated (Beardslee, Keller, Lavori, Staley, & Sacks, 1993; IOM, 1989). In fact, 70% to 80% of depressed teenagers do not receive treatment (Keller, Lavori, Beardslee, Wunder, & Ryan, 1991; Rohde, Lewinsohn, & Seeley, 1991).

Depression has typically been operationalized in three ways, including depressed mood, depressive syndromes, and depressive disorders (Angold, 1988). Depressed mood is delimited by a single symptom or group of symptoms that involve dysphoric affect. Most commonly, self-report measures have been used to identify depressed mood. Depressive syndromes involve sets of symptoms that have been shown to co-occur empirically. Depressive disorders are reflected by categorical diagnoses, such as those proffered in the Diagnostic and Statistical Manual of Mental Disorders (DSM IV; American Psychiatric Association [APA], 1994) or in the International Classification of Diseases (ICD-10; World Health Organization, 1996).

Stated simply, depressive disorders, also referred to as mood disorders, involve disturbances of emotion that affect an individual’s entire psychic life (IOM, 1994). There are two types of mood disorder. These are bipolar disorder, which we do not address in this article, and depressive disorder, which has two major subtypes: MDD, marked by a single episode or recurrent episodes of depression; and dysthymia (DD), which involves a chronic disturbance of mood. Depressive disorders are characterized by a pervasive mood disturbance that involves feelings of sadness and loss of interest or pleasure in most activities in conjunction with disturbances in sleep, appetite, concentration, libido, and energy. Efforts have been made to recognize that symptoms of these disorders may be manifested differently in children and adolescents than in adults (APA, 1994; Birmaher, Ryan, Williamson, Brent, Kaufman, Dahl, et al., 1996; Kovacs, 1996). However, most often the criteria associated with adult depression have been applied to children, and developmental considerations that may affect the etiology, course, and outcome of depression in children and adolescents have been minimized or disregarded entirely.

Depressive Disorders in Childhood and Adolescence

Although the mood disorders of children and adolescents have been investigated for a shorter period of time than the mood disorders of adults, nonetheless, in recent decades there has been a proliferation of research activity.
in the area of childhood and adolescent depression. In contrast to earlier beliefs that called into question whether or not veridical depressive illness could occur before puberty (Rie, 1966), contemporary emphases have shifted from a focus on debating which criteria should be used to diagnose childhood mood disorders to more sophisticated examinations of the epidemiology, causes, course, sequelae, and treatment responses of depressed and/or dysthymic children as well as of children who are considered to be at risk for depression because they have one or more relatives with a mood disorder (Birmaher, Ryan, Williamson, Brent, Kaufman, Dahl, et al., 1996; Cicchetti & Schneider-Rosen, 1986; Downey & Coyne, 1990; Kovacs, 1996; Puig-Antich, 1986; Todd, Newman, Geller, Fox, & Kickock, 1993; Weissman et al., 1987).

Epidemiology and Clinical Characteristics of Child and Adolescent Depression

Estimates of the point prevalence of MDD range from 0.4% to 2.5% for children and from 0.4% to 8.3% for adolescents (Birmaher, Ryan, Williamson, Brent, Kaufman, Dahl, et al., 1996; Cicchetti & Schneider-Rosen, 1986; Downey & Coyne, 1990; Kovacs, 1996; Puig-Antich, 1986; Todd, Newman, Geller, Fox, & Kickock, 1993; Weissman et al., 1987). Adolescent MDD occurs twice as frequently in teenage girls, paralleling the gender ratio obtained for adult MDD (Fleming & Offord, 1990; Lewinsohn, Clarke, Seeley, & Rohde, 1994).

The consequences of depression during childhood and adolescence cannot be minimized. Depressive disorders are neither normal developmental occurrences nor short-lived problems that dissipate with time (Kovacs, 1989). Even when episodes remit, they commonly recur and interfere with children's ability to function competently (Kovacs, Feinberg, Crouse-Novak, Paulauskas, & Finkelstein, 1984; Kovacs, Feinberg, Crouse-Novak, Paulauskas, Pollock, & Finkelstein, 1984). Moreover, when DD as compared to MDD is the first to emerge in children, there is a greater risk for developing subsequent mood disorders. For example, Kovacs and colleagues (Kovacs, Akiskal, Gatsonis, & Parrone, 1994) found that 76% of children with earlier onset DD developed a subsequent MDD, and 69% of the children with DD as the first emergent mood disorder developed a combined DD and depression (i.e., double depression). In a recent review of the literature, Birmaher and colleagues (Birmaher, Ryan, Williamson, Brent, Kaufman, Dahl, et al., 1996) concluded that the average length of an episode of MDD in children and adolescents was seven to nine months. Approximately 90% of MDD episodes remit within two years post onset, whereas the remaining episodes last for a more protracted period of time. Additionally, MDD frequently recurs in children and adolescents.

As compared to MDD, DD has a much more prolonged course, with the average length of an episode being four years. Children with DD generally experience their first MDD episode two to three years after the onset of DD. Because the development of DD is one of the major pathways to recurrent depressive disorder, early identification and treatment and the implementation of preventive interventions for DD are critical strategies that should be used (Kovacs et al., 1994).

With respect to comorbidity, 40% to 70% of depressed children and adolescents develop an additional, or comorbid, disorder, with 20% to 50% estimated to have two or more comorbid diagnoses. The most frequent comorbid diagnoses include DD, anxiety disorder, disruptive disorder, and substance abuse (Harrington et al., 1996; Kovacs, 1989, 1996). In children and adolescents, the majority of anxiety disorders (typically separation anxiety disorder) precede the depressive episode, whereas in adults, depression usually predates the anxiety disorder (Kovacs, 1996). MDD typically precedes the onset of alcohol or substance abuse by approximately four and a half years, thereby providing an important time window for the prevention of substance abuse in depressed adolescents (Birmaher, Ryan, Williamson, Brent, Kaufman, Dahl, et al., 1996). In general, comorbid diagnoses appear to enhance the risk for recurrent depression and to affect the duration of the depressive episode, suicide attempts, functional outcome, response to treatment, and the use
of mental health services (Birmaher, Ryan, Williamson, Brent, & Kaufman, 1996; Kovacs et al., 1994).

**Gender Differences**

From a developmental perspective, gender differences in depression are especially important because knowledge of gender-specific pathways may enhance our understanding of the etiology of depression as well as help to guide preventive approaches (Gjerde, 1995). Although not all studies of depressed youth have revealed gender differences (cf. Leadbeater, Blatt, & Quinlan, 1993), research suggests that at some point in early-to-middle adolescence, the overall prevalence of depressive symptoms increases significantly for both sexes, but that girls begin to manifest significantly higher rates of depressive symptoms (Angold & Rutter, 1992; Nolen-Hoeksema & Girgus, 1994). In moving beyond studies of depressed symptomatology to actually examining clinically relevant depression, three longitudinal investigations of community samples found age and gender effects consistent with expectations that, beginning in adolescence, girls are more likely to experience depression than boys (Giaconia et al., 1993; Kashani et al., 1987; McGee et al., 1990). A number of explanations have been proffered to explain this gender difference. One obvious hypothesis has invoked the role of biological changes associated with puberty. However, Angold and Rutter (1992) found that pubertal status did not predict depression beyond the effect of age. Nolen-Hoeksema and Girgus (1994) suggested that gender differences in personality or behavioral style (e.g., more rumination among girls) that may exist prior to adolescence interact with increased developmental challenges for girls in adolescence, resulting in the observed gender differences in rates of depression. Although more work is needed to gain a better understanding of the mechanisms that may contribute to these age and gender effects, a consensus regarding increases in depression during adolescence, especially in girls, has emerged.

**Concepts in a Developmental Psychopathology Approach to Depression in Children and Adolescents**

Given the prevalence of depressive disorders across extensive periods of development and the various risk factors associated with depression and other forms of psychopathology, it is essential to gain a firm grasp of the developmental processes that contribute to the emergence and perpetuation of depressive disorders. Depressive disorders are of particular interest to developmentalists because of the complex interplay of psychological (e.g., affective, cognitive, socioemotional, social-cognitive), social (e.g., community, culture), and biological (e.g., genetic, neurobiological, neurophysiological, neurochemical, neuroendocrine) components that are involved. Further, depressive conditions may be viewed as forming a spectrum of severity from transient and universally experienced dysphoria, to elevated levels of depressive symptoms that do not meet the diagnostic criteria for disorder, to extended periods of DD and episodes of MDD (Gotlib, Lewinsohn, & Seeley, 1995). Even within more narrowly defined disorders (e.g., MDD), there are likely to be heterogeneous conditions with phenotypic similarity despite differences in etiology. There are diverse pathways to depressive disorder, and potential risk factors for depression may result in a multitude of outcomes of which depression may be one. Moreover, depressive phenomena and disorders are present throughout the life span from early childhood through senescence. To structure our understanding of depression in childhood and adolescence, we next discuss major developmental principles central to both normal and abnormal patterns of development.

Developmental psychopathology seeks to unify diverse disciplinary perspectives to provide an understanding of multiple levels of individual adaptation and development, the interrelations and integrations of these varied systems across the life course, the spectrum of potential developmental pathways that evolve, and the causal processes contributing to these varied trajectories (Cicchetti, 1993). Depressive disorders constitute a particularly important area of study because of the diverse systems that influence these disorders. Aberrations in cognitive, socioemotional, representational, and biological domains are present to varying degrees among individuals with mood disorders (see Figure 1). Notably, these varied systems do not exist in isolation. Rather, they are complexly interrelated and mutually interdependent. In adaptively functioning individuals, there is a coherent organization among these domains. In contrast, in depressed individuals, there is either an incoherent organization among these systems or an organization of pathological structures, that is, a depressotypic organization. Depressotypic organizations evolve developmentally and may eventuate in depressive disorders at different points across the life course. Thus, understanding the interrelations among these systems is vital for delineating the nature of these disorders as well as for elucidating how these systems also promote adaptive functioning.

Given the multiplicity of systems affected by depressive disorders, the developmental approach serves to direct attention to the early developmental attainments that may be related to later appearing patterns of depressive symptomatology. For example, an understanding of the deviations in affective regulation or the core negative attributions about the self observed in depressed persons may emerge through an examination of the early development of these features, their developmental course, and their interrelations with other psychological and biological systems of the individual.

**Organizational Approach to Development**

In this regard, an organizational perspective on development has provided developmental psychopathologists with a valuable framework for conceptualizing develop-
mental phenomenon as they relate to the evolution of depressive disorders. At each stage, children are confronted with new developmental challenges (i.e., stage-salient developmental issues; Cicchetti & Schneider-Rosen, 1986; Sroufe, 1979; Sroufe & Rutter, 1984). The quality of the resolution of these stage-salient tasks influences how the particular developmental issue is incorporated into psychological and biological systems as reorganization occurs. Positive adaptation to a developmental challenge contributes to competence and better preparedness for adaptively resolving subsequent tasks of development. In contrast, compromised or inadequate resolution of a stage-salient developmental challenge, which is also integrated as reorganization proceeds, results in a decreased likelihood of positive adaptation to later developmental demands. Thus, although not inevitable, early competence tends probabilistically to foster later competence, and similarly, early incompetence tends to promote later incompetence.

Because the course of development is marked by considerable variability in outcomes, a diversity of developmental pathways is to be expected (Cicchetti & Rogosch, 1996). Multifinality specifies that diverse outcomes are likely to result from any one source of influence. For example, although children of parents with depressive disorders are considered at risk, including at genetic risk, for the development of depression, certainly not all such individuals develop depressive disorders, and a wide spectrum of adaptation is seen. Harrington and colleagues (1996), on the basis of research they conducted with clinical and epidemiological samples, concluded that depressive disorders represented a heterogeneous group of phenomena. Although children and adolescents with depressive disorder were more likely than comparable psychiatric controls to evidence depressive disorders in adulthood, individuals who also had comorbid conduct disorders tended to be less likely to develop depression in adulthood than those without conduct problems. The findings of Harrington et al. (1996) not only demonstrate a degree of specificity in adult outcomes for depressive disorders but also illustrate multifinality in developmental pathways through observing alternate patterns of adaptation among children and adolescents who had experienced depressive disorders.

In contrast to multifinality, the principle of equifinality suggests that the same outcome may emanate from diverse routes. Gjerde (1995) provided an illustrative example of equifinality in the development of chronic depressive symptoms by young adulthood as predicted through repeated assessments from the preschool years. Strong predictive patterns were evident for young adult men beginning in the preschool period, a time in which undersocialization and antagonistic interpersonal behavior were linked with depression in adulthood. Similar constellations of these personality characteristics in later childhood and adolescence also were related to chronic depressive symptoms in young adults. Conversely, among women this personality organization did not predict adult depression. Moreover, early precursors of depressive symptomatology were not identified for women, and it was not until adolescence that patterns of oversocialization and excessive introspection were found to predict
depressive symptoms in young women. Thus, very different trajectories to the same depressive outcome in adulthood were identified. In view of the varied pathways that are likely to result in depressive outcomes, a model that is sufficiently complex and comprehensive and that allows for the explication of precursors to depressive illness as well as for full blown depressive syndromes and disorders is essential.

**An Ecological Transactional Model**

To be able to account for the diverse influences on the emergence of depression, a transactional model that addresses the interrelations among dynamic biological, psychological, and social systems is necessary (Sameroff & Chandler, 1975). Such a model views the multiple transactions among environmental forces, caregiver characteristics, and child characteristics as dynamic, reciprocal contributions that may exacerbate or decrease the likelihood of a depressotypic organization and the emergence of a depressive illness. The application of a transactional model to the evolution and maintenance of depression requires that attention be directed to the risk factors associated with the development of the disorder. As such, the multiple factors that have been implicated in the etiology of depression must be understood. In accord with a transactional developmental formulation, it is likely that numerous general factors across broad domains of psychological and biological development will be related to the manifestation of depression as well as to positive adaptation in the context of risk for depression. Whether depression occurs is affected not only by the presence or absence of specific vulnerability or protective factors. Rather, the interplay that occurs between these factors and current and previous levels of adaptation as well as the developmental period during which risk factors are experienced (Cicchetti & Tucker, 1994; Sroufe, Egeland, & Kreutzer, 1990) also are vital contributors to depressive outcomes.

An ecological transactional model provides a framework for understanding how multiple factors can influence the emergence of depression in children and adolescents (see Figure 2). According to such a perspective, an individual’s ecology is seen as being comprised of a number of co-occurring levels, some of which are proximal to and others which are more distal to the individual (Belsky, 1993; Bronfenbrenner, 1979; Cicchetti & Lynch, 1993). Depending on how proximal the influence is to the individual, the role that it exerts on the emergence of a depressotypic organization and of a depressive disorder may be more or less evident. Processes from each level

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**Figure 2**

*Transaction of Multilevel Potentiating and Compensatory Processes in the Development of Depressotypic Organization and Depressive Outcomes*

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Distal Outcomes
- Dysthymia
- Major Depressive Disorder
- Double Depression
- Co-Morbid Disorders
- Positive Adjustment
of the environment as well as characteristics of the individual mutually influence each other over time and shape the course of child development, including whether or not a depressotypic organization and a depressive illness emerge.

Cicchetti and Lynch (1993) discuss the role that potentiating and compensatory risk factors exert at various levels of the ecology. Risk factors within a given level of the ecology also can influence outcomes and processes in surrounding ecological levels. The ongoing transactions among these risk factors determine the amount of biological and psychological risk that the individual faces. Potentiating factors increase the likelihood that a depressotypic organization and a depressive illness will occur, whereas compensatory factors decrease the probability of their occurrence. Each factor may exert enduring or transient influences that affect the probability of a depressive outcome. Importantly, the manner in which children deal with the challenges that impinge on them by family, community, and societal conditions is seen in their own ontogenic (i.e., individual) development. As enduring vulnerability factors and transient challengers at various ecological levels increase, the occurrence of a depressotypic organization and depression becomes more likely. Conversely, the presence of enduring protective factors and transient buffers at various ecological levels may help to explain why some children deal adaptively and avert depression even in the face of conditions seemingly linked to a depressotypic organization and depressive illness.

The two most distal levels of an individual’s ecology include the macrosystem, which contains the beliefs and values of the culture, and the exosystem, which includes aspects of the community in which children and families live. More proximal influences are exerted by the microsystem, or the immediate environment, most typically the family, and ontogenic development, or those factors within the person that affect his or her adaptation. In accord with a transactional approach, ongoing transactions of risk and protective processes within and among each level of the ecology are conceived as contributing to the emergence and development of a depressotypic organization and to the onset and recurrence of depressive disorders. To examine how depression may evolve, we discuss illustrative research relevant to the development of depressotypic organization and depressive illness that has been conducted on factors operating at different levels of the ecology.

Because the amount of research conducted at various levels of the ecology varies, in this article we place a greater emphasis on the ontogenic and microsystem levels, that is, those that are most proximal to the child or adolescent. Moreover, because we believe that it is how the individual copes with various influences and not any ecological factor per se that contributes to the emergence of a depressive illness, we first discuss ontogenic development and its relations to a depressotypic organization. Subsequently, we describe how the individual is embedded within progressively more distal levels of the ecologies and how risk and protective factors operating at levels of the ecology can serve to exacerbate or diminish the likelihood of a depressotypic organization and a depressive illness.

**Ontogenic development.** The quality of the resolution of early stage-salient developmental tasks may contribute to early aberrations that are hierarchically integrated and portend the development of pathways to depressive disorder through depressotypic organizations of developmental structures. For heuristic purposes, four early stage-salient developmental issues that bear theoretical relevance for diverse components of evolving depressotypic organizations and depressive disorders are highlighted. These issues include (a) the development of homeostatic and physiological regulation, (b) affect differentiation and the modulation of attention and arousal, (c) the development of a secure attachment relationship, and (d) the development of the self-system. As development proceeds, each of these issues sequentially reaches ascendancy and becomes a primary arena for which internal resources must be elaborated and extended to hierarchically integrate the imposed developmental challenges to foster positive adaptation. Moreover, rather than subsequently declining in importance, each issue remains a lifelong component of adaptation.

The research we discuss on aberrations in these areas is drawn from studies on the offspring of parents with depressive disorders, from investigations on children and adolescents with mood disorders, and from population-based epidemiological studies that have focused on depressed symptoms and depressive disorders. Although the majority of work has been conducted on children with depressed parents, it is important to note that there are clear linkages between families having a depressed parent and those with a depressed child. Specifically, a significant proportion of children with a depressed parent also are depressed (Downey & Coyne, 1990). In addition, relations in the time of the occurrences between episodes of depression in mothers and children have been found (Hammen, 1991), and one third to one half of parents with a depressed child are themselves depressed (Brumbeck, Dietz-Schmidt, & Weinberg, 1977; Puig-Antich et al., 1989).

**Homeostatic and physiological regulation.** In the early months of life, the infant is challenged to maintain homeostatic equilibrium of internal physiological states. Homeostatic systems strive to maintain a set point of functioning, and departure from this optimal level generates tension. Consequently, behavioral and biological systems must be activated to reduce the tension, thereby helping the infant to regain a state of equilibrium. Early innate motoric reflexes allow the infant some capacity to regain equilibrium. However, significant environmental support must be provided for the infant to regulate physiological states and maintain equilibrium. Thus, early physiological regulation requires support from caregivers. Infants develop capacities to communicate their
needs to caregivers through affective responses, and sensitive caregivers must be able to read these signals accurately.

As the infant's brain develops, the infant becomes increasingly self-sufficient in modulating arousal generated by physiological tension. This growing capacity corresponds to maturation of forebrain inhibitory tracts and neurotransmitter systems, which allows increasing control of lower hindbrain and midbrain limbic structures. Right-brain activation has been associated with distress, whereas left-brain activation and inhibition of right-brain activity have been linked to positive affect (Davidson, 1991). The development of interhemispheric connections enhances the infant's ability to self-regulate (Cicchetti & White, 1988; Tucker & Williamson, 1984). The development of these neurological systems is experience expectant, necessitating external input from caregivers (Greenough, Black, & Wallace, 1987). Moreover, the quality of caregiving received is likely to contribute to variations in neurobiological growth and development, resulting in long-term effects on the organization and development of the brain.

Parents vary in how well they are able to assist their infants in the maintenance of homeostatic regulation, thereby indirectly influencing the process of brain development. Further, extremely frequent, novel experiences and an unstable environment may more routinely activate the right brain, resulting in negative affect expression. In contrast, stability and consistency in the environment may support dominance of the left brain, which may strengthen the inhibitory effects on negative arousal. Thus, the quality of parenting may influence the development of interhemispheric connections and the emotion regulatory abilities that the infant develops.

Differences in homeostatic regulation have been investigated in infants of parents with mood disorders, where as early as the neonatal period, difficulties have been noted. These include elevated levels of epinephrine and norepinephrine, difficulties with self-quieting, lower activity levels, more negative affect, and attentional problems, all of which are suggestive of more difficult temperaments (Abrams, Field, Scafidi, & Prodromides, 1995; Field, 1992; Sameroff, Seifer, & Zax, 1982).

Even simulated depression portrayed by nondepressed mothers has been shown to result in negative infant affectivity and disruption in the infant's effective self-regulation (Cohn & Tronick, 1983). In more recent investigations, a number of problematic depressed maternal interactive behaviors have been observed. Three distinct interactive profiles have been identified in depressed mothers, including (a) a withdrawn, unavailable, and understimulating pattern; (b) a hostile-intrusive overstimulating pattern; and (c) a positive pattern characterized by the absence of depressed symptoms on maternal self-report inventories of depression (Cohn, Matias, Tronick, Lyons-Ruth, & Connell, 1986; Field, Healy, Goldstein, & Getherz, 1990). Cohn and his colleagues (1986) speculated that restricted maternal affect and lack of responsiveness eventuated in increased infant distress, whereas maternal hostility and intrusiveness were associated with more infant avoidance.

In addition to the importance of the affective aspects of mother–infant interaction, infants also have shown dramatic reactions to violations of the expected temporal relationship between their own and their mother's positive affect (Cohn & Campbell, 1992). When normal mothers were instructed in a laboratory analogue simulation to sober in response to their infant's positive affect displays, the babies were found to manifest sober expressions and to avert gaze from their mother (Cohn & Elmore, 1988). Clearly, the temporal aspects of the affective displays of depressed mothers are likely to be quite impaired. Bettes (1988) examined the vocal utterances of mothers varying from mild to moderate in their level of depressed symptoms. She found that the vocalizations of mothers with some degree of depressed symptomatology were more variable and their vocalizations were of longer duration than was the case in the nondepressed mother control groups.

Beyond infancy, affect regulatory difficulties also have been observed among older offspring of parents with mood disorders. Jameson and colleagues (Jameson, Gelfand, Kulcsar, & Teti, 1997) found that depressed mothers were less likely to repair interactions with their toddlers than were nondepressed mothers with their toddlers. Additionally, the toddlers of the depressed mothers were less likely to maintain interactions with their mothers than were the toddlers of nondepressed mothers. Finally, nondepressed mothers and their toddlers displayed more interactive coordination than did depressed mothers and their toddlers.

Toddlers of mothers with MDD also have been shown to exhibit more disregulated, out-of-control behavior than toddlers of nondepressed mothers (Zahn-Waxler, Iannotti, Cummings, & Denham, 1990). These differences predicted behavior problems as perceived by mothers at age five and as reported by the child at age six, and were found to occur predominantly for children of depressed mothers who were less able to modulate, control, and provide structure and organization during their toddler's play.

Difficulties in the regulation of affect also may contribute to problematic peer relations. Zahn-Waxler and colleagues (Zahn-Waxler, Denham, Cummings, & Iannotti, 1992) noted that preschool-aged children of parents with mood disorder characteristically engage in uncontrolled and poorly regulated social exchanges with peers. Boys of depressed mothers have been found to generate aggressive strategies to solve hypothetical peer conflicts (Hay, Zahn-Waxler, Cummings, & Iannotti, 1992). Thus, these findings suggest that early regulatory difficulties in offspring of parents with mood disorders may continue to affect adaptation as new experiences and situations are encountered. These difficulties may portend problems in the modulation of affective reactions that may heighten the risk for the development of future depressive disorders. For example,
tension is cognitively generated by those evaluations of the environment on the basis of past experiences, and (e.g., as threatening, overstimulating; Sroufe & Waters, 1979). However, increasingly, the infant now also evaluates the presence. In a related vein, depressed mothers who had received psychotherapy. In particular, depressed mothers not in treatment had difficulty recognizing negative emotions such as anger and sadness (Free, Alechina, & Zahn-Waxler, 1996). Thus, infants growing up with depressed mothers are likely to experience aberrant affective interchanges that contribute to divergences in their early affect development. These early affect differences provide further impetus for an evolving depressotypic organization.

The development of a secure attachment relationship. The development of an attachment relationship with the primary caregiver during the latter half of the first year is a fundamental achievement that organizes evolving affect, cognition, and behavior in relation to the quality of physical and emotional availability of the caregiver. On the basis of evolutionary needs to maintain safety from environmental threats, the caregiver provides a secure base that helps the infant to modulate arousal.
and maintain internal security (Bowlby, 1969/1982). Variation in the quality of caregiving, particularly in terms of sensitivity and responsivity, contributes to individual differences in the manner in which the infant negotiates the attachment relationship with the primary caregiver. Although infants may experience different types of relationships with various caregivers, the attachment relationship with the primary caregiver, typically the mother, is very important in influencing the way in which affect, cognition, and behavior are organized. Ainsworth and colleagues (Ainsworth, Blehar, Waters, & Wall, 1978), using a laboratory procedure involving a sequence of separations and reunions between the infant and the caregiver, identified three major types of attachment organization that can differentiate between securely and insecurely attached infants. The traditional classifications include Type B, securely attached, and two types of insecurely attached infants, Type A, insecure–avoidant, and Type C, insecure–ambivalent. Further investigation, particularly involving high-risk samples, led to the identification of additional atypical insecure attachment patterns involving a blending of both avoidant and resistant behaviors (Type A/C; Crittenden, 1988) or a disorganized–disoriented pattern (Type D; Main & Solomon, 1990), including a variety of undirected behavioral responses and unusual behaviors such as freezing, stilling, hand flapping, and other stereotypies exhibited in the presence of the caregiver. The traditional and atypical attachment classifications represent individual differences in the strategies that infants use to regulate emotions and behavior and are related to the history of distress remediation and emotional synchrony experienced with the caregiver.

The differences in attachment types are important for understanding early forms of divergent organization of socioemotional, cognitive, representational, and biological systems that may relate to an emerging depressive-typic organization. As development proceeds beyond infancy, the experience of the attachment relationship increasingly becomes internally represented. As such, representational models serve to channel the manner in which interpersonal relations are perceived and negotiated as well as the accompanying affects and cognitions that are exhibited.

Children of depressed caregivers are at risk of experiencing deviations in care as a consequence of their caregivers' struggles with depression. Additionally, these children may experience a sense of loss akin to the actual loss of a parent (Bowlby, 1980) during periods of caregiver depression. Resultant insecure representational models may make it more difficult for these children to cope with the experience of psychological unavailability of the caregiver during depressive episodes. Prolonged anxiety, sustained grieving, and difficulty in resolving the loss may further contribute to problems in the organization of cognitive, affective, representational, and biological systems. Subsequent loss experiences, either real or symbolic, may precipitate depressive episodes (Beck, 1967).

The quality of attachment in infants and children of parents with mood disorders has been examined in a number of studies (DeMulder & Radke-Yarrow, 1991; Radke-Yarrow, Cummings, Kuczynski, & Chapman, 1985; Teti, Gelfand, Messinger, & Isabella, 1995). Overall, investigations have found that offspring of depressed caregivers are significantly more likely to develop insecure attachments with their caregivers. Importantly, those studies that have not found increased rates of insecurity in offspring of mood-disordered mothers have emphasized the role of severity and chronicity of maternal depression in affecting child outcome (Cohn & Campbell, 1992), with offspring of mothers who had more transient depressive episodes appearing to be more comparable to offspring with well mothers. Issues associated with sampling also have been raised as accounting for variability of results, with differences being associated with hospitalized versus community samples of depressed mothers.

Studies that followed offspring into later childhood found that children who continued to evidence insecure attachments with their caregivers were more likely to exhibit behavior problems (Easterbrooks, Davidson, & Chazan, 1993). Insecure attachments among children of depressed mothers also have been found to contribute to interpersonal difficulties as these offspring negotiate relationships with peers. Rubin and colleagues (Rubin, Booth, Zahn-Waxler, Cummings, & Wilkinson, 1991) found that insecurely attached offspring of depressed mothers exhibited withdrawal, passivity, and inhibited behavior when observed with a familiar peer in free play at age five.

During adolescence, clinically depressed teenagers, as compared with normal or nondepressed psychiatric controls, have been found to express less secure attachments to their parents (Armsden, McCauley, Greenberg, Burke, & Mitchell, 1990). Similarly, Kandel and Davies (1986) found that depression in adolescence was related to problems in the emotional relationship with parents, difficulties in forming an opposite-sex relationship, and spousal difficulties in young adulthood. In late adolescence, an insecure attachment organization has been linked to higher levels of depressive symptomatology (Kobak, Sudler, & Gamble, 1991) and to more interpersonal difficulties during the transition to college (Kobak & Scerey, 1988). Among mildly depressed college women and those recovering from major depression, Carnelley, Pietromonaca, and Jaffe (1994) found insecure relationships with parents were frequent and romantic relationships were often characterized by preoccupation, fearful avoidance, or both. The attachment organization of these women more strongly predicted relationship functioning than did their depression status alone.

In summary, there is growing evidence of insecure attachment organizations among offspring of parents with mood disorders as well as among youth with depressive disorders. The quality of early attachment relationships contributes to internal representational models of self and
other that organize cognition, affect, and behavior, and these models serve to canalize perceptions and experiences as development proceeds. In the case of individuals with insecure attachment organizations, their internal representational models are likely to contribute to a depressotypic organization of psychological and biological systems. Affective regulation and expression are less optimal, and significant others are perceived as unavailable or rejecting while the self is regarded as unlovable. These attachment-related aspects of a depressotypic organization may contribute to a proneness to self-processes that have been linked to depression (e.g., low self-esteem, helplessness, hopelessness, negative attributional biases).

The self-system: Self-awareness and self-other differentiation. Toddlerhood may be a particularly sensitive period for the formation of a depressotypic organization because many of the social, emotional, and cognitive competencies implicated in the development of later depressive disorder are at crucial stages of development during toddlerhood (e.g., the development of autonomy, the emergence of the affect of shame, and the construction of an internal representational model of the availability of the self and of the self in relation to others). Building on the quality of the attachment relationship that has evolved, toddlers begin to develop a sense of themselves as separate and independent entities in the second half of the second year (Lewis & Brooks-Gunn, 1979). Growing capacities for language and play during the second and third years of life constitute a means through which children symbolically represent the self and relationships. Increasingly, children are able to use symbolic means to communicate needs and feelings and evidence increased abilities to label emotion states, intentions, and cognitions of self and others (Kagan, 1981). These representational attainments also correspond to increased capacities for self-regulation.

Emotional and cognitive components are integrated into internal representational models in which the self becomes represented as does the self’s relation to the attachment figure (Bretherton, 1987; Sroufe, 1990). Although the growing toddler increasingly acquires capacities for self-regulation, parental involvement remains vital, and parental availability and responsivity influence how the self is represented. Caregiver accessibility and responsivity correspond to self-representations as acceptable and valued, whereas parental unavailability or rejection relate to self-representations as unlovable and unworthy.

Evidence has been obtained that demonstrates difficulties in self-development and corresponding affective functioning in toddlers of mothers with mood disorders. The self-critical styles of depressed caregivers appear to be transmitted to their offspring. One such mode of transmission relates to the speech used by depressed parents. Murray and colleagues (Murray, Kempton, Woolgar, & Hooper, 1993) found that the speech used by depressed women to their infants expressed more negative affect, was less focused on infant experience, and evidenced less acknowledgement of infant agency. Even in adolescence, increased irritability has been observed in the verbal interchanges between affectively ill mothers and their adolescents (Tarullo, DeMulder, Martinez, & Radke-Yarrow, 1994).

In addition, maternal attribution patterns may affect the types of self-attributions that children make. For example, Radke-Yarrow and colleagues (Radke-Yarrow, Belmont, Nottelmann, & Bottomly, 1990) found that mood-disordered mothers conveyed significantly more negative affect in their attributions, particularly in regard to negative attributions about child emotions. Moreover, among the mood-disordered mothers and their toddlers, there was a higher correspondence in the affective tone of attributions and statements about the self (e.g., mother says, “I hate myself,” child says, “I’m bad”). This suggests a heightened vulnerability among these children for negative self-attributions, with negative implications for risk for later depressive tendencies.

Further evidence for self-system dysfunction in offspring of depressed caregivers was found by Cicchetti and colleagues (Cicchetti, Rogosch, Toth, & Spagnola, in press), who examined visual self-recognition in toddlers of depressed and nondepressed mothers, by using the mirror-and-rouge paradigm (cf. Lewis & Brooks-Gunn, 1979). In this paradigm, after toddlers look at themselves in a mirror, an experimenter surreptitiously places a dot of rouge on the toddler’s nose while the toddler is looking away from the mirror, and the toddler reexamines his or her rouge-altered mirror image. Although self-recognition was attained similarly in both groups, children of depressed mothers who exhibited self-recognition were more likely than children of nondepressed mothers to display nonpositive affect and to shift affect from positive to nonpositive after the rouge application. In addition, within the group of children of depressed mothers, Cicchetti et al. (in press) found that toddlers who did not evidence self-recognition and who shifted affect were lower in attachment security and had mothers with fewer positive affect characteristics.

As development proceeds, early representational features of the self are further elaborated, and these aspects of self-representation possess implications for understanding a depressotypic organization. Self-understanding constitutes cognitive representations of the self with roots in the internal representational models of the self derived from attachment relationships. In contrast, self-esteem represents an affective component of the self that is positively or negatively valenced. Self-cognitions are particular usages of the cognitive structure in reference to the self. They have both content, in terms of what aspects of the self are the focus, and style, involving the manner in which thoughts about the self are derived. Self-cognitions and self-esteem mutually influence each other, and when self-cognitions and associated affects are repeated over time, they contribute to representations of the self known as self-schemata that tend to be enduring.
Important developmental shifts have been noted with respect to the complexity of self-cognitions, with a significant transition occurring in the seven- to eight-year-old range (Harter, 1983). Younger children view themselves in concrete, physical terms such as their appearance, possessions, and preferred activities, whereas older children begin to view themselves in more abstract psychological terms that include personal characteristics that are enduring over time. These advancing abilities may have negative consequences for children who are at risk for an affective disorder (Damon & Hart, 1982).

Nolen-Hoeksema, Girgus, and Seligman (1992) investigated children with depressed symptoms and their negative life events, explanatory style, and helplessness beginning in third grade and followed these children for five years. In early childhood, only negative events were related to depressive symptoms. However, later in childhood and notably after the cognitive shift previously described had occurred, a pessimistic explanatory style (cf. Peterson & Seligman, 1984) contributed to depressive symptoms, alone or in combination with negative events. In both interpersonal and achievement contexts, the depressed children exhibited helplessness. Moreover, their negative explanatory style worsened during depressive episodes, and their pessimism persisted subsequently.

Depressed children may maintain excessively high expectations for themselves, contributing to attributions of failure when those expectations are not met. Lauer and colleagues (1994) investigated memory and metamemory abilities among nine- to twelve-year-old depressed children. Only severely depressed children exhibited memory impairments, but all depressed children were found to have performance difficulties on metamemory tasks, involving an overestimation of their abilities. Depressed children appeared to be either overcompensating for feelings of inferiority or setting unrealistic standards for themselves, which would tend to confirm their negative self-cognitions and sense of failure.

Research on the cognitive components of depression in children has generally been consistent with findings from the adult literature (for a review, see Garber, Quiggle, & Shanley, 1990). Depressed children have been found to process negative self-referent words selectively, to make more internal, stable, and global attributions for failure and more external, unstable, and specific attributions for success, to perceive outcomes as being beyond their control, and to view their futures as hopeless. Moreover, these cognitive processes have been found more commonly in depressed children than in children with other psychopathological conditions, suggesting that there may be some specificity to the cognitive processing associated with depression (Garber, Quiggle, Panak, & Dodge, 1991). In fact, in a prospective analysis of the relation between negative self-cognitions and depression, Hammen (1988) found that children who had more negative self-concepts became more depressed than children who had more positive self-views, even when initial depression was controlled for.

A number of major psychological theories of adult depression have focused on the cognitive processing of depressed persons (Beck, 1967; Rehm, 1977). To integrate aspects of social information processing and various knowledge structures into a developmental framework that is useful for understanding child and adolescent depression, Dodge (1993) hypothesized that early experience and biologically based aspects of memory and neural functioning interact to form schemata for past experiences, future expectations, and affectively charged vulnerabilities. Dodge believes that early life experiences involving interpersonal loss and instability or excessive pressure to achieve at an unrealistic level may lead children to develop negative self-schemata and low self-esteem.

Disturbances in the self-system also can contribute to suicide in depressed individuals. In examining the cause of adolescent suicide, Chandler (1994) proposed that the identity formation demands accompanying the transition to adolescence can adversely affect the sense of identity needed to maintain an investment in the future. Consequently, Chandler (1994) believed that this loss of identity may contribute to suicide. To test this hypothesis, Chandler grouped hospitalized adolescents as being at either low or high risk for suicide. In accord with his assumptions, Chandler (1994) found that over 80% of the high-risk adolescents were unable to identify any means of justifying their own or others’ self-continuity in the context of change as opposed to 8% and 0% of low-risk adolescents and nonpatient controls, respectively.

**Developmental Biological Systems in Depression**

The development of a mood disorder, as well as the age of its onset, is influenced not only by the emergence of salient issues or tasks that must be confronted but also by timed biological events that create challenges and provide new opportunities as they figure prominently in every developmental phase. A number of investigations have shown that there is a greater prevalence of mood disorders in the relatives of depressed persons than in the general population (Weissman, Warner, Wickramaratne, Moreau, & Olsson, 1997) and a higher probability of disorder among relatives who are more closely related (McGuinn & Katz, 1989; Tsuang & Faraone, 1990). Moreover, twin studies reveal greater concordance of depressive disorder in monozygotic rather than dizygotic twins (McGuinn & Katz, 1989). Adoption studies also have been used to disaggregate shared genetic and environmental influences. These studies have shown increased rates of depression in biological relatives as compared with adoptees (Cadolet, 1978; McGuinn, Katz, Watkins, & Rutherford, 1996). Regardless, developmental geneticists maintain that genetic contributions to psychopathological disorders must be conceptualized within a dynamic framework that considers the operation of genetic factors in concert with environmental factors across the life span (Goldsmith, Gottesman, & Lemery, 1997;
Rutter, 1991). Genes are unlikely to operate in a static fashion throughout development. Rather, their influence may vary across the life course. Although some genes' effects may be enduring, others may be transient. At varying developmental periods, genes may be turned on or off, and diverse factors that regulate gene activation and deactivation are likely to vary developmentally. Although genes may influence the development of early structures (e.g., receptors for neurotransmitters) that influence normal and pathological dispositions, later gene activation and deactivation (as well as experience) also may modify those structures at subsequent periods in ontogenesis (Cicchetti & Tucker, 1994). Accordingly, the changing relative influence of genetics and environment at different stages of the life course within varying individuals must be incorporated into developmental models of depressive disorders and evolving depressotypic developmental organizations.

A number of investigations have examined various biological structures and processes among depressed children and adolescents and nondepressed controls (see Birmaher, Ryan, Williamson, Brent, Kaufman, Dahl, et al., 1996; Dahl & Ryan, 1996). Steingard et al. (1996), using magnetic resonance imaging (MRI), found decreased brain frontal-lobe volume and increased lateral ventricular volume in a sample of hospitalized children with depressive disorder compared with a group of psychiatrically hospitalized nondepressed controls. These findings are congruent with similar results using MRI techniques in adults with MDD.

Variations from normal patterns of growth hormone (GH), prolactin secretion, and serotonergic functioning also have been observed in response to various psychopharmacological challenges among children and adolescents with depressive disorders (Birmaher et al., 1997; Dahl & Ryan, 1996). The change in GH secretion and serotonergic regulation may be one component of a depressotypic organization that portends an earlier onset of depression (cf. Dahl & Ryan, 1996). Although difficulties have been observed in hypothalamic-pituitary-adrenal (HPA) axis regulation in adults with depressive disorder (Gold, Goodwin, & Chrousos, 1988a, 1988b), inconsistent results have been found in studies of depressed children and adolescents (Birmaher, Ryan, Williamson, Brent, Kaufman, Dahl et al., 1996). Despite these equivocal findings, subsets of depressed children and adolescents have been identified in which early alterations in HPA functioning may predict a recurrent course of unipolar depressive disorder in adulthood (Rao et al., in press).

Lateralization of neurotransmitter systems and hemispheric asymmetries may influence arousability to stimulation and individual differences in emotion processing. Through the interaction of noradrenergic and serotonergic systems, the right hemisphere is sensitive to change, alerts the brain to novelty in the environment, and thus is associated with general arousal and brain activation. In contrast, through domination by dopaminergic and cholinergic systems, the left hemisphere is biased toward redundancy, being relatively influenced by novelty to maintain behavior in the face of change (Tucker & Williamson, 1984). Electroencephalogram (EEG) studies suggest that negative emotional states correspond to relatively less left-frontal or greater right-frontal activation or both, whereas positive emotional states correspond to the opposite pattern of activation.

A number of investigations have discovered hemispheric activation asymmetries in infants of depressed mothers. Jones and colleagues (Jones, Field, Fox, Lundy, & Devalos, 1997) found that one-month-old infants of depressed mothers exhibited greater relative right-frontal EEG asymmetry (due to reduced left-frontal activation) than did one-month-old infants of nondepressed mothers. Moreover, the presence of right-frontal hemispheric asymmetry was significantly related to three-month EEG asymmetry. In addition, Field, Fox, Pickens, and Nawrocki (1995) discovered that depressed mothers and their three- to six-month-old infants both displayed right-frontal EEG asymmetry. Furthermore, Dawson and her colleagues (Dawson, Grofer Klinger, Panagiotides, Hill, & Spieker, 1992; Dawson, Grofer Klinger, Panagiotides, Spieker, & Frey, 1992) examined the EEGs of 14-month-olds of mothers with elevated depressive symptomatology and of nonsymptomatic mothers during various emotion-eliciting situations. Evidence was found that infants of the symptomatic mothers displayed reduced left-frontal brain activation during baseline and playful interactions. Moreover, securely attached infants of symptomatic mothers evidenced this left-frontal hyoactivation compared with securely attached infants of nonsymptomatic mothers. Further, during distress-eliciting maternal separation, the infants of the symptomatic mothers did not display a greater right-frontal activation or the same degrees of distress that was seen in the infants of the nonsymptomatic mothers, and these differences were observed regardless of the attachment status of the high-risk infants. Dawson and her colleagues interpreted these findings to suggest that both maternal depressive symptoms and attachment security are reflected in infant frontal lobe functioning and emotional behavior. In tandem, the results of the Jones et al. (1997), Field et al. (1995), and Dawson, Grofer Klinger, Panagiotides, Spieker, and Frey (1992) investigations support the view that a genetic diathesis for depression and the quality of caregiving experienced both have an impact on neurobiological development. Convergence between biological and psychological systems is suggested, providing a window on the complexity of developmental organization that may heighten risk for depression.

The microsystem. Because research has found that depression tends to run in families, it is not surprising that a considerable amount of work has been directed toward the family context in which depression occurs. Although genetic factors account for some within-family depression, it is clear that genetics alone cannot explain fully the development of depression. Support for influ-
ences beyond genetics have been obtained in studies of twins, where the heritability of severe and moderate depression has been found to be only modest (Kendler, Neale, Kessler, Heath, & Eaves, 1992). Moreover, Rende, Plomin, Reiss, and Hetherington (1993), in an investigation of the relative contribution of genetic and environmental factors to depressive symptomatology in an unselected sample of adolescents participating in a combined twin and stepfamily study, found moderate genetic influence for the full range of individual differences in depressive symptoms. In addition, Rende et al. (1993) discovered that there was nonsignificant genetic influence and significant shared environmental influence on extreme depression symptomatology. In view of findings such as these, environmental contributions to depression must not be minimized.

Because children are often very helpless to alter the environments with which they are faced, contextual influences may be even more significant in early-onset depressions. The fact that many depressed children evidence prompt recovery when hospitalized, even in the absence of additional interventions, lends further credence to family influences on depression (Kashani et al., 1987; Puig-Antich et al., 1987). Rather than seeking to ascertain whether genetic or environmental influences contribute to depressive illness, we must strive to understand how genetic and other biological vulnerabilities interact with contextual influences to eventuate in an evolving depressotypic organization and in depressive disorders during childhood and adolescence.

Investigations of family variables associated with child and adolescent depression have focused on two primary areas: families of depressed children and children of depressed parents. A number of family characteristics have been associated with the development and maintenance of depression, including parental psychopathology, family structure, and negative life events (Kaslowsky, Deering, & Racusin, 1994). Specifically, a high incidence of psychopathology has been found in parents and extended family members of depressed children, with mothers and fathers of depressed children exhibiting increased levels of depression, anxiety, substance abuse, and antisocial behavior (Kutcher & Martin, 1991; Puig-Antich et al., 1989; Todd et al., 1993). The fact that depressed persons tend to marry partners who also experience psychological difficulties (i.e., assortative mating) most likely increases the incidence of psychopathology in both parents of a depressed child or adolescent (Merikangas, Prusoff, & Weissman, 1988). In examining family structure, girls from single-parent families and children of divorced parents have been found to exhibit increased levels of depression and anxiety as well as slower rates of recovery when they do experience a depressive episode (Feldman, Rubenstein, & Rubin, 1988; Hoyt, Cowen, Pedro-Carroll, & Alpert-Gillis, 1990; Wallerstein & Corbin, 1991). Moreover, low socioeconomic status also has been linked with an increased risk for depression (Garrison, Schluter, Schoenbach, & Kaplan, 1989; Gibbs, 1985). Acute and chronic life events, most typically involving significant losses through parental death, divorce, or separation, or involving child maltreatment, also have been associated with the occurrence of depression during childhood and adolescence (Burbach & Bornstein, 1986; Hoyt et al., 1990; Toth, Manly, & Cicchetti, 1992).

To gain knowledge of microcontextual influences on depression, many investigators have studied children reared in families with a depressed caregiver. In addition to possible genetic influences, the overarching rationale of such studies is based on the premise that depression in a caregiver results in permutations in the caregiver environment that may place a child at risk for maladaptation, including depressive illness. Overall, studies have found that children of depressed parents evidence increased rates of general problems in adjustment, putative markers of risk for depression, and clinical depression (Downey & Coynne, 1990). However, these linkages cannot be taken as confirmatory evidence that parental depression necessarily leads to these problems or even that it is the primary influence. Rather, the interpersonal context within which a depressed caregiver resides and its affect on children must be considered. Specifically, marital discord and family stress are much more common in families with a depressed caregiver (Downey & Coynne, 1990). Additionally, social impairments other than diagnosis also are more common in depressed persons and their spouses. Thus, co-occurring risk factors other than depressive illness may account for the difficulties experienced by children with depressed caregivers.

Although much work remains regarding the specification of family variables that are related to a depressotypic organization and depressive disorder in childhood and adolescence, it is clear that the family environment can exert significant influences on the development and maintenance of early-onset depression. In accord with an ecological transactional model, the role of such factors must be conceptualized along with other psychological, social, and biological mechanisms that are operating at other levels of the ecology.

The exosystem. Relative to research on ontogenic development and microsystem influences on an evolving depressotypic organization and on depressive disorders, there has been a paucity of research addressing the role of the exosystem in the etiology of depression. However, evidence has accumulated to suggest that, in addition to the family influences just described, schools and neighborhoods contribute to patterns of academic and psychological adjustment, especially during the transition from elementary to middle schools (National Research Council, 1993). Therefore, the school environment is likely to be implicated in the development of depression. This view is corroborated, at least partially, by the fact that certain forms of psychological distress, including depressive symptoms, increase during the middle-school years. Perceptions of being academically competent and receiving good grades have been linked with a
reduced risk for emotional and behavioral difficulties, whereas low perceived academic competence is related to depressive symptoms in children (Blechman, McEnroe, Carella, & Audette, 1986; Cole, 1991). Moreover, adolescents who do not feel a connection with school may be more likely to engage in antisocial activities and substance abuse in efforts to boost their self-esteem and sense of belonging (Rosenberg, Schooler, & Schoenbach, 1989). In general, it has become increasingly clear that problems of academic alienation, poor school performance, and minor delinquency that become more prominent in early adolescence are linked to negative mental health problems such as depression that are manifested later in adolescence (Eccles, Lord, & Roeser, 1996). Such negative outcomes have been related to diminished support for the development of competence, for feelings of belongingness, and for autonomy promotion characteristic of elementary school, but not of middle-school, environments (Higgins & Parsons, 1983). Evidence such as this suggests that the failure of the school environment to facilitate development as children progress into middle schools may contribute to motivational and mental health problems. Inappropriate school environments during early adolescence can place many young people at risk for the negative trajectories that some lives take, including alienation from prosocial activities and peers, depression, and involvement in antisocial pursuits. Importantly, positive adjustment to school, including academic engagement and achievement, is likely to serve as a protective factor against negative mental health outcomes (Eccles et al., 1996).

The exosystem is the ecological level most directly linked to community supports that can be mobilized when a child or adolescent is depressed. Thus, in a supportive exosystem, high-quality treatment services would be readily available and their accessibility could serve to reduce the likelihood and chronicity of depressive illness in children and adolescents. To examine the potential efficacy of services, we address the extant literature on treatment for early-onset depression.

Unlike the management of adult depression, for which professional guidelines have been issued based on controlled treatment trials (APA, 1993; Depression Guideline Panel, 1993; Persons, Thase, & Crits-Christoph, 1996), compelling empirical work that can guide the recommended management of early-onset depression has not yet taken place (Kovacs, 1997). Interventions for depression during childhood and adolescence include two general classes: pharmacological and psychological.

Although virtually all medications found to be effective in the treatment of adult depression have been tested with children, systematic studies with clear results are rare. The drugs most commonly used for treating depression in children and adolescents are the tricyclic antidepressants, including imipramine, amitriptyline, and nortriptyline (Stark et al., 1996). In general, the superiority of antidepressant medication as opposed to placebo has not been proven as an effective treatment for children and adolescents (Kovacs, 1997; Stark et al., 1996). However, these negative results must be tempered because most studies have used very small samples, and this, as well as other methodological problems, may have clouded the findings (Birmaher, Ryan, Williamson, Brent, & Kaufman, 1996). For example, small sample sizes preclude controlling for the heterogeneity of depressive illness and therefore subgroups of depressed children and adolescents who may be more or less responsive to specific medications cannot be identified (Kye & Ryan, 1995). Moreover, unlike studies on adults, methodologically sound investigations on the relative effectiveness of antidepressants, psychotherapy, and a combination of these two types of therapy have not been conducted.

With respect to psychotherapy research with depressed youth, results have been more positive than research on pharmacological treatments. In general, therapies for the treatment of depression in children and adolescents have been found to be more effective than treatment wait-list or various comparison conditions (cf. Kovacs & Bastiaans, 1995; Weisz, Rudolph, Granger, & Sweeney, 1992). However, these positive results must be tempered because studies typically have included only nonreferred children with mild to moderate levels of depressive symptoms and not psychiatrically diagnosed patients. Moreover, almost all research has focused on middle- or upper-class nonminority youth (Lewinsohn, Clarke, & Rohde, 1994). Because more severe depression as well as depression accompanied by stressors associated with poverty and minority status most likely exacerbate difficulties encountered in the treatment of depression, much more research on treatment with diverse populations is needed. Additionally, evaluation studies have generally been conducted with behavioral or cognitive-behavioral approaches, and it is therefore unclear whether other therapies that are used with depressed children and adolescents are equally effective. Finally, much more work on the prevention of a depressotypic organization and of early-onset depression through psychological and pharmacological treatments is needed.

The macrosystem. At first glance, it may seem that cultural values and beliefs are unlikely to be related to an evolving depressotypic organization and to depressive disorders. However, there are aspects of the macrosystem that have been shown to exert influences on the emergence of depression. Moreover, because societal attitudes can affect the availability of resources and supports as well as the likelihood that treatment will be sought by families with a depressed child, the macrosystem can exert a significant impact on whether depression develops as well as on how it is addressed when it is present.

Relatively little research has been conducted on macrosystem influences and depression during childhood or adolescence. However, research on risk for suicide sheds some light on the role of culture and adaptation.
A number of findings on ethnicity also point to the role of culture and, specifically, to cultural changes as a risk for suicide. In Micronesia, suicide varies greatly by geographic location, with suicide rates being the highest in islands that have undergone a rapid transition from a traditional culture to a Western orientation (Brent & Morris, 1996). Similar factors appear to be operative in Native American populations, in which increased suicide is found on reservations where traditional cultural practices have eroded. Specifically, Levy and Kunitz (1987) found that Hopis at increased risk for suicide included the children of parents who had entered into traditionally disapproved marriages, such as marriage across tribes, mesas, or clans of disparate social status. The resultant labelling of parents as deviant was considered to contribute to the stigmatization experienced by the children, thereby contributing to the initiation of a series of stressors. Finally, although current statistics reveal that Whites have higher rates of suicide than African Americans, this difference may diminish as a function of rapid increases in rates of suicide among African American men in recent decades (Centers for Disease Control and Prevention, 1990). Within African American populations, geographic influences on suicide have been found. Suicide rates are highest in the urbanized and industrial Northeast and Midwest and lowest in the rural South, again suggesting that the absence of more traditional, culturally sanctioned supports may be operative (Shaffer & Fisher, 1981).

**Summary, Intervention, and Social Policy Implications**

The developmental psychopathology perspective proffers important insights useful for efforts to prevent depressotypic organization from evolving into depressive disorders as well as for intervention once depression has occurred (Kellam, 1990; Kellam & Rebok, 1992). Understanding the organization of psychological and biological developmental domains among the offspring of depressed parents and among depressed children and adolescents is invaluable for conceptualizing the meaning of symptom expression and the capacities of different depressed persons to benefit from different types of treatment (Shirk & Russell, 1996).

Given that the roots of depressotypic developmental organizations may originate in infancy, prevention efforts that focus on early intervention in high-risk conditions are likely to be important for promoting competent early developmental attainments on the sequence of stage-salient developmental issues (Cicchetti & Toth, 1992). Although parental depression as a risk condition has been a focus in this article, numerous disadvantageous family and societal circumstances (e.g., parental psychopathology, parental substance abuse, marital violence, child maltreatment, persistent poverty) constitute situations that may detract from children's ability to competently resolve developmental challenges, thereby contributing to risk for a depressotypic organization and depressive outcomes. Broad-based community programs, such as Head Start (Zigler & Valentine, 1979) or the Perry Preschool Project (Schweinhart, Barnes, Weikart, Barnett, & Epstein, 1993), that promote child competence and support adaptive family relationships are likely to be important in preventing developmental failures associated with depressotypic organizations, thereby reducing the prevalence of depressive disorders on a population level. Additionally, the preventive program of Beardslee and colleagues (Beardslee et al., 1997), in which efforts to educate families about the effects of parental depression have been found to result in improved illness-related communication between parents and children and in improved child understanding of parental illness, appears to be very promising.

Because of the potential for increased genetic as well as psychological risk with which the offspring of depressed parents are faced, preventive interventions for such families may be particularly important. Such prevention strategies, beginning when children are very young, should incorporate multiple foci and strategies, including attending to the alleviation of the parental depression, enhancing parent-child adaptive communication and interaction, and reducing larger family stresses, such as marital discord. In so doing, the likelihood of promoting competence as these children confront the universal challenges of development will be enhanced, and depressive outcomes may be prevented (see, e.g., Beardslee et al., 1997). The need for and provision of preventive services for offspring of depressed parents will likely require changes in social and health-care policy. All too often services are designated restrictively for the individual with the disorder, while the larger needs of family systems, offspring, and the functioning of depressed adults as parents are neglected (Beardslee et al., 1997).

When providing therapy for early-onset depression, intervenors must always be mindful of how the varying capacities of children at different developmental levels influence their capacity to utilize various therapeutic strategies (Shirk, 1988; Toth & Cicchetti, in press). Moreover, children continue to develop in an ongoing matrix of potentiating and compensatory factors that influence the course of their adaptation (Cicchetti & Aber, 1986; Cicchetti & Toth, 1998). Interventions to alter parental, family, and social-contextual sources of risk are necessary to alleviate ongoing contributors to the difficulty depressed children have in resolving stage-salient issues of development. Because depressed children and offspring of depressed parents are likely to have experienced maladaptive resolutions of earlier stage-salient issues (e.g., homeostatic and physiological regulation, affect differentiation and the modulation of attention and arousal, the development of a secure attachment organization, and self-awareness and self—other differentiation) prior to the onset of a depressive disorder, attention to reorganizing these critical domains through therapeutic interventions is crucial (see Gillham, Reivich, Jaycox, & Seligman, 1995; Stark et al., 1996). Moreover, helping
children to attain adaptive functioning in current stage-salient issues (e.g., peer relations, school achievement) is likely to be beneficial in beginning to reorganize and rework prior developmental incompetencies.

Despite the fact that depression is a preventable and treatable illness, considerable social stigma continues to be associated with seeking treatment for a depressive disorder, and large numbers of individuals who are clinically depressed remain untreated. Increasing the awareness of the availability and utility of treatments for depressive disorders in children and adolescents and reducing the negative public attitudes that interfere with seeking treatment require social and health policies that educate the public regarding depressive disorders, their effects, and the importance of intervention as well as policies that underscore the criticality of increasing the use of treatment. Only then will we triumph over combating the significant personal, family, and societal toll engendered by depressive illness.

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