INTRODUCTION

“Pain or suffering of any kind, if long continued, causes depression and lessens the power of action; yet it is well adapted to make a creature guard itself against any great or sudden evil”.

The Life and Letters of Charles Darwin, Charles Darwin, 1887

The World Health Organization, in collaboration with the World Bank and Harvard University, mounted an ambitious research effort in the mid-1990s to determine the “burden of disability associated with the whole range of diseases and health conditions suffered by people throughout the world. Possibly the most striking finding of the landmark Global Burden of Disease study is the impact of mental illness on overall health and productivity in the United States and throughout the world is profoundly under recognized. Mental disorders are the second leading cause of disability and premature mortality and collectively account for more than 15 percent of the overall burden of disease from all causes and slightly more than the burden associated with all forms of cancer.

Mental health refers to the successful performance of mental function, resulting in productive activities, fulfilling relationships with other people, and the ability to adapt to change and to cope with adversity. Mental health is indispensable to personal well being, family and interpersonal relationships, and contribution to community or society. Mental illness refers collectively to all diagnosable mental disorders. Mental disorders are health conditions that are characterized by alterations in thinking, mood, or behavior (or some combination thereof) associated with distress and/or impaired functioning. Alterations in thinking, mood, or behavior spawn a host of problems- patient distress, impaired functioning, or heightened risk of death, pain, disability, or loss of freedom (DSM-IV, 1994).

David Satcher (2000) issued the first Surgeon General’s report ever issued on the topic of mental health and mental illness. The science-based report
conveyed two essential messages i.e., mental health is fundamental to health and mental disorders are real health conditions that have an immense impact on individuals and families throughout the nation and the world. Appreciation of the clinically and economically devastating nature of mental disorders is part of a quiet scientific revolution that not only has documented the extent of the problem, but in recent years has generated many real solutions. The single, explicit recommendation of the report is to seek help if you have a mental health problem or think you have symptoms of a mental disorder.

THE SOCIOECONOMIC BURDEN OF DEPRESSION

Disability-adjusted life years (DALY) was calculated using a sophisticated method for assessing the duration and severity of a disability, stated that depression will become a leading cause of disability in the coming decades, second only to ischemic heart disease by the year 2020 (Holsboer, 2001). The World Health Organization Global Burden of Disease Study stated that depression is the single most burdensome disease in the world in terms of total disability-adjusted life years among people in the middle years of life (Murray and Lopez, 1996). A substantial proportion of affective disorders follow a chronic and recurrent course causing immense personal distress and suffering to individuals and their families. Affective disorders also contribute to premature mortality through suicide, adding to the community burden due to these conditions (Bhugra et al., 2005).

The word ‘depression’ in everyday parlance covers a wide range of emotional states that range in severity from transient moods of sadness to major psychotic episodes accompanied by increased risk of suicide. Depression in the form of a brief sad mood is a universal experience and is a normal part of living that accompanies the losses, frustrations, failures, and disappointments that all of us face. Clinical depression in contrast, is a syndrome, or constellation of co-occurring psychiatric symptoms that affects about 20 percent of the population. Major Depressive Disorder, the psychiatric label for clinically significant depression, is characterized by at least a two-week period of persistent sad mood or a loss of interest or pleasure in daily activities, and four or more additional symptoms, such as marked changes in weight.
or appetite, sleep disturbance, psychomotor retardation, fatigue, feelings of guilt or 
worthlessness, and concentration difficulties (Gotlib & Rottenberg, 2001).

Although depression has been classified and operationalized, it is worth noting
that it is not a universal condition. Obeyesekere (1985) urges that the quality and
intensity of symptoms are relevant to understanding the experiences of depression.
Depression is the principal or secondary reason for around 25% - 30% of visits to the
general practitioner. Mood disorders cause significant hidden burden as they often go
undetected and are inadequately treated. Major recent analyses by the World Health
Organization (WHO) and the Harvard School of Public Health, in light of the
seminal World Bank’s burden of disease analysis, showed that depression accounted
for 17% of the disease burden due to neuropsychiatric disorders (Desjarlais et al.,
1995). Depression ranks fifth in disease burden among women and seventh among
men, and it is expected to occupy second place in terms of relative disease burden by
2020 (Bhugra et al., 2005).

EPIDEMIOLOGY OF DEPRESSION IN DEVELOPED & DEVELOPING
COUNTRIES

A national survey conducted in the United States of America has shown that
the average 12-month prevalence in the general population is 10.3% with a 1-month
prevalence ranging between 1.5% and 2.5% amounting to a lifetime prevalence of
17% (Kessler et al., 1994). In an interesting secondary analysis of the data set from
two studies, Maercker (2001) reported that when levels of individualism were high in
the society (as indicated by autonomy and egalitarianism), these were correlated with
high levels of psychiatric diagnosis and symptoms. Particular cultural value patterns
were, thus, linked with more specific psychological complaints or disorders.
Maercker (2001) hypothesized that although the overall rate of psychological
morbidity is constant, cultural factors may determine differential rates of morbidity.
Although the two studies were conducted at different periods of time, this approach
deserves to be studied further. Unlike Simon et al. (2002), Maercker (2001) showed
that sleeping disorders and fatigue were more sensitively related to cultural values.
In a focus group of Punjabi women (Hindu, Muslim and Sikh) in the UK, Bhugra et al. (1997a) found that although the word depression was not mentioned by the participants, they were fully aware of the symptoms such as loss of appetite, loss of sleep, poor concentration, sadness, bouts of crying, feeling of low self-esteem, forgetfulness, ruminative intrusive thoughts and physical weakness. These subjects were able to identify life events and other stressors as possible precipitants in the genesis of depression. Family conflict, domestic violence and alcohol abuse were reported as common stress-inducing factors. Thus, it is worth emphasizing that family dynamics may well play a significant role in cultures within the Indian subcontinent. It can be argued that in cultures where collectivist values are held, the rates of depression may well be low.

Depression and social change have been shown to be linked (Jenkins et al., 1991). Among vulnerable individuals both macro- and micro-level changes can produce depression. Bhugra and Mastrogianni (2004) hypothesize that globalization will change the way patients identify, name and seek treatment for depression. Inadequate living conditions (poor sanitation, pollution, inadequate or contaminated water supplies and poor-quality housing), high rates of unemployment and breakdown of traditional family support systems are the more likely daily realities in cities of the developing world. It is therefore not surprising that the urban poor, especially urban poor women, in developing countries form a vulnerable group with a high risk for depression, a finding confirmed by research. Blue et al. (1995) found that rates of depression were elevated among residents of slum areas in Mumbai with 17.6% of women suffering from mental illness. The two slum settlements furthest from the health care facilities showed an elevated rate of 25% - 28%. Social class may be proxy variable – the social stresses associated with low social class are likely to provide a better explanation of this difference.

The available data from South Asia, especially WHO-sponsored studies in the region, point to a similar incidence and prevalence of affective disorders as in western populations i.e., median prevalence of 3.1% point, 6.5% within 6 months to 1 year and 16.1% lifetime prevalence of major depression (Wittchen et al., 1996). The notion that individuals in the developing world are somehow protected from affective
disorders, either due to cultural or biological influences, is highly unlikely (Bhugra et al., 2005).

One of the most consistent and intriguing findings of mental health research is that women are especially at risk for depression with double the rates as compared to men. This finding has been replicated across the world in societies at different stages of economic development. The gender difference first appears in adolescence, increases during the menopause, with older men and women reporting rates of depression. Although females have been shown to be more likely than males to suffer from depression, the variation in gender rates reported from several studies across cultures does exist. In addition to biological and genetic factors such as unemployment, poverty, migration (rural to urban as well as across nation states), racism, urbanization and globalization may all contribute. There is no doubt that all these stresses and other vulnerability factors will influence the self-esteem of individuals. It is worth restating that an awareness of individuals’ cognitive schema is also essential in understanding the genesis and perpetuation of symptoms of depression.

HISTORICAL PERSPECTIVE ON DEPRESSION

"O the mind, mind has mountains;
cliffs of fall Frightful, sheer, no-man-fathomed.
Hold them cheap May who ne’er hung there"

GERARD MANLEY HOPKINS

When Hopkins was appointed Professor of Classics in University College, Dublin, he entered the darkest years of his life; in those years, from 1885 to 1889, he wrote the “terrible sonnets”, a record of that experience we call depression. It was Hippocrates in 4th Century BC who offered a generic description of melancholia as ‘a state of aversion to food, despondency and sleep disturbance’ and thought that it was due to the accumulation of black bile. Artetaeus in 2nd century AD outlined features of mania: ‘the person laughs, plays, dances day and night and behaves as if he is an expert’. Robert Burton in 1621 provided an exhaustive account of depression in his magnum opus’ Anatomy of Melancholy’. Theopile Bonet in 1686 coined the term,
“manico-melancolicus”, to denote the connection between mania and melancholia (as quoted by Eapen et al., 2005).

In the 1830’s, two French physicians, Farlet and Baillarger became the first to identify and distinguish the symptoms of the disease which they called “circular insanity”. Remarkably, the signs and symptoms Farlet described are almost similar to those described today (Eapen et al., 2005). Kraeplin began the flood when he called this experience a disease. In 1913, he further developed this construct and distinguished the condition from other chronic progressive psychiatric disorders like schizophrenia. He used the nineteenth-century model of disease, which was based on the new findings of pathology and bacteriology. He saw disease as something new that had occurred in the life of the patient, although some people, he thought, had a constitution, which provided a fertile soil for this disease. Freud, while allowing the possibility of a constitutional basis, saw melancholia as a ‘reaction to loss’. The extensive psychoanalytic literature on depression (Mendelson, 1974) elaborated this concept of loss and drew attention to the presence of aggressive feelings intertwined with the melancholy. Karl Jaspers and Henri Ey made learned studies of the symptoms of depression. Adolf Meyer stressed the importance of environmental and cultural influences in depression. Aubrey Lewis and Russell Davis described depression as an adaptation of an intolerable situation (Rowe, 1978).

Once the existence of depression as a disease was accepted by the medical profession, the debate then began as to how many diseases of depression there were and what they should be called. Leonhard and his colleagues in 1954, differentiated between illnesses characterized by recurrent episodes of depression, which they termed unipolar from bipolar disorders in which they alternated with periods of mania. Words proliferated – endogenous and reactive depression, manic depression, psychotic and neurotic depression, retarded and agitated depression, involutional melancholia, affective psychosis, masked depression, senile depression, metabolic depression, unipolar and bipolar depression.

Carney and Sheffield commented that “in the absence of a generally agreed physical basis for depression, responsiveness to treatment is probably the most useful
independent criterion of depressive classification". The treatment which gives this post hoc diagnosis (that is, if a patient says that he is no longer depressed after this treatment, then he had had an endogenous depression) is electroconvulsive therapy (ECT), which was accidentally discovered by Cerletti in 1938 and which maintains its popularity despite its undoubted damaging effects on memory (as quoted by Rowe, 1978).

PHENOMENON OF DEPRESSION / DISTINGUISHING DEPRESSIVE AFFECT FROM ILLNESS

Depressive affect denotes a characteristic set of feelings or a disposition to feelings that are aroused in a person confronted with psychic loss, disappointment, or failure (Isenberg & Schatzberg, 1978). Although this constellation of feelings is broad, feelings of helplessness and hopelessness are pivotal. When most writers refer to depressive affect, they also include the feelings of being let down, sadness, psychological pain resulting from a threat to one’s sense of well-being, and the fear of failure of not being able to live up to one’s own expectations. These depressive affects can be mobilized in people who are relatively healthy as well as those who are predisposed to severe depressive illnesses. Both Zetzel (1966) and Hartmann (1952) stressed that the ability to develop conscious feelings of sadness and to acknowledge one’s own and others’ limitations are a pre-requisite for mature emotional functioning. The capacity to accept and tolerate sadness might be an indication of sufficient ego strength to be incompatible with a depressive illness. Zetzel emphasized that those people who cannot tolerate depressive affect show a special kind of vulnerability and hence a predisposition to psychopathology, not necessarily depression.

Depressive moods, Jacobson (1971) emphasized, were persistent states of depressive feelings, which colored both, one’s perceptions and one’s ability to respond adequately to experiences. Although this mood may influence most of one’s experiences for days or weeks, it does not necessarily become associated with a complex of symptoms which has been characteristic of the various depressive disorders.
The depressive syndrome and/or disorders, on one hand, are marked by a host of somatic and psychological symptoms which vary as to their intensity and type. The patient may be deeply depressed, apathetic, retarded or agitated, anorexic, anhedonic, and sleep poorly. If clearly psychotic, he may experience hallucinations and express delusions of guilt, inferiority, nihilism, and abject poverty. At a less psychotic level, thinking is characterized primarily by retardation and subjective difficulty without frank delusions. There may be preoccupation with the bodily processes, which are often slowed down; and guilt and self-depreciation are common, again without delusional conviction. The future looks hopeless, and life seems hardly worth living. At the mildest level of depression, he may simply have a sense that life is not interesting, and experience some social withdrawal and a lack of pleasure. Vegetative signs may vary greatly. The patient may not be aware of the depressive component to his everyday functioning and somber mood.

Initially, the emphasis was on primary depressions by some groups (Feighner, Robins, Guze et al., 1972) and unipolar endogenous depression in others (Schildkraut & Klein, 1975). Rubinfine emphasized that personality variables may predispose certain individuals to becoming depressed, implying a stepwise relationship between premorbid personality structure and depression, and he felt that evidence suggests a primarily oral personality disturbance.

Ever since Freud postulated the central importance of “the loss of self-regarding feeling”, psychoanalytic investigators, including Bibring, Jacobson, Sandler, and others, have repeatedly emphasized the crucial role of the fall of self-esteem in depressive disorders. Bibring felt that this was secondary to a shock like awareness of the patient’s helplessness, whereas Jacobson believed that self-esteem could fall in a variety of ways, at times due to an as yet undefined psychosomatic factor. The onset of the disorder in these views is preceded by the subject’s becoming aware of the unlikelihood of obtaining or maintaining an ideal state of well-being. Joffe and Sandler (1966, 1968) stated that such an awareness of helplessness can be so painful and threatening that the subject fears an exhaustion of all available psychological resources in the near future and loses a sense of self-confidence. The seriously depressed subject experiences his actual state as so far removed from his
ideal state of well-being that his survival is at stake. When he feels he has no way of coping with or changing the situation, further activity seems to offer little hope of bettering his plight. Since further loss is expected, any effort seems to be the wisest choice. This sequence results in a progressive inhibition of ego functions as exemplified in psychomotor retardation and an inability to care for one-self (wash, eat, etc).

**CLINICAL FEATURES**

Clinical presentation of depression varies both, in terms of patterns in which they occur as well as in term of intensity. Three major kinds of manifestations occur (Eapen et al., 2005):

❖ Some are exaggerations of normal experience like sadness. They become significant only when they are severe and out of proportion to the context. A depressed mood can be distinguished from normal feelings of sadness by its greater intensity, duration and pervasiveness. Sometimes the person is so depressed that he cannot express his feelings and facial appearances can provide clues about the state. Quite often the reluctance to talk is because of cultural beliefs and perceived stigma about disclosure.

❖ The second set of phenomena involves impairments of normal functioning, like the inability to experience pleasure (anhedonia), loss of energy and fatiguability and tiredness (anergia), retardation, inefficiency in thinking, impaired attention, loss of interest and bodily symptoms.

❖ The last group of phenomena involves the impact of the emotional state on other aspects of psychological functioning, like thinking, perception, etc. These include depressive cognitions, hypochondriacal beliefs, delusions, perceptual changes and obsessive-compulsive phenomena.

**IS DEPRESSION AN ADAPTATION, AN ADAPTATION GONE AWRY, OR A PATHOLOGICAL STATE UNRELATED TO ANY FUNCTION?**

Low mood and depression have been difficult to distinguish from each other and from related states such as sadness, grief, demoralization, guilt, and boredom.
Sadness, depression, guilt, and grief may be partially differentiated members of a "fuzzy set" (Watson & Clark, 1992; Shaver et al., 1987), whose commonalities are explained partially by phylogeny and partially by the benefits certain responses offer in any unpropitious situation. Many people believe that low mood is normal and probably useful in certain situations, but when excessive, prolonged, or expressed in the wrong situation, it becomes pathological depression.

Depression is painful and interferes with normal function, so it seems obviously maladaptive. However, other useful capacities, such as pain, nausea, and fatigue, are also aversive and disruptive. In fact, their very aversiveness is likely to be a product of natural selection, probably because they promote escape and avoidance of situations that decrease fitness. From this perspective, the intrinsic aversiveness of most low mood and depression suggests that they may be related to a defense (Nesse, 2000). The regularity of the relationship between loss and negative affect and the proportionality of low mood to the magnitude of a loss (Monroe & Simons, 1991; Brown & Harris, 1978) imply that mood is regulated.

In fact, major depression disorder affects about 10% of the US population in a year (Kessler et al., 1993). Depressive syndromes that do not meet diagnostic criteria are even more common (Judd et al., 1997; Olfson et al., 1996). Furthermore, there is no point of rarity in the distribution that can differentiate pathologic from nonpathologic depression (Kendler & Gardner, 1998), and subjects move frequently between syndromal and subthreshold depression over time (Akiskal et al., 1997; Angst et al., 1997; Whybrow et al., 1984). Also, the incidence of depression is highest at the ages where reproductive value peaks, a pattern characteristic of few diseases.

Opinions range from the dismissal of the possibility that depression or low mood could be useful to the conviction that even severe depression is an adaptation with a specific function (McGuire et al., 1997). The fact that low mood, and perhaps some depression, may be useful should not distract attention from recognition that depression is one of humanity's most serious medical problems (Murray & Lopez, 1996).
Depression has also been interpreted as a communication designed to manipulate others into providing resources (Hagen, in press; Watson & Andrews, 1998). Engel and Schmale (1972, 1975) used the phrase *conservation-withdrawal* to refer to the presumed utility of the “despair” phase of the behavior pattern of a lost infant monkey. Depression in adults after a loss has been interpreted as analogous to this pattern of infant behavior, and many authors (Thierry et al., 1984), including Beck (1996), emphasize the role for depression in conserving resources. Bowlby (1969) emphasized the value of attachment and of aversive feelings when an attachment is threatened or lost. He and his followers have generally treated grief and depression in adults as an epiphenomenon of attachment (Archer, 1999). Price and others (1967, 1994) have suggested the utility of depression as a yielding signal in hierarchy conflicts. This theory suggests a possible role for high as well as low mood in negotiating hierarchies (Gardner, 1982) and is supported by clinical evidence of episodes of depression precipitated by social competition and resolved by reconciliation (Brown et al., 1995; Gilbert, 1992). Hartung (1988) has suggested a variation in which self-deception about one’s abilities (“deceiving down”) lulls superiors into thinking one is no threat. Clinicians have long noted that depression is common in people who are pursuing unreachable goals (Davis, 1970; Bibring, 1953). “Failure to yield” may be example, in the status competition domain, of this more general situation. The helplessness literature takes a related view but emphasizes the frequency and costs of incorrect perceptions of lack of control instead of the benefits of a passive strategy (Peterson et al., 1993; Seligman, 1975; Wortman & Brehm, 1975). Klinger (1977, p.21) concluded “depression is ordinarily, therefore, a normal, adaptive part of disengaging oneself from an incentive”. May other researchers also see a close relationship between depression and difficulty in disengaging from major commitments (Brickman, 1987; Janoff- Bulman & Brickman, 1982). Several related theories have led to evidence that low mood is aroused by a mismatch between achievements and expectations (Brunstein, 1993; Gilbert, 1992; Schwartz, 1990; Higgins, 1987). Closely related work on the information functions of mood (Schwartz, 1990). Low mood shifts cognition to the more systematic (Alloy & Abramson, 1988) and perhaps the more realistic (Martin & Tesser, 1996; Taylor & Brown, 1988).
The characteristics that pose the core conundrum of depression are pervasive pessimism, low self-esteem and reduced initiative. When depression is instead seen as a state shaped to cope with unpropitious situations, it is clear how it could be useful, both to decrease investment in the current unsatisfactory life enterprise and also to prevent the premature pursuit of alternatives. Failure to disengage can cause depression, and depression can make it harder to disengage. This may explain why the low-mood system is so prone to getting stuck in positive feedback loops (Nolen-Hoeksema & Morrow, 1993; Pyszczynski & Greenberg, 1987; Teasdale, 1983; Beck, 1967). Also, the costs of low mood may be small compared with those of inappropriate high mood, so in certain situations the “smoke detector principle” biases the system toward low mood (Williams & Nesse, 1991).

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**Figure 1.1**: A schema of four stages in the development of a depressive disorder. Each shift of one stage to the right represents a regressive step or a deepening into the depressive or nodal point along this continuum. The arrows to the left indicate the operation of several coping mechanisms or defenses which have a thrust in the direction indicated. Introjection appears to be a coping mechanism which confounds the patient's equilibrium, because introjection appears to contribute to some of the most serious depressive symptomatology rather than to protect the patient, even temporarily, as does flight, avoidance, denial, and repression (Source: Isenberg & Schatzberg, 1978).
DEPRESSION IN ADOLESCENCE – AN INTERNALIZING DISORDER

Depression is a disorder and disturbance of mood that has been described from ancient times with continued efforts to clarify the nature of this aspect of the human condition. Although we no longer ascribe the etiology of depression, or melancholia, to the function of bodily humors such as black bile or "perturbations of the soul" (Jackson, 1986), there are many commonalities between ancient and contemporary descriptions of the phenomenology of depression.

In the conceptualization of psychopathology in children and adolescents, a predominant theme over the past two decades has been the differentiation of disorders as a function of their general phenotypic expression. This has led to the categorization of disorders as primarily internalizing or externalizing, based on their general symptom manifestation (Reynolds, 1992a). Internalizing disorders are those that tend to be composed of relative covert symptoms, many of which are cognitive, internal to the youngster, or relatively difficult to observe. Externalizing disorders are overt, highly observable either directly or indirectly (such as in the outcome of certain antisocial behaviors), and typically expressed as behavioral excess. Depression and depressive (mood) disorders in children and adolescents may be viewed as internalizing disorders.

Depressive disorders represent serious forms of psychopathology of childhood and adolescence, given that some depressive episodes may lead to potentially life-threatening or negative outcomes. Previous notions of "adolescent turmoil" or the perspective of the adolescent who is "just going through a moody stage" are no longer viable conceptualizations (Offer & Schonert-Reichl, 1992). This is amply evident when one considers the large numbers of depressed and suicidal youth, a significant number of whom do not survive to adulthood or do so with significantly reduced psychosocial competence or functioning.

Internalizing disorders such as depression are often undetected due to the more covert nature of their symptoms. For instance, feelings of subjective misery, hopelessness, fatigue, anhedonia, difficulty sleeping and other symptoms of
depression may not be readily observable, particularly in youngsters who may be characterized as shy or somewhat withdrawn. Depression differs from other behavior disorders of childhood and adolescence in its comorbidity with suicidal risk.

It can be estimated that depression affects almost two million children and adolescents in USA and Canada. As an internalizing disorder in young people, it is a source of intense misery and distress to those affected. Because of its internalizing nature, we may view depression as an insidious psychological disturbance in young people. Thus, for some youngsters depression may be inconspicuous yet have deleterious effects on the child (Reynolds, 1998).

Wamboldt (1994) examined the nature of medical and psychiatric disorders reported in child and adolescent patients seen at nine medical-psychiatric settings that are considered Type III or IV (moderate to severe medical and psychiatric problem treatment) child and adolescent medical-psychiatric units in USA. With regard to psychiatric disorders, he found a greater prevalence of internalizing disorders than externalizing disorders, with 32% of the patients demonstrating mood disorders, primarily depression.

Major depressive disorder is a common disorder for adolescents. It has a wide array of symptoms affecting somatic, cognitive, affective, and social processes. Academic failure, poor peer relationships, behavioral problems, conflicts with parents and other authority figures, and substance abuse are some of the consequences of major depressive disorder in this age group (Hauenstein, 2003). Depression in children and adolescents, within the scope of current nomenclature and systems of classification, is considered to be a mood disorder similar in many respects to this disorder in adults. From another perspective, depression in children and adolescents may be considered as significant perturbation of mood. A depressed youngster may experience a range of symptoms, some of which may be overt, such as irritability or distinctly sad appearance, and others may be covert, as illustrated by feelings of low self-worth, hopelessness, suicidal thoughts, and guilt. Depression in children and adolescents can cause significant impairment in daily functioning and personal and
social involvement (Puig-Antich et al., 1985). In young children, affective state may be characterized as being moody or distant from others and slow to respond to social interactions with peers and family. Although this may characterize many adolescents, the depth and quality of these characteristics in depressed adolescents, and in particular, their lack of positive response to previously reinforcing events or activities, generally distinguishes depression in adolescents from more normative trials and tribulations associated with the normative course of adolescence.

Epidemiological data suggests that suicidal behaviors are a serious problem among children and adolescents. Although some youngsters who demonstrate suicidal thoughts and behaviors do not manifest clinical levels of depression, the majority of suicidal youngsters are depressed, with many who view suicide as a way of ending their intense psychological distress (Reynolds & Mazza, 1994). Depression can take a variety of courses and chronicity, with relapse and recurrence relatively common over the lifespan (Kovacs, 1996a; Keller, 1994). Kovacs and colleagues, in a noteworthy longitudinal study of depression in children, found that for some youngsters, particularly those with a dysthymic disorder, their depression may last for many years, and in some children represents a precursor or risk factor for other psychiatric disorders (Kovacs, 1996b).

CONTEMPORARY INTEREST IN CHILDHOOD & ADOLESCENT DEPRESSION: HISTORICAL PERSPECTIVE

The existence of major depressive disorder in puberty has been a controversial issue. Though the literature on the subject goes back to the 1960s, no child psychiatry textbook mentioned the existence of pre-adolescent depression until Rutter and Hersov's in 1977 (Hersov, 1977). The study of depression can be dated back to the time of Hippocrates, but the burgeoning of interest in the study of childhood depression is of relatively recent origin. To appreciate the infancy of this area, one needs only to look at a classic encyclopedic text, Child Psychiatry (Kanner, 1957; 770-777) and fail to find the term "childhood depression" listed in the index. The past
15–20 years has evidenced a growing body of knowledge marked by shifts in theoretical orientation.

In the 1950s through 1970s, there were two prevailing viewpoints on depression (or manic depression) and its relative absence in prepubescent children. Although pediatricians had long recognized that institutionalized infants fared poorly, both physically and emotionally, Rene Spitz brought the deplorable situation to the forefront by describing the “vitiated condition of the body due to the long confinement in a hospital” as hospitalism. Subsequently, but along the same continuum of emotional and stimulatory neglect, Spitz and Wolf articulated the concept of anaclitic depression. That condition occurred when an infant, usually between the ages of 6 and 11 months, was separated from a primary caregiver with whom the child had formed an emotional bond. If the infant did not see the caregiver very often and if there was no substitute caregiver, an emotional response began after several weeks that the authors thought resembled adult depression. It began with dejected, sad, or apprehensive facial expressions and progressed through disappointment when the observer withdrew, to crying and screaming, to apathy, reduced babbling, and reduced physical activity, to complete withdrawal, dejection, and detachment from the environment. The response was accompanied by greater susceptibility to intercurrent illness and a decline in the developmental quotient. Upto a certain point the condition was reversible or partially reversible. Spitz found if it continued longer than five months, the condition of hospitalism along with accompanying poor appetite, weight loss, had a much worse prognosis.

An early extreme view was also that the psychosexual development of young children precluded the development of depressive disorder (Rie, 1966). The classic psychoanalytic view held that childhood depression could not exist because the necessary psychic structures, i.e., the superego, were not yet sufficiently developed (Rochlin, 1959). The initial challenge to the psychoanalytic position came from those positing that depression in children was masked or expressed in depressive equivalents. Proponents of this view believe that depressive symptomatology as seen clinically in the adult population is rarely seen in children. The view holds that
depressive symptoms are not directly expressed by children but must be inferred from other behaviors that mask the underlying depression i.e., the manifestation of these feelings is indirect (Glaser, 1968; Toolan, 1962). This commonly held theory gave rise to the concepts of ‘masked’ depression, and depressive ‘equivalents’. Some of the behaviors which have been identified as masking the underlying depression or that are depressive equivalents are hyperactivity, aggressiveness, temper tantrums (Toolan, 1962), somatic complaints such as headaches, stomachaches, enuresis, encopresis (Cytryn and McKnew, 1974; Sperling, 1973) and school problems such as poor academic performance, truancy, and school phobia (Glaser, 1968) and under-achievement (Kovacs & Beck, 1977). The underlying depression is used to account for the above behaviors even in the absence of dysphoric mood.

This perspective has not been widely accepted and numerous criticisms have attacked its logic. One of the major criticisms is that this view has no clinical value because the behaviors identified as masking depression cover the range of childhood psychopathologies (Kaslow and Rehm, 1983; Carlson and Cantwell, 1980) and thus, no basis is provided for a differential diagnosis. For example, it is clear if the hyperactive child is masking depression or is simply hyperactive. The general theory was that these disparate behavior disorders emerged because of the child’s defensive work against the underlying depression. In contrast, Conners (1976) argued that the immature cognitive and emotional development of the child was responsible for the adult-child differences in the behavioral manifestations of the same depressive disorder. Some exceptions to these perspectives were postulated (e.g., Sandler & Jaffe, 1965) although for a number of reasons, did not significantly influence the existing status of depression in childhood (Bemporad, 1994).

Early research and clinical interest in depression in children and adolescents also focused on bipolar disorder. The extreme nature of symptoms in manic depressive illness and mania, and the similarity between symptoms of these disorders in children and adolescents to those found in adults, led to a number of clinical reports (e.g., Campbell, 1952; Beres & Alpert, 1940). Carlson (1994) notes that Emil Kraeplin in 1921 was one of the first to describe manic depressive illness in a 6-year-
old child in the 1952 volume of *Nervous Child*. Rather, the designation referred to the presence of severe, probably endogenous, probably recurrent depressions. In 1960, E.J. Anthony and P. Scott described manic depression in ways that are now synonymous with bipolar disorder and further clarified the importance of distinguishing between prepubertal and postpubertal youth in studies of depression. However, research reports on manic depressive illness in children and adolescents are scarce before the 1970s.

In contrast to the above view concerning the lack of a clear depressive syndrome in childhood, Frommer (1967) and Crtryn & McKnew (1972) described childhood depressive disorders, and proposed their sub-classification. Frommer (1967) divided childhood depression into three groups: enuretic depressives, phobic depressives, and pure depressives or mood disorders. He stated explicitly that they 'rarely showed a frank depressive picture or its typical mood disorder, and sometimes even denied any feeling of depression'. The 'mood disorder' group, were described as weepy, irritable, with temper outbursts and sleep disturbances, some with quite serious antisocial behavior. Furthermore, from the clinical descriptions, the clinician was not on firm ground regarding the distinctions among the three depressive subtypes.

In the 1970s, a small but significant number of clinical and research reports appeared that suggested the importance of studying depression in young people. An important article that shed light on the existence of depression in childhood was published in 1970 by Pozanaski and Zrull who described clinical cases gleaned from the outpatient files of the Children's Psychiatric Hospital at the University of Michigan Medical Center from the years 1964 to 1968. Based upon an examination of records of 1788 children, 98 cases were identified that showed significant evidence of depression, with 14 cases providing sufficient file data for an in-depth examination and reporting among children ranged in age from 3 to 12 years.

The development of operationalized criteria and structured interviews for depression and mania in adults in the 1970s was accompanied by a similar effort in
children. **Weinberg and his colleagues** in St. Louis in 1973 are frequently associated with the development of the first contemporary diagnostic criteria for depression in children (Weinberg, Rutman, Sullivan, Penick, & Dietz, 1973; Ling, Oftedal, & Weinberg, 1970). The Weinberg criteria were an outcome of the observation of depressive symptoms in children that were phenomenologically similar to symptoms of the disorder as specified for adults. An examination of the symptoms and criteria delineated by **Weinberg et al. (1973)** shows a great deal of communality with contemporary diagnostic formulations. In the late 1970s, **Petti (1978)** developed the Bellevue Index of Depression (BID), a semi-structured clinical interview of depression designed for use with children based on Weinberg symptomatology. Structured interviews for psychiatric disorders in children and adolescents were developed shortly thereafter in the hope of better defining disorders and eliminating some of the methodological problems that hampered identifying children with mood disorders. Weinberg’s criteria were probably too unspecific as the diagnosis could be made in the absence of a majority of the symptoms of the classical depressive syndrome. Further, they did not indicate how the clinical judgements were arrived at; whether by interview with parents, children, or both, or what was the reliability of the assessment.

**Lefkowitz and Burton (1978)** argued that depressive features in children reflect a developmental phenomenon rather than a clear clinical syndrome and these dissipate with time. In rebuttal, **Costello (1980)** argued that while single symptoms of depression may be prevalent and transient in childhood, the syndrome of depression, a cluster of highly correlated symptoms, may not be. For instance, a symptom of depression such as eating disturbance may be prevalent and may dissipate with time in six-year olds but the presence of an eating disturbance in conjunction with dysphoric mood, anergia, and low self-esteem may not be as transient. Nevertheless, serious challenges to this position have been arising from developmental psychopathologists (e.g., **Rutter, 1986; Cicchetti and Schneider-Rosen, 1984**) whose perspective focuses on the continuities and discontinuities between normality and pathology as well as across time, i.e., from one stage of development to the next.
Around this time period, a number of reports began to emerge as to the clinical use of adult antidepressant medications with children. Antidepressant medications were relatively new but, within 10 years of early reports with adults, Frommer (1967) described the use of antidepressants with children. Later researchers advanced the argument that the affective regulatory systems of children and adults might differ, since stimulant drugs did not induce mood elevation in prepubertal hyperactive children.

Based on reports (Weinberg et al., 1973; Ctryn & McKnew, 1972) and clinical observations, Puig-Antich et al. (1978) advanced the hypothesis that there were depressed children who displayed a disorder similar to the adult major depressive disorders identifiable by means of the Research Diagnostic Criteria (RDC) (Spitzer et al., 1978). They identified 13 prepubertal children who continued to meet RDC criteria for major depressive disorder after at least one month of psychosocial treatment alone. The family histories obtained from the parents conformed to the pattern described by Winokur as ‘unipolar spectrum disease’ (Winokur et al., 1973) established that the Research Diagnostic Criteria for major depressive disorder in adults were also appropriate for prepubertal children. Furthermore, given the apparent clinical similarity between child and adult major depressions with regard to clinical picture, family history, and clinical response to imipramine, Puig-Antich advanced the hypothesis that child and adult major depressive disorders represented the same illness.

Two other independent studies appeared at the same time. Pearce (1977) analyzed the clinical records of 547 child and adolescent psychiatric patients (ages 1 – 17) and divided them into two groups according to the reported presence or absence of dysphoric mood. There were three main overall findings: (1) symptoms of the depressive syndrome tended to cluster with symptoms of depressed mood, (2) about one-third of the children with depressed mood did not present a depressive syndrome, (3) in very young children, the depressive syndrome might be present without a clear-cut report of depressed mood. Kovacs & Beck (1977) using a self-report form (the Child Depression Inventory) similarly found that the items of the depressive
syndrome tended to cluster with depressed mood in a sample of 8–13 year old from a regular school. In a clinical study of 175 consecutive child and adolescent psychiatric patients, Kupferman & Stewart (1979) reported that 13 percent of girls and 5 percent of boys met adult criteria for depression. This consensus has been reflected in the official American Psychiatric association classification of mental disorders (DSM – III) where no distinction was made in the diagnostic criteria for prepubertal, adolescent and adult depression (Puig-Antich & Gittelman, 1982).

In 1980, the American Psychiatric Association (APA) published the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; APA 1980). Depressive (mood) disorders were listed in the adult disorders section, differential symptom criteria and duration specifications for major depression and dysthymic disorder, respectively were provided for these disorders in children and adolescents as compared to the disorders in adults. The publication of DSM-III, as well as research findings such as those by Christ et al. (1981), led to a focus on symptoms of depression in children and adolescents as similar in many regards to adults.

The acceptance of the largely adult specifications for depression in the DSM is not universal nor, as suggested by Nurcombe and colleagues (Nurcombe, 1994; Nurcombe et al., 1989), strongly supported by research evidence. Nurcombe (1994) examined the validity of major depression as a diagnosis in children and adolescents using as criteria: natural history, psychobiological markers, genetic studies, response to treatment, and construct validity. The last was based on multivariate analyses of various checklists and measures. As presented, much of the evidence is mixed, suggesting that depressive disorders in children may not be as currently defined by DSM, with more work needed for the presentation of diagnostic validity of the disorder.

Based on diagnostic and epidemiological surveys of children and adolescents, it is evident that depression is a significant problem among youngsters (Reynolds & Johnston, 1994). Epidemiological studies suggest that between 3% to 5% of children
and 7-10% of adolescents manifest clinical levels of depression (Poznanski & Mokros, 1994; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Fleming & Offord, 1990; Whitaker et al., 1990), with lifetime rates in young people significantly higher (Lewinsohn et al., 1993). These data, and the general undertreatment of depression in children and adolescents (Keller, Lavori, Beardslee, Wunder, & Ryan, 1991) point to the critical need for study, identification, and treatment of depression in young people.

GENESIS OF AFFECTIVE DISORDERS IN CHILDREN AND ADOLESCENTS

In so called “masked” depression or depressive equivalent, the depressive affect has supposedly reached an abnormal level and is then confronted by a series of defense and compensatory mechanisms – obsessional devices, manic reactions, somatic disturbances – that vary with the stage of development. In the earlier stages, there are dysfunctions of eating and sleeping (infantile disturbances of the sleep rhythm have been included by some among dispositional indices); in the middle years, learning disturbances and school phobias may be present; and in adolescence, acting out the delinquency may occur.

According to Anthony (1978), the affective disturbance can be best understood, in terms of imbalances in a number of basic regulatory mechanisms that include the following:

1. The regulation of self-esteem (between superiority and inferiority).
2. The regulation of hedonic capacity (between pleasure and un-pleasure).
3. The regulation of self-confidence (between omnipotence and helplessness).
4. The regulation of hopefulness (between optimism and pessimism).
5. The regulation of psychic input and output (between fullness and emptiness).
6. The overall regulation of affect (between elation and depression, between euphoria and dysphoria).
These regulations presuppose a biological core determined by hormonal and autonomic mechanisms surrounded by psychobiological factors associated with conservation-withdrawal, with overlayers of psychological responses of ego and self to more nuclear events.

INFANCY

Spitz (1946) and Bowlby (1960, 1980) described infant reactions to maternal deprivation or separation in terms similar to the adult symptomatology of depression. Spitz's term, *anaclitic depression*, referred to the weepiness, withdrawal, apathy, weight loss, sleep disturbance, and developmental decrements observed in six to twelve month old institutionalized children. Bowlby wrote of the observed sequence of protest – despair – detachment in toddlers, aged six months to four years, separated from maternal caretakers. Furthermore, similar conditions in infancy can be produced by lack of cognitive stimulation, malnutrition, or organic deficiency diseases. In addition, this reaction obviously lacks the other components of the syndrome such as self-blame, guilt, and decreased self-esteem (Bemporad and Wilson, 1978). These infants experience negative affective changes, which can be persistent and disabling. Even in non-clinically depressed mothers, it has been suggested that infants' negative affective reactions may be related to the mother's state during this period, as she exhibits more depressed affect, less animation, and exhausted behavior (Field, 1986).

Engel (1962) suggested that when the initial distress and crying of an infant fails to produce relief – that is, to produce the good satisfying mother – the child is faced with an overwhelming anxiety and a consequent exhaustion. He depicted depression-withdrawal as a defense mechanism involving regression to a stage of development prior to the formation of identification with specific environmental objects, especially the mother.

Schmale (1958) described this as the emotional states that occur in man when he feels that "it is the end." *Helplessness* is the term applied when the individual gives up because of a failure of external sources of emotional support. *Hopelessness* is a state that occurs when he feels personally responsible for the situation and believes
that nothing he or anyone else can do will alter it. He also has feelings of worthlessness, believing that he does not deserve assistance from others. Adult helplessness has its analogue in infancy, the initial reaction of intense anxiety felt by a baby who has been separated from its parents; and adult hopelessness has an analogue in infancy, the state of depression-withdrawal.

PRESCHOOL YEARS

Less was known about the syndrome of depression in this period as few systematic data is available as a result of researchers' questioning of depression as a true clinical entity. Clinical observations of depression in preschoolers have been provided by various investigators (Herzog and Rathbun, 1982; Philips, 1979; Ushakov and Girich, 1972; Poznanski and Zrull, 1970). Ossofsky (1974) described depressed preschoolers as manifesting mood disturbance (irritability, sadness, and excessive crying), vegetative disturbance (insomnia, decreased appetite) and behavior disturbance (hyperactivity, temper tantrums, absence of normal play). Ushakov and Girich (1972) reported that in their sample of 12 depressed preschoolers, no specific clinical picture was evident. Researchers have found that their grief reactions are milder and of shorter duration than in older children. In an attempt to explain the instability of symptoms in this developmental period, Bemporad and Wilson (1978) utilized Piagetian concepts of cognition in the preoperational stage. Kashani and Ray (1983) were among the first to attempt to systematically investigate the frequency of depressive symptoms and major depressive disorder among a nonclinical population of preschoolers and found that individual depressive symptoms such as sleep disturbance, appetite disturbance, and agitation or hyperactivity were found while anhedonia and feelings of worthlessness did not occur at all in this group. Furthermore, Kashani and Carlson (1987) noted a relatively high rate of somatic complaints which suggests that younger children may be more likely to express their discomfort with physical symptoms.
SCHOOL AGED YEARS

In an attempt to compare symptom patterns across development, Carlson and Kashani (1988) compared the frequency of depressive symptoms in three studies of four clinics referred age groups: preschoolers, prepubertal children, adolescents and adults. Some symptoms which increased with age included anhedonia, diurnal variation and psychomotor retardation. Those which decreased with age included depressed appearance and somatic complaints. Fatigue, agitation and anorexia were less frequent among prepubertal children and adolescents than they were in the preschool and adult groups. Symptoms such as depressed mood, diminished ability to concentrate, sleep disturbance, and suicidal ideation were consistent across the life span measured.

Three patterns of depression were identified in a group of six to thirteen year olds (McConville, Boag and Purohit, 1973). Symptoms in the six to eight year old group were characterized as the “affluent” type and included dysphoric mood and feelings of loneliness, helplessness and loss. The symptoms in the eight to ten year old group were described as the “self-esteem” type. These included hopelessness and negative self-esteem. The symptomatology in the ten to thirteen year old group was characterized as the “guilt” type.

It has been suggested that the shift from the affectual type to the self-esteem type is due to the ability of the older groups to verbalize their feelings and thoughts about themselves and is a function of their developing cognitive structures. They are also beginning to misattribute blame which in depressed children, might lead to unwarranted self-blame and guilt (Bemporad and Wilson, 1978; McConville et al., 1973).

In Garber’s study (1984) of seven and thirteen year old clinic referred females, there was an increase in frequency of depressive syndrome and of individual depressive symptoms. Those symptoms that were found to increase with age were appetite disturbances, hyperactivity, pervasive loss of interest, capacity to have fun,
guilt, hopelessness, irritability, fatigue, problems at school, low self-esteem, difficulty concentrating, and depressed feelings. Those symptoms which decreased with age (among the depressed children only) were crying and morbid ideation.

ADOLESCENCE

Unlike earlier points in development, depressive symptoms in adolescence are typically more similar to that in adulthood. Ushakov and Girich (1972) found that among 14 to 17 year old depressives, genuine melancholy, psychomotor retardation, difficulty concentrating, and a significant rate of suicidal ideation and attempts were observed. As compared to younger children, crying was less frequent. However, Friedman, Hurt, Clarkin and Corn (1983) found that adolescents experience less self-pity than adults.

In other studies comparing child and adolescent depressives, the frequency and severity of hypersomnia, hopelessness, anhedonia, weight change, and drug and alcohol use were greater among adolescents than among children (Mitchell et al., 1988; Ryan et al., 1987). Furthermore, depressed adolescents with coexisting disorders (e.g., anxiety and conduct problems) had higher mean depression severity scores than those adolescents who suffered from adolescents alone (Mitchel et al., 1988). This finding was more significant for adolescents than for children. Another difference found between child and adolescent depressives was the less frequent self-report of anxiety symptoms by adolescents.

Another significant feature differentiating childhood and adolescent depression is the shift in gender differences. Studies of school age children have found either a slightly greater incidence of depressed males than females or an even distribution in females than in males (Rutter, 1986). In Carlson and Kashani's (1988) comparison of depression across four age groups, suicidal ideation appeared to be consistent. However, suicidal attempts and successful suicides evidence different patterns across development, with adolescence standing out as a particularly vulnerable period. As reported by Rutter (1986), United States statistics show that
suicide prior to puberty is relatively rare with the rate rising rapidly during midteens and continuing to rise through adulthood. Attempted suicide peaks in the midteen years and decreases in adulthood. In contrast, in another study comparing child and adolescent depressives, there was no difference found in the rate of suicidal attempts, nor in the severity of suicidal ideation or seriousness of suicidal intent (Ryan et al., 1987). There was a difference in lethality of method chosen. The suicide rate escalates alarmingly towards mid-adolescence together with the use of drugs, which has sometimes been equated with self-medication for the increasing experience of depression. Vulnerable adolescents, that is, those with a marked dispositional tendency, tend to show at this time an increase in suicidal ideation and fantasy prior to any depressive "breakdown" (Anthony, 1978).

While adolescent depression is similar to adult depression in many aspects, developmental issues may cloud the diagnostic process. As Garber (1984) points out, adolescence is typically a time of emotional turmoil and mood fluctuations as a result of peer pressure, increasing expectations for adult behavior, and physiological changes. The difficult task of the diagnostician is to distinguish between normal behavior of adolescents and more pathological states. Therefore, he or she must look beyond the mere presence of symptoms and rely more heavily upon duration, severity, and clustering of symptoms than one might at other points in development (Garber, 1984).

Less debatable forms of depression begin in adolescence – one form of depression has been referred by some psychodynamic theorists as a stage of mourning which is gradually worked through during the course of adolescence and is related to the loss of childhood and the final renunciation of parental objects. Typical adolescent depressions are associated with separation-individuation and identity problems having their origin in preoedipal life, normal depressive moodiness characteristic of this stage, and schizoaffective disorders beginning with a depressive coloring.

Anthony (1970) has described two typical syndromes of adolescent depression, the one seemingly having its roots in the first three years of life and the
other characteristically beginning with a depressive turmoil during middle latency. The first is based on a heavily symbiotic relationship with an omnipotent, sadistic mother. These patients are extremely dependent and demonstrate considerable lowered self-esteem, shame, and feelings of weakness. The second type is characterized by a punitive superego and guilt. Anger towards parents for not fulfilling their idolized images is directed against the self, resulting in self-disgust and possibly self-destructive behavior.

**COMPARISON BETWEEN CHILD, ADOLESCENT & ADULT FORMS OF PRESENTATION OF SYMPTOMATOLOGY**

Descriptive clinical studies make a strong case for the position that childhood, adolescent and adult MDD are phenomenologically similar (Kutcher and Marton, 1989; Ryan et al., 1986; Strober et al., 1981), and the one follow-up study of adolescents with depressive symptomatology into adulthood, (Harrington et al., 1990), is consistent with this view. Researchers do note, however, that there is some variability in the symptoms and presentation of MDD associated with developmental factors. Carlson and Kashani (1988) compared clinical signs of depression reported from early infancy through adolescence and found that the basic clinical picture of depression is consistent regardless of age. There is only one study in which direct comparisons of depressed adolescents (n = 26; 15-18 years of age) with depressed adults (n = 27; 18-45 years of age) were conducted (Friedman et al., 1983). Of thirty symptoms, only one (self pity for adolescents) significantly varied in the two age groups providing additional support for the hypothesized consistency of symptoms across age.

Prepubertal children with MDD, more frequently than their adolescent or adult counterparts, present comorbidity with phobias, anxiety disorders and somatic complaints. For these children, these other complaints may appear more salient both due to limitations on the types of verbal expression available to young children as well as the tendency for young children to be more reactive to exogenous events, and therefore less likely to be continually depressed. In adolescence, an irritable,
negativistic or oppositional profile may emerge with the MDD, which again can appear more obvious than the sadness or anhedonia more typically associated with MDD in adults. Hopelessness and increased lethality of suicide attempts is more common in the adolescent depressives then in the prepubertals (Ryan et al., 1987).

The presence of an adult-like syndrome in young people, however, does not rule out developmental differences in symptomatology, i.e., individual symptoms of depression may vary across age groups. Five studies have reported comparative data on symptomatology in adults and young people. Hudgens (1974) and Carlson and Strober (1979) compared symptom endorsement in their populations of hospitalized depressed adolescents with symptoms reported in studies of hospitalized, depressed adults. They reported that their young patients described less terminal insomnia and diurnal mood variation and more suicidal behavior than their older counterparts. Otherwise, the rates of endorsement for depressive symptoms were similar. Strober et al. (1981) reported, however, that certain subtypes of depression (endogenous, psychotic) occurred less frequently in adolescent patients than in adults.

In a similar study, Inamdar et al. (1979) found a number of important age differences. Their adolescent subjects reported more suicidal acts and less terminal insomnia than depressed adults; however, unlike subjects of other studies, their younger subjects reported less anorexia and weight loss, less anergia, and fewer somatic complaints. Very few, or none, of their adolescents described psychomotor agitation or retardation, hopelessness, or psychotic symptoms. These authors concluded that there is enough disparity between the depressive syndrome in the two age groups to warrant cautious comparison and further study. Their study was hampered, however, by its selection criteria. Adolescents who simply acknowledged depressed mood were compared with adults with the full depressive syndrome. The younger group, therefore, may have been overly inclusive and not comparable with a more exclusively defined older population.
Table 1.1: Frequency (%) of DSM-III and DSM-III R Age-Specific Symptoms in Youth and Adults

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Adult (n = 54)</th>
<th>Adolescent (n = 92)</th>
<th>Children (n = 95)</th>
<th>Preschool age (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Felt Sad</td>
<td>93%</td>
<td>99%</td>
<td>95%</td>
<td>100%</td>
</tr>
<tr>
<td>Sad appearance</td>
<td>74</td>
<td>47</td>
<td>64</td>
<td>89</td>
</tr>
<tr>
<td>Crying</td>
<td>50</td>
<td></td>
<td></td>
<td>89</td>
</tr>
<tr>
<td>Irritability</td>
<td>67</td>
<td>83</td>
<td>83</td>
<td>78</td>
</tr>
<tr>
<td>Social withdrawal</td>
<td>78</td>
<td>73</td>
<td>64</td>
<td>89</td>
</tr>
<tr>
<td>Anxiety</td>
<td>30</td>
<td>59*</td>
<td>78*</td>
<td>67*</td>
</tr>
<tr>
<td>Somatic complaints</td>
<td>74</td>
<td>66</td>
<td>83</td>
<td>100</td>
</tr>
<tr>
<td>Sulkiness</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>67</td>
</tr>
<tr>
<td>Conduct disorder / fighting</td>
<td>NA</td>
<td>5</td>
<td>38</td>
<td>11</td>
</tr>
<tr>
<td>Mood-congruent hallucinations</td>
<td>2</td>
<td>10</td>
<td>22</td>
<td>NA</td>
</tr>
</tbody>
</table>

* Separation anxiety
NA = Not Available


PERSPECTIVES ON DEPRESSION IN CHILDREN AND ADOLESCENTS

A great old scholar of the seventeenth century, Robert Burton (who could rightly claim to be the first psychopathologist), set it down in Part I, Section III, Subsection IV, of his Anatomy of Melancholy that maids, virgins, nuns, barren women, and widows were especially prone to insomnia, troublesome sleep, dejection of mind, discontent, weariness, grieving, and weeping “without any manifest cause.” As matters are developing today, he might very well have needed to add children to his list of vulnerable individuals or, at any rate, if we are to maintain his sex bias, little girls.

Initially the work on childhood depression (its existence taken fully for granted), three models were used to investigate it more fully (Anthony, 1978). The empirical model ran along medical lines and has attempted to examine antecedent conditions, concomitant disturbances, natural course, typologies, and therapeutic
response, more especially to drugs. The adult model of depression had exerted a powerful and binding influence and to extrapolate from them what might be expected in the child. Another model that was used to explore childhood depression was a psychodynamic one in which internal conflict plays an important role. This model, in the strong tradition of psychoanalysis, attempted to relate the first few years of childhood to what develops in the subsequent phases of the life cycle. The classical Abraham-Freud theory stemmed from the analysis of melancholic patients where psychotic regression was a prominent feature. It was largely based on superego pathology and incorporated such concepts as aggression turned against the self, excessive orality, narcissistic injury, marked ambivalence, masochism, and the internalization of a lost object. The developmental model postulated that the child at different stages presents himself with different clinical faces involving different constellations of symptoms especially in affective syndromes. The developmental psychologist is also acutely conscious of the child’s “developmental environment”, meaning thereby, the changing input from parents, families, and culture. The clinical picture of the affective disorders was also influenced by the cognitive level, so that the child’s competence and coping skills and problem-solving capacities help to shape the clinical picture.

**Developmental Perspective:** The nosological database in children and adolescents is considerably skimpier. A number of issues have yet to be resolved, however one is whether there are specific subclusters of symptoms or other associated features that differ for different age groups. Another issue is whether modifications in the stated symptoms are necessary to be meaningful developmentally to different age groups. (Carlson & Abbott, 1995).

As Garber (1984:37) cogently asserts, if we apply the developmental model to the study of depression researchers first need to:

1. Determine whether the expression of depressive symptoms varies with development.

2. Identify additional age-appropriate symptoms and areas of dysfunction to include in the diagnostic process.
3. Assess the influence of children’s cognitive, affective, and social competence and their physical maturation on the experience and the expression of depressive symptoms (p. 37).

Developmental variables are likely to influence both the reporting and manifestations of depression. For example, self-report measures require children to endorse specific behaviors as characteristic of themselves but young children have difficulty distinguishing between sadness and anger (Borke, 1973) and are more likely to deny experiencing sadness (Glasberg and Aboud, 1981). In addition, children are unable to provide reliable information on duration of symptoms. It may be more difficult to identify depressed mood in children (Digdon and Gotlib, 1985) as they are more likely to shift rapidly in response to environmental stimuli. Due to these difficulties in assessing depression in children, multiple sources of data are required, including child, parent, teacher, and even peer reports.

A comparison of similar populations of clinically referred depressed preschoolers, children, adolescents, and adults from different sites attempted to address these issues. Certain symptoms had no significant relation with age (for example, depressed mood, poor concentration, insomnia, suicidal behavior), while others did. Anhedonia, diurnal variation, hopelessness, psychomotor retardation (including hypersomnia), and delusions increased in frequency with age. Sad appearance, low self-esteem, somatic complaints, and hallucinations decreased in frequency with age. Symptoms like fatigue, agitation, and anorexia appeared to occur more frequently in the youngest and older populations.

It is possible to speculate on an interaction between core psychopathology and developmental stage for each of those groups. For instance, children whose cognitive structure does not comprehend future and guilt as easily as adolescents and adults do, are less likely to have symptoms like guilt and hopelessness. In addition, although mild endogenous symptoms occur in children, the pervasive anhedonia and psychomotor retardation seen in severely depressed adolescents and adults are extremely uncommon. There is some suggestion that biological, rather than cognitive maturity is the variable accounting for the difference in phenomenology. Somatic
complaints (not feeling well, stomachaches, headaches) relate to depression in children over and above co-occurring anxiety disorders (of which they are a criterion), possibly because they are one of the ways a young child has of saying he or she doesn’t feel well.

The need to consider depressive symptomatology in relation to what is normal for a particular stage of development becomes quite apparent when we consider specific examples. For instance, frequent tearfulness might be one indication of depression in a 16-year old but would have a quite different meaning if we were instead evaluating a two-year old. Thus the prevalence of particular behaviors in normal children must be considered before attributing these to depression (Digdon and Gotlib, 1985).

A developmental model must consider the continuities and discontinuities in psychopathology over time. For example, is a depressed eight year old child likely to be depressed as an adult, even if the specific symptom pattern may change? Or instead, does depression at one stage portend a different form of psychopathology at a later stage? A third possibility is a discontinuity between child and adult disorder so that a depressed child is no more likely to become a depressed adult than is any other child and a depressed adult is unlikely to have a history of childhood depression.

Regarding the relationship between depression in childhood and adulthood, however, knowledge is more limited. In a review of four retrospective studies (Orvaschel, Weissman, and Kidd, 1980), it was concluded that while adult depressive patients were likely to have suffered from family discord and parental abuse, rejection, and inattention, there was no unique clinical picture in childhood. Robins (1979) reviewed follow-up and follow-back studies of childhood psychopathology, reported that adults with depressive disorders were rarely known to clinics as children. However, among those depressed and anxious adults who were known to clinics as children, symptoms of depression, anxiety and somatic complaints were common in their earlier records. In addition, Zeitlin found this continuity to be stronger for depression than for many other syndromes, with the exception of obsessive-compulsive phenomena. While these studies are inconclusive in
demonstrating a strong, predictive relationship between childhood and adult depression, there is no suggestion that they are not independent clinical phenomena.

**Biological Perspective:** Biological bases of depressive disorders have been extensively examined in adults and to a more limited extent, children and adolescents (Emslie et al., 1994). Domains of inquiry range from brain neurochemistry, such as serotonin mechanism, adrenal-hyper-cortical functioning and activity, and sleep architecture to genetics. Also included in this research domain is the study of antidepressants. For example, a great deal of research in psychiatry has focused on serotonergic mechanisms, specifically the role of serotonin (5-HT) in the regulation of psychological and biological functioning. This research has had a major impact on the development of new classes of pharmacological agents for the treatment of depression, such as antidepressant medications that are selective in their serotonin reuptake properties. Likewise, a great deal of interest and research has focused the hypothalamic – pituitary – adrenocortical (HPA) axis as a biological focal point in the environmental stress-depressive response pathway. A number of biological systems and factors, such as prolactin, growth hormone, melatonin metabolism, sleep, and neurotransmitter activity have been linked as of potential etiological relevance to depression (Leibenluft, Fiero, & Rubinow, 1994). There is also evidence to suggest a genetic loading for various depressive disorders, such as major depression and bipolar disorder, with bipolar disorder having the greater genetic component (Herdman, Gough, Liskowski, & Hall, 1995).

**Psychological Perspective:** The domain of psychological influences may best be described as the psychological characteristics, both strengths and weaknesses, as well as vulnerabilities that make up the youngster's emotional and mental health competence. These factors may be either protective or risk factors. A number of cognitive and behavioral processes are particularly relevant to the understanding of the development of depression. Some of these components are linked to theories of depression. For example, the role of self-regulation (Kanfer, 1970) as it relates to problems in self-control (self-monitoring, self-evaluation, self-reinforcement) has been postulated by Rehm (1977) as a model of depression. Problems or deficits in
cognitive appraisal, maladaptive behavioral response to reinforcement, dysfunctional cognitions, and erroneous attributional style have been identified by various contemporary theories and models as linked to the development of depression (Abramson, Seligman, & Teasdale, 1978; Lewinsohn, 1975; Beck, 1967; Ellis, 1962).

Social Perspective: The social component represents the social–environmental domain and by its nature includes a very wide range of influences. Included here are: stressful events, including major negative life events as well as daily hassles and chronic strains; parental influences, including attachment, nurturance, parental discord, support, and parenting style; social support; peer and interpersonal relationships; and a host of social and environmental influences such as exposure to violence, victimization, maltreatment, social disadvantage, peer and social pressures, and school and work stress among others. As an example, parental nurturance is a broad domain that includes parenting behavior such as the nature and use of discipline (e.g., consistent/inconsistent, reasonable/harsh, etc.), communication style, over/under controlling; parental warmth, and other behaviors, characteristics, and competencies (Reynolds, 1998). Oakley-Browne et al. (1995) found that adverse parenting in childhood had an increased risk for depression in adulthood. It is also important to note that, during adolescence, there is often a transition in the relationship that youngsters have with parents and peers, with peers becoming increasingly important as a source for support and attitude development.

Biopsychosocial Perspective: In psychiatry, the biopsychosocial perspective represents a shift from a biomedical model, with its focus primarily on the biology of disease or disorders, to one of integration of psychological and dynamic characteristics, social and environmental forces, and biological bases. The biopsychosocial model is useful for the study and understanding of depression in children and adolescents, as well as adults. The nature, course, severity, treatment viability, and other characteristics of depression in young people as well as adults is a function of the nature of the interactions and the domain-specific characteristics and vulnerabilities.
The biopsychosocial perspective has also been applied to numerous other aspects of human behavior and disorders (Paris, 1994; Shalev, 1993). Meyer's perspective of psychiatric disturbance in part exacerbated by environmental causes, can be viewed as a precursor to the current biopsychosocial perspective. Likewise, the biobehavioral model of depression proposed by Akiskal (1979) is consistent with this perspective and integrates biological, psychosocial, developmental, and environmental stressors as a basis for the development of depression.

Lewis and Lewis (1979) described a psychobiological perspective on depression that recognized psychoanalytic, genetic, and biochemical bases for the development and subsequent treatment of depression in children. This model is also reciprocal and in this factors within the domains may interact as well as affect other domains in a reciprocal manner. For example, some psychosocial stressors have been noted to be responsible for suppressed T-lymphocyte function (Brosschot et al., 1994) and a host of hormonal changes. Such abnormalities in immune function and endocrine changes can increase vulnerability to depression. In adolescents, major negative life events have been linked to reduced immune function and neuroendocrine changes (Birmaher et al., 1994).

The tripartite model proposed by Reynolds (1997a) is a biopsychosocial perspective on depression in children and adolescents. Similar to the model of reciprocal determinism postulated by Bandura (1978) for understanding human behavior, the biopsychosocial prospective considered reciprocal pathways. The strength of these pathways in the pathogenesis, maintenance, or exacerbation of depression or depressive disorders may vary as function of developmental level, individual strengths and deficits within each domain, nature of the reciprocal interactions, and the nature of the depressive outcome. Two aspects, which are critical, are that this model is dynamic and developmental. There is evidence to infer that depression in children is, in a number of respects, different from depression in adults. There are a number of possible developmental trajectories that may relate to differences in the nature as well as expression of depression in youngsters as compared to adults.
Magnusson and colleagues (Magnusson & Torestad, 1993; Stattin & Magnusson, 1989) have delineated an interactional approach with a major focus on the developmental and biological nature of person, and in particular adolescent and environment interactions, cumulating in the description of a holistic, dynamic model of personality. This approach also has utility for the study and understanding of depression in young people. Remschmidt (1992) notes that the interactions between biological and psychosocial variables that influence the development of psychopathology in children and adolescents may be multiplicative as well as additive. Thus, a youngster may show a deficit in the interpersonal skills domain as a psychological influence, or may demonstrate competence in interpersonal skills that may reduce vulnerability for depression. In a similar vein, personality may enhance psychological resilience or act as a factor that exacerbates biological or social-environmental events. Likewise, biological competence may provide a protective factor for adverse or stressful major life events.

**EPIDEMIOLOGY OF DEPRESSION IN ADOLESCENTS**

Reporting meaningful information on rates of depression in either clinically referred or community samples of children is complicated by (1) whether the study is measuring depression as a symptom, a syndrome, or a disorder; (2) the age group of children measured, which, in studies, often varies from 4 to 16 years; and (3) the fact that the clinician is dealing with a symptom or disorder that requires a discussion with the child, not just reports about his or her behavior (that is, the most reliable way of obtaining information is time-intensive, and rates depend on the measures used); and (4) how impairment is defined (Carlson & Abbott, 1998).

Population studies of children and adolescents have reported prevalence rates of depression in children and ranging between 0.4% and 2.55% in children and between 0.4% and 8.3% in adolescents (Anderson and McGee, 1994; Lewinsohn et al., 1993a, 1994). The lifetime prevalence rate of major depressive disorder (MDD) in adolescents has been estimated to range from 15% to 20%, which is comparable with the lifetime rate of MDD found in adult populations, suggesting that depression in adults often begins in adolescence (Kessler et al., 1994; Lewinsohn et al., 1993a).
The few epidemiological studies on dysthymia disorder (DD) have reported a point prevalence rate from 0.6% to 1.7% in children and 1.6% to 8.0% in adolescents (Lewinsohn et al., 1993a, 1994). According to Charoensuk (2007) the prevalence rate of depressive symptoms varied from 20-21% among Thai adolescents.

Roughly 15% of adolescents admit to having suffered from depression at some time or other and increase with age with a preponderance of girls over boys (Essau & Petermann, 2000). Major depression was prevalent in 5.8% of adolescents aged 16 to 17-year-olds and during lifetime in 11.45, 4 girls for every boy. Dysthymia without major depressive episodes was diagnosed in 1.1%, two girls for every boy (Olsson, 1998). In a study among homeless and runaway adolescents (aged 10 to 16 years).

In children, MDD occurs at approximately the same rate in girls and in boys, whereas in adolescents, the female-to-male ratio is approximately 2:1, paralleling the ratio reported in adult MDD (Kessler et al., 1994a; Lewinsohn et al., 1994; Fleming and Offord, 1990). While the nature of this sex difference is as yet unclear, it has been attributed to genetics, increased prevalence of anxiety disorders in females, biological changes associated with puberty, cognitive predisposition, and sociocultural factors (Breslau et al., 1995).

Depression is more common as a symptom than as a syndrome. In a large high school sample rated with both the Beck Depression Inventory (BDI) and the Center for Epidemiologic Studies of Depression Scale (CED-D), rates of mild depression were 13.1 percent and 31.4 percent, respectively, and rates of severe depression were 4 percent and 12.1 percent, respectively. Among nonclinical samples, the rate among 9-year-olds was about 1.8 to 2.5 percent and among adolescents about 4.5 to 6 percent. In clinical samples the age related increase is also apparent. In preadolescents and adolescents, whether the child is an inpatient or outpatient made a difference in rates. However, rates of around 15 to 20 percent have been reported for hospitalized preadolescents and rates of 28 to 40 percent for hospitalized adolescents. The sex ratio, which shows a female preponderance in adults, is reversed in prepubertal youngsters, that is, boys outnumber or equal girls (Carlson & Abbott, 1995).
The twentieth century has been called the century of the child. With advancements in the medical sciences, growing research in developmental psychology and education, and increasing concern about social deviance and delinquency in children, the importance of childhood in the life span of individuals was greatly emphasized (Malhotra, 2005). In any country, adolescents represent a major potential human resource for the overall development of a nation. The International Conference on Population and Development (ICPD) held at Cairo in September 1994, recommended among other things, focussing on all issues concerning the adolescent in their entirety and has suggested development of an integrated approach to their health, education and social needs.

Though accorded its due status in the West, child psychiatry is still lagging behind in the developing world. Eighty percent of the world’s children live in developing countries, where mental health services are meagre or nonexistent. In addition, the stresses of rapid social change, such as urbanization and industrialization, have exacerbated the mental health problems of children. Developing countries have been struggling with the issue of survival of newborns, and children and mental health have been relegated to a low priority status. In a multicentre WHO collaborative study of childhood mental disorders in primary healthcare in four developing countries, Giel et al. (1981) reported that 12%-29% of children attending primary healthcare facilities in Colombia, India, Senegal and Sudan had identifiable psychiatric disorders, 80%-90% of which were consistently missed.

In Pakistan, children with chronic medical illnesses were shown to be 12 times more likely than controls to have psychiatric morbidity. Of these children (in the age group of 5 – 15 years), 80% were likely to suffer from mood disorders, dysthymic disorder and generalized anxiety disorder (Taj et al., 2002). Prevalence figures from India provide varying estimates of psychiatric disorders in children, namely 2.5% (Nandi et al., 1975); 35.6% (Lai and Sethi, 1977); 33.7% (Deivasigamani, 1990).
These wide variations in the prevalence rates appear to be due to the differences in the methodologies used in these studies.

There are a few scattered epidemiological studies identifying prevalence rates of psychological disturbance among Indian adolescents. Sethi, Gupta & Rajkumar (1972) found a 2% prevalence among 509 rural adolescents (aged 11 – 20 years), based on an interview with the head of the family. Rao (1978) identified a point prevalence rate of 19.62% in 428 high school students (aged 13-16 years), using the General Health Questionnaire. Also, using the GHQ-60 he identified 22.8% of 13-16 year old adolescent girls as distressed. The gender distribution indicated rates of 18.8% in boys and 22.8% in girls. D'Arcy & Siddique (1984) used the GHQ-30 and obtained an overall prevalence rate of psychological distress of 27.5% in urban adolescents aged 14-17 years. Rozario (1988) reported an overall point prevalence rate of 6.42%, based on teacher report, in a sample of 1371 high school students (aged 12 – 16 years). The proportion of disturbed boys to girls was 11:1. The use of teachers as informants may have led to the under-identification of the less observable emotional problems in girls. Dalal (1989) screened 663 girls (aged 12 – 16 years), on the General Health Questionnaire and obtained a prevalence rate of 30.92%.

In a more recent study (Malhotra et al., 1992) on school children done on a stratified random sample using a multistage, multi-informant procedure, involving clinical interviews and ICD-10 diagnostic criteria, an overall prevalence of 6.33% was found in 4 to 11-year-old school children living in Chandigarh, India.

Sinha (1997) used a two-stage screening procedure and reported that 5.69% of school-going Indian adolescent boys, aged 14-15 years, were emotionally disturbed. Bhola & Kapur (2000) screened 446 school going girls for identifying the prevalence of emotional disturbance using GHQ and the Youth Self-Report and obtained a prevalence rate of 10.99% among adolescent girls aged 13-16 years. There is remarkable emergence of clinical observation, developmental findings, and epidemiological data, pointing to this striking gender difference in adolescent symptomatology (Schonert-Reichl & Offer, 1992; Nolen-Hoeksema & Girgus, 1994; Ollendick & King, 1994). Adolescent girls constitute a vulnerable group that
clearly has a proclivity towards inwardly turned symptoms of distress such as depression and anxiety.

**Ganguli (2003)** examined the growth, beliefs, emotions, ambitions and relation with parents/teachers among 230 adolescents (grades 8th and 9th) in a rural area near Pune. Results reported that changes of mood were felt by 57 male students (58.76%) and 61 female students (60.9%). The study further revealed that the female students felt more depression (80.4%) and among males 13.4% reported occasional feeling of depression. The most common cause of depression was feeling lonely. Equivocal findings reported by Ghai et al. (2000) stated in his book that adolescents, both boys and girls were prone to experience a state of depression due to various reasons.

**Khurana et al. (2004)** found 20.7% of children being high on hopelessness and 8% had depression. Among children with high hopelessness, 3.2% had ever attempted suicide and 8.3% gave history of suicidal attempts. Eighteen percent of youth (aged 11, 13 and 15) reported symptoms of depression. A higher proportion of females (25%) reported depressive symptoms than males (10%) (Saluja et al., 2004).

Major depression is prevalent in 0.5% - 1% of school-aged (aged 5-13 years) children over a six-month period and increases to 3% in adolescents. The prevalence rate continues to rise for boys up to adolescence (Malhotra et al., 2005). According to Bhatia and Bhatia (2007), major depression affects 3 to 5 percent of children and adolescents.

Addressing the needs of the adolescents in the **Tenth Five-Year Plan (2002-2008)**, Mr K C Pant, Deputy Chairperson, Planning Commission said that 10th Five-Year Plan will ensure and integrated approach in handling Adolescent Welfare and Development. The strategy is to harness the strengths of educators, NGOs, Youth and Community based organization towards catering to all the unmet needs to the population segment. The Planning Commission report declares that the adolescent have very special and distinct needs, which can no longer be overlooked (Ganguli, 2003).
A diagnosis may be viewed as a hypothesis that represents "the clinician's best judgment" (Rush, 1990, p. 5) of the individual's condition. The diagnosis of depression involves the evaluation and comparison of the characteristics and duration of an individual's symptoms with criteria and classification rules for one or more disorders. In this manner, diagnosis constitutes an assessment or measurement procedure. Sources of potential measurement error in making a diagnosis include error due to the informant, the diagnostic instrument (e.g., clinical interview), the diagnostician, as well as the classification system. Research suggests that the diagnosis of psychopathology and, more specifically, depressive disorders in children is not perfect (Vitiello, Malone, Buschle, Delaney, & Behar, 1990; APA, 1980). For the evaluation of depression in children and adolescents, diagnosis is one method with the outcome being a characterization of specific disorder or disorders based on a formal classification scheme. Other measurement procedures, such as the assessment of severity of depressive symptoms, either by clinical interview or self-report without comparison to a classification system, are often used in clinical and research applications with children and adolescents. The latter may be viewed as providing a focus on the assessment of depression as a syndrome rather than delineating categorically distinct depressive disorders.

There are a number of classification systems that have been developed or provide for the diagnosis of depressive disorders in children and adolescents. Most of the current systems are based on a taxonomic approach for the differentiation of type and subtypes of depressive disorders based on particular clusters of symptoms and symptom duration. In North America the primary system used for the classification of depressive disorders in children, adolescents, and adults is the DSM-IV (APA, 1994).

For certain research applications, the Research Diagnostic Criteria (RDC), developed by Spitzer et al. (1978) is used for the diagnosis of depressive subtypes. The RDC provides greater specificity of subtypes of depression and may be evaluated using a formal diagnostic clinical interview, the Schedule for Affective Disorders and...
Schizophrenia (SADS; Endicott & Spitzer, 1978) for adults as well as adolescents, and the Kiddie SADS for children (K-SADS; Puig-Antich & Chambers, 1978), which has also been used for adolescents (e.g., Lewinsohn, Rohde, & Seeley, 1995).

Poznanski and Zrull (1970) using a chart-review method, provided descriptions of clinical symptoms constituting depression in children. Later, Poznanski and her colleagues (Poznanski et al., 1985; Poznanski, 1982) reformulated their clinical description of symptomatology for depression in children, and developed a clinical interview for the assessment of depression in children (Poznanski et al., 1984; Poznanski, Cook, & Carroll, 1979). In the early 1970s, Weinberg and colleagues proposed a set of criteria for depression in children (Weinberg et al., 1973) with Weinberg and Brumback (1976) providing diagnostic criteria for mania in children. Although the Weinberg criteria are rarely used today, they are noteworthy for their early impact as well as their similarity to symptom specifications for current DSM-IV criteria for depressive disorders in young people.

There have been a number of suggestions that criteria for DSM depressive disorders are too stringent and result in an underestimation of persons with significant levels of depressive symptoms. Thus, a person may show a level of depressive symptoms that is not sufficient to meet criteria for a DSM disorder yet, by its symptom severity level, may be considered clinically relevant and a valid target for intervention. Bland (1994) noted the problem of threshold in the use of DSM-specific criteria for diagnosis, in that a certain number of symptoms are required for a diagnosis and the patient who falls one symptom short of this does not receive a diagnosis, even when presenting symptoms are severe.

Gotlib, Lewinsohn, and Seeley (1995) found that youngsters who had clinically significant levels of depressive symptoms but did not meet criteria for major depression were twice as likely to develop a psychiatric disorder as compared to a nondepressed group, and also demonstrated significantly poorer psychosocial functioning than the nonsymptomatic group. Youngsters with clinically significant depressive symptoms were similar in psychosocial functioning to a comparison group of youngsters with major depression. These results suggest the clinical importance of
attending to youngsters who may not meet criteria for major depression yet show significant levels of depressive symptomatology.

**Judd, Rapoport, Paulus, and Brown (1994)** examined depressive symptom data from the National Institute of Mental Health Epidemiological Catchment Area (ECA) study and developed a clinical condition that they described as "subsyndromal symptomatic depression." Similarly, **Horwath, Johnson, Klerman, and Weissman (1994)** focused on ECA incidence data specific to persons who developed an episode of major depression over a one year interval between interviews. They found that persons with prior symptoms of depression or a dysthymic disorder were at relative risk for depression, with 50% of individuals who developed a major depressive episode demonstrating a history of depressive symptoms. The authors highlight the clinical importance of depressive symptoms by noting that "if depressive symptoms could be identified and treated before major depression first developed, many first onset cases of major depression could potentially be prevented" (p. 333), and that "measures designed to identify this group prior to the onset of major depression may represent an important step in primary prevention" (p. 333). Although the data from **Judd et al. (1994)** and **Horwath et al. (1994)** is specific to adults, it can be generalized to children and adolescents.

**DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS (DSM)**

Depressive disorders in the DSM-IV fall under the category of mood disorders, and include major depression, bipolar disorders, cyclothymic disorder, and dysthymic disorder as primary mood disorders. Disorders are differentiated by specific clusters of symptoms, age of onset, and the duration of symptoms. Disorders that are most likely to be found in school-aged youngsters are major depression and dysthymia, although adjustment disorder with depressed mood is also diagnostically relevant. The depressive disorders subsumed under the domain of mood disorders in the DSM-IV. It should also be noted that diagnostic categories of: mood disorder due to medical condition; substance-induced mood disorder; and mood disorder not otherwise specified may also be diagnosed in children and adolescents.
Table 1.2: DSM-IV Mood Disorders in Children and Adolescents

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Subdiagnosis</th>
</tr>
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<tbody>
<tr>
<td>Major depressive disorder</td>
<td>single episode</td>
</tr>
<tr>
<td></td>
<td>recurrent</td>
</tr>
<tr>
<td>Dysthymic disorder (early onset)</td>
<td></td>
</tr>
<tr>
<td>Depressive disorder not otherwise specified (NOS)</td>
<td></td>
</tr>
<tr>
<td>Bipolar disorder I</td>
<td>single manic episode</td>
</tr>
<tr>
<td></td>
<td>most recent episode depressed</td>
</tr>
<tr>
<td></td>
<td>most recent episode hypomanic</td>
</tr>
<tr>
<td></td>
<td>most recent episode manic</td>
</tr>
<tr>
<td></td>
<td>most recent episode mixed</td>
</tr>
<tr>
<td></td>
<td>most recent episode unspecified</td>
</tr>
<tr>
<td>Bipolar disorder II</td>
<td>hypomanic</td>
</tr>
<tr>
<td></td>
<td>depressed</td>
</tr>
<tr>
<td>Cyclothymic disorder</td>
<td></td>
</tr>
<tr>
<td></td>
<td>May also include:</td>
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<tr>
<td></td>
<td>with melancholic features</td>
</tr>
<tr>
<td></td>
<td>with atypical features</td>
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<tr>
<td></td>
<td>with catatonic features</td>
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<tr>
<td></td>
<td>with postpartum onset</td>
</tr>
<tr>
<td></td>
<td>chronic</td>
</tr>
<tr>
<td></td>
<td>severity/psychotic/remission specifiers</td>
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<tr>
<td></td>
<td>course specifiers:</td>
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<tr>
<td></td>
<td>with seasonal pattern</td>
</tr>
<tr>
<td></td>
<td>with rapid cycling</td>
</tr>
<tr>
<td></td>
<td>with/without interepisode recovery</td>
</tr>
</tbody>
</table>

\[1\] with melancholic features

\[2\] with atypical features

\[3\] with catatonic features

\[4\] with postpartum onset

\[5\] chronic

\[6\] severity/psychotic/remission specifiers

\[7\] with seasonal pattern

\[8\] with rapid cycling

\[9\] with/without interepisode recovery
Major depressive disorder: Major depressive disorder (MDD) in DSM-IV is presented as a relatively severe, acute form of depression. Major depressive disorder may be classified as a single episode or recurrent, the latter specifying a history of previous MDD episode(s). As a disorder, MDD is defined by the following criteria and symptomatology: the presence of five of the following symptoms, one of which is either dysphoric or irritable mood, or loss of interest or pleasure in all or almost all activities. The other symptoms include: sleep problems as manifested by insomnia or hypersomnia; complaints or other evidence of diminished ability to think, decision making, or difficulty in concentrating; loss of energy or general fatigue; eating problems as manifested by decreased or increased appetite or significant weight loss or gain (in young children failure to make expected weight gains is symptomatic); psychomotor retardation or agitation; suicidal or morbid ideation, death wishes, or suicide attempts; and feelings of self-reproach, worthlessness, or excessive or inappropriate guilt (which may be delusional). Symptoms need to be present nearly every day for a period of at least two weeks. Exclusion criteria are also specified that preclude a number of other pathologies, such as organic mental disorder and other etiologies (i.e., a normal response to bereavement) as concomitant problems, for a diagnosis of major depression.

Dysthymic disorder: In the DSM-IV, dysthymic disorder represents a relatively chronic depressive disorder that is typically less severe in symptom distress than major depression, but typically of greater duration. Diagnostic criteria for dysthymic disorder in children and adolescents includes a depressed or irritable mood for most of the day, and manifested most of the time over a period of at least one year, although there may be periods of up to two months during which symptoms are not present. In addition to symptoms of depressed or irritable mood, at least two of the following symptoms must be present when depressed: appetite loss or gain, insomnia or hypersomnia, fatigue or low energy level, low self-esteem, difficulty in concentrating or problems in making decisions, and feelings of hopelessness. Exclusion criteria include: showing no evidence of major depressive episode during the initial year of the disorder; never having a manic or hypomanic episode; and not being superimposed on a chronic psychosis, or due to or maintained by an organic factor. Diagnostic criteria for dysthymia in children and adolescents differ from those for adults for whom symptoms of this disorder must be present for two years for a diagnosis.
Table 1.3: Comparisons of Child-related Modifications of Major Depressive Episode in DSM-III, DSM-III-R, DSM-IV, and ICD-10

<table>
<thead>
<tr>
<th>DSM-III</th>
<th>DSM-III-R, DSM-IV</th>
<th>ICD-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysphoric mood; includes sad, blue, hopeless, irritable: <em>for child &lt; 6 yr old, mood inferred from persistently sad faces</em></td>
<td>Depressed mood most of the day, most days; <em>irritable mood for youth</em></td>
<td>Depressed mood most of the day, most days; not influenced by circumstances</td>
</tr>
<tr>
<td>Loss of interest or pleasure (anhedonia); <em>in children &lt; age 6, signs of apathy</em></td>
<td>Markedly diminished interest in all or almost all activities; observation by others of apathy</td>
<td>Markedly loss of interest or pleasure in activities normally pleasurable</td>
</tr>
<tr>
<td>Loss of energy; fatigue</td>
<td>Fatigue or loss of energy nearly everyday</td>
<td>Decreased energy or increased fatigability</td>
</tr>
<tr>
<td>Poor or increased appetite or weight; <em>in children &lt; age 6, failure to make expected weight gain</em></td>
<td>Significant weight loss or gain; <em>in children, consider failure to make expected weight gain</em></td>
<td>Change in appetite with corresponding weight change</td>
</tr>
<tr>
<td>Insomnia or hypersomnia</td>
<td>Insomnia or hypersomnia nearly everyday</td>
<td>Sleep disturbance of any type</td>
</tr>
<tr>
<td>Feelings of worthlessness, guilt, self-reproach</td>
<td>Feelings of worthlessness, guilt, self-reproach</td>
<td>Unreasonable feelings of self-reproach or guilt</td>
</tr>
<tr>
<td>Diminished ability to think or concentrate (slowed thinking, indecisiveness)</td>
<td>Diminished ability to think or concentrate nearly everyday</td>
<td>Diminished ability to think or concentrate; indecisiveness or vacillation</td>
</tr>
<tr>
<td>Recurrent thoughts of death or suicide; suicide attempts</td>
<td>Recurrent thoughts of death or suicide</td>
<td>Recurrent thoughts of death or suicide</td>
</tr>
</tbody>
</table>

In all cases signs and symptoms must be present for at least a two-week period and must represent a change from previous functioning. In DSM at least 5 symptoms should be present and in ICD, at least 4. In DSM, at least one of the symptoms must be either a depressed (or irritable) mood or loss of interest or pleasure. In ICD, at least 2 of the first three criteria should be present.

COMORBIDITY

The study of the coexistence of other psychopathology along with depression in children, adolescents, and adults has emerged as a significant field of inquiry (Anderson & McGee, 1994; Caron & Rutter, 1991). Comorbidity is a term originally used in medicine to describe the existence of a distinct, separate disease or disorder that is present during the course of a primary disorder or disease that is the focus of attention.

A question in the study of comorbidity is that of the temporal relationship between disorders, that is, do consistent findings suggest that one disorder commonly predates another, and if so, does this indicate a predisposition of one disorder to another? Towards understanding the nature of comorbidity, investigators have begun to examine the course and potential causal pathways of depression and comorbid psychopathology (Loeber, Russo, Stouthamer-Loeber, & Lahey, 1994; Caron & Rutter, 1991).

The examination of the coexistence of other psychopathology and depression in children and adolescents is important for a number of reasons. As Alessi and Magen (1988) noted, the existence of multiple disorders may in some individuals lead to the development of a depressive disorder. Likewise, the co-occurrence may create an increased stress response with psychological and biological interactions. Clearly the biopsychosocial approach is critical when examining comorbidity.

Clinical (e.g., Biederman et al., 1995a; Ryan et al., 1987; Kovacs et al., 1984a, b) as well as epidemiological investigations (Anderson and McGee, 1994; Angold and Costello, 1993) have shown that 40% to 70% of depressed children and adolescents have comorbid psychiatric disorders, and at least 20% to 50% have two or more comorbid diagnoses. The most frequent comorbid diagnoses are DD and anxiety disorders (both at 30% to 80%), disruptive disorders (10% to 80%), and substance abuse (20% to 30%). Except for substance abuse, MDD is more likely to occur after the onset of other psychiatric disorders (e.g., Biederman et al., 1995a; Reinherz et al., 1993). However, conduct problems may develop as a complication of the depression and persist after the depression remits (Kovacs et al., 1988).
A few studies have reported that more than 60% of depressed adolescents have comorbid personality disorders, with borderline personality disorder accounting for 30% of all comorbid personality disorders (Marton et al., 1989). After the depression has remitted, however, personality disorder symptoms are no longer evident highlighting the importance of giving only provisional personality disorder diagnoses during acute depressive episodes. Approximately 70% of the early-onset DD patients have a superimposed MDD and 50% have other preexisting psychiatric disorders, including anxiety disorders (40%), conduct disorder (30%), ADHD (24%), and enuresis or encopresis (15%), with 15% having two or more comorbid disorders (Kovacs et al., 1994a).

In particular, youths with “double depressions” (MDD and DD) have been found to have more severe and longer depressive episodes, a higher rate of comorbid disorders, more suicidality, and worse social impairment than youths with MDD and DD alone (Ferro et al., 1994; Kovacs et al., 1994a). The comorbidity of depression and anxiety may also have clinical and functional implications as evidenced by studies showing an increased severity and duration of depressive symptoms, increased risk for substance abuse, increased suicidality, poor response to psychotherapy, and more psychosocial problems (e.g. Brent et al., 1993a; Kendall et al., 1992).

The comorbidity with major depression was examined by Mitchell, McCauley, Burke, and Moss (1988) separately for children (n=45) and adolescents (n=50) who were seen in a clinical setting based on RDC using the K-SADS. The most frequent comorbid disorder in both children and adolescents was separation anxiety (42% and 44%, respectively). Conduct disorder was comorbid in 14% of adolescents and conduct disorders were only present in preadolescent boys, whereas in adolescents, a somewhat greater proportion of girls (17%) than boys (10%) had conduct disorder comorbid with major depression. Lewinsohn et al. (1995) examined a sample of 1507 youngsters aged 14 to 18 and reported that there were 342 youngsters with a lifetime diagnosis of major depression. Of these adolescents, there were 67 with comorbid anxiety disorders, 78 with substance abuse disorder, and 36 with disruptive behavior.
Genetic predisposition has also been noted as a potential explanation for some patterns of comorbidity. Walters et al. (1992), using evidence from twin studies suggests that there may be a partial shared common genetic basis for bulimia nervosa and major depression. Similarly, Paul (1988), in examining the comorbidity between anxiety disorders and depression and a range of biological aspects of these disorders, suggests that anxiety and depression may represent different phenotypes of a common neurobiological substrate.

**ASSESSMENT OF DEPRESSION IN CHILDREN AND ADOLESCENTS**

Assessment is a procedure basic to the identification of depression in children and adolescents for clinical purposes, as well as for use in the study of biological, social, and psychological characteristics; sequaele; outcome; determination of treatment efficacy; and other research applications. There are two primary categories of assessment measures: those designed for obtaining a diagnosis, and measures developed to evaluate the severity of depressive symptomatology. In general clinical practice and in a multitude of research applications, the assessment of depression in children and adolescents is typically accomplished using self-report severity measures or clinical interviews, the latter including diagnostic measures and severity measures.

A characteristic of all early studies was the lack of methodological rigour as exemplified by (1) the confusion between sad affect and depressive syndrome, (2) the lack of standardized diagnostic criteria, (3) the lack of descriptions and of reliability studies of the assessment methods used, (4) the uncontrolled nature of the experimental design, (5) the frequency of assertions going beyond the data, and (6) the lack of statistical analysis (Puig-Antich & Gittelman, 1982).

In adult populations, symptomatic assessments are mainly based on patient interviews. Other sources of information are used only when the patient has severe cognitive impairment or when the clinician suspects distortions in self-report. In contrast, with children, two sources of information besides the patient have been used routinely in clinical practice and research; the parent and the school. These are necessary because of the cognitive limitations inherent to the developmental stage of
the prepubertal child, which makes it usually impossible for him to provide an accurate chronological structure for the present episode of illness.

At the same time, it is difficult for others, such as parents and teachers, to be aware of the child’s moods and feelings. Therefore, although the child’s ability to report past status is limited, most researchers working with depressed children think the child is the best reporter for present state and place great emphasis on the interview with the child to evaluate current emotional status (Carlson & Cantwell, 1980; Cyrtryn et al., 1980; Puig-Antich et al., 1978). The emerging consensus seems to be that, in the assessment of psychiatric symptoms that are manifested mostly intrapsychically and which reflect subjective phenomena (emotions, feelings, and ideas), information obtained from the child is essential. Thus, these symptoms may remain totally unknown to parents, especially when they are not obviously responsible for changes in behaviour. This situation is more likely for an older child, as with increasing age children tend to become more private about their inner lives. The validity of the child interview data is further suggested by the Isle of Wight follow-up, where previous data obtained from the children’s interviews and interviews with parents were more predictive of later psychopathology than ratings by teachers and parents.

The use of self-report questionnaires for the assessment of depressive symptomatology in young people has witnessed a rapid growth in research and clinical applications. The manner in which we assess depression in youngsters guides how we organize our perspective of depressive phenomena. For the determination of depression according to formal classification criteria that includes both inclusion and exclusion components, the standard practice in research investigations has been the use of diagnostic clinical interviews. Structured clinical interviews are typically formulated to evaluate all symptoms, their duration, and potential exclusion criteria as specified by a formal set of diagnostic criteria.

On the other hand, there are several characteristics of depression that support the use of self-report assessment procedures, given linguistic and metacognitive competence in the child. Depression as an internalizing disorder includes primary
symptoms that are internal to the youngster and are not easily observable. Cognitive symptoms of guilt, self-deprecation, suicidal ideation, hopelessness, and feelings of worthlessness are depressive symptoms that are subjective to the child. Some vegetative symptoms such as insomnia, appetite loss, and other problems are sometimes difficult for others to observe and may go undetected by parents and significant others (Reynolds, 1998).

Self-report severity measures typically evaluate a range of depressive symptoms with the assessment format specific to the depth of symptom expression e.g., frequency of occurrence, severity, etc. (Reynolds, 1994). Using the nomenclature of Compas, Ery, and Grant (1993), single-scale self-report depression measures assess depressed mood in youngsters, and self-report measures that are components of a comprehensive assessment measure or that were developed to assess all symptoms of classification system specified depressive disorders allow for the assessment of depressive syndromes. Thus, self-report scales are not designed as diagnostic measures.

Sensitive parents are also usually considered the best informants for observable behavior, including past verbal statements by the child. Regarding the child’s intrapsychic affective and cognitive content, the parents are possibly better informants when their children are younger and more communicative, when the child’s intrapsychic symptoms clearly alter behaviour, and when they themselves are empathic, relatively free of psychopathology, know their child well, and their relationship to the child has a positive emotional tone.

There are a number of well-known validated instruments used to diagnose pediatric depression and other child psychiatric disorders including the Child and Adolescent Psychiatric Assessment (CAPA; Angold and Costello, 2000), K-SADS-PL (Present and Lifetime Version; Kaufman et al., 1997) and other versions of K-SADS (Ambrosini et al., 2000), Diagnostic Interview Schedule for Children (DISC; Shaffer et al., 2000) and Diagnostic Interview for Children and Adolescents (DICA; Reich, 2000a). Such “fully structured” or “respondent based” instruments are
frequently used in epidemiologic studies because lay interviewers may be quickly trained to reliably administer them (Ryan, 2001).

Limitations on the use of self-report are primarily developmental. Linguistic competence, including reading and comprehension ability, are important considerations when using self-report measures. Likewise, it is important to consider metacognitive demands of the measure. Thus, task demands go beyond reading to include the ability to recognize one's emotional and behavioral characteristics as they apply to a specified time-frame and evaluate the frequency, severity, or nature of these characteristics (Reynolds, 1992b).

CLINICAL CHARACTERISTICS

NATURAL COURSE:

**Major depressive disorder:** Clinical and epidemiological studies in children and adolescents have reported that the mean length of an episode of MDD is approximately 7 to 9 months (Rao et al., 1995; Lewinsohn et al., 1994; Strober et al., 1993). Approximately 90% of the major depressive episodes have remitted by 1.5 to 2 years after the onset, with 6% to 10% becoming protracted (Sandford et al., 1995; Strober et al., 1993).

Longitudinal studies of clinical (Rao et al., 1995; Sanford et al., 1995; Strober et al., 1993) as well as epidemiological samples (Lewinsohn et al., 1994; Fleming et al., 1993; Warner et al., 1992) have consistently found that MDD is a recurrent condition with a cumulative probability of recurrence of 40% by 2 years and 70% by 5 years.

Investigations of adolescents going into adulthood (Rao et al., 1995) and adult patients using catch-up longitudinal designs where initial diagnoses have been retrospectively reconstructed from clinical data summaries (Harrington et al., 1990) have also shown that depression persists into adulthood, with recurrence rates estimated to be 60% to 70%. These results are identical with the 70% rate of recurrence reported in a 5-year prospective follow-up of adults with unipolar depression.
depression (Corynell et al., 1989). Very few studies have investigated the psychosocial and biological factors that may contribute to the recurrence of childhood MDD (e.g., Warner et al., 1992; Asarnow and Ben-Meir, 1988). For example, it has been reported that depressed children who live in conflictive family environments have higher recurrence rates than those who live in families with less conflict (Asarnow et al., 1994; Asarnow and Ben-Meir, 1988).

The reported MDD duration values for child and adolescent patients have included a median of 16 weeks (Keller et al., 1988) and means from 32 to 36 weeks (Strober et al., 1993; Kovacs et al., 1984a). Regarding MDD recurrence in treated children and adolescents, Kovacs et al. (1984b) found that 26% of recovered patients relapsed within 1 year and that 40% had relapsed within 2 years.

Risk of developing Bipolar I and II disorders: Follow up studies have found that 20% to 40% of adolescents with MDD develop bipolar I disorder (periods of MDD and mania) within a period of 5 years after the onset of depression (Rao et al., 1995; Geller et al., 1994; Strober et al., 1993). Clinical characteristics associated with an increased risk of developing bipolar I disorder in adolescents and adults with MDD include early-onset depression, depression accompanied by psychomotor retardation or psychotic features, family history of bipolar disorder or heavy loading for mood disorders, and pharmacologically induced hypomania (e.g., Akiskal et al., 1995; Geller et al., 1994).

Dysthymic disorder: Early-onset DD has protracted course, with a mean episode length of about 4 years, and is associated with an increased risk for subsequent MDD (70%), bipolar disorder (13%), and substance abuse (15%) (Kovacs et al., 1994a; Lewinsohn et al., 1991).Dysthymic children usually have their first episode of MDD 2 to 3 years after the onset of DD, suggesting that DD is one of the ‘gateways’ to the development of recurrent mood disorders and indicating the need to develop preventive interventions targeted at this population (Birmaher et al., 1996; Kovacs et al., 1994a).
SEQUELAE

During the episode of depression, children and adolescents with clinical depression frequently experience impairment in school performance and relationships with others (e.g., Williamson et al., 1995c; Rao et al., 1995; Strober et al., 1993; Hammen, 1990). Furthermore, independent of the depression, other factors, such as comorbid psychiatric disorders, poor family functioning, low socio-economic status, and exposure to stressful life events impact the psychosocial functioning of the depressed patients, emphasizing the importance of managing these problems in addition to treating the depression (e.g., Warner et al., 1995; Asarnow et al., 1994; McCauley and Myers, 1992). Depression in children and adolescents is also associated with an increased risk of suicidal behaviors, homicidal ideation, tobacco use, and abuse of alcohol and other substances during later adolescence (Deykin et al., 1992) and adulthood (Rao et al., 1995). In general, MDD precedes the onset of alcohol or substance abuse by an average of 4.5 years, providing a window of opportunity for the prevention of substance abuse in depressed adolescents.

Prospective studies have also found that after recovery, children and adolescents may continue to show subclinical symptoms of depression, negative attributions, impairment in interpersonal relationships, increased smoking, impairment in global functioning, early pregnancy, and increased physical problems (e.g., Rao et al., 1995; Kovacs et al., 1994b; Rohde et al., 1994; Puig-Antich et al., 1993; Nolen-Hoeksema et al., 1992).

Paralleling the secular increase in MDD, the adolescent suicide rate has quadrupled since 1950 (2.5 to 11.2 x 100.000) and currently represents 12% of the total mortality in this age group (e.g., Lewinsohn et al., 1993b; Brent et al., 1988). Similarly, adolescent suicide attempts have also increased in recent years and have been found to have 1-year and lifetime prevalence rates of 1.7% to 5.9% and 3.0% to 7.1%, respectively (Fergusson and Lynskey, 1995; Centers for Disease Control, 1994; Lewinsohn et al., 1993b).

Numerous factors have been associated with the onset, duration, and recurrence of early-onset depression, including the following: demographic factors
(e.g., age, gender, socioeconomic status); psychopathology (e.g., preexisting diagnosis, subsyndromal depressive symptoms, negative cognitive cognitive style); familial factors (e.g., parental psychopathology, early-onset mood disorders, high familial loading for mood disorders); and psychosocial factors (e.g., poor support, stressful life events, poor maternal functioning). These factors appear to influence differentially the onset and natural course of the disorder (e.g., Birmaher et al., 1996; Sanford et al., 1995; Reinherz et al., 1993).

ETIOLOGY

a) Genetic influences: Twin and adoption studies in adult populations have provided that the evidence that genetic factors account for at least 50% of the variance in the transmission of mood disorders (Birmaher et al., 1996). Genetic studies have also suggested the importance of the impact of environmental factors, particularly nonshared intra-and extrafamilial environmental experiences including differences in the ways in which individual parents treat each of their children (Kendler, 1995; Plomin, 1994).

Individuals at high genetic risk appear to be more sensitive to the effects of adverse environment than individuals at low genetic risk (Kendler, 1995). Furthermore, it has been suggested that environmental effects may be, at least in part, under genetic influence (Plomin, 1994). Rice et al. (2002) found that self-rated depressive symptoms were significantly genetically influenced. There was evidence of significant heterogeneity according to age, with shared environmental factors more important and genetic factors less important for children aged 8 to 10 than for adolescents aged 11 to 17 years. For self-rated depressive symptoms, the importance of genetic and environmental factors may also differ by sex, with genetic influences more important for boys (Birmaher et al., 1996).

b) Family aggregation studies:

"Top down" studies: The children of depressed parents are three times more likely to have a lifetime episode of MDD. The lifetime risk for MDD in children of depressed parents has been estimated to range from 15% (Orvaschel et al., 1988) to 45%
(Hammen et al., 1990a). Factors in the depressed parent such as early onset and recurrence appear to confer the highest risk for MDD in children (e.g., Warner et al., 1995; Mufson et al., 1992; Orvaschel, 1990). These studies, as well as twin studies, have suggested that what may be inherited is a vulnerability to depression and anxiety and that certain environmental stressors may be required for the manifestation of one of these disorders (Kendler, 1995; Warner et al., 1995; Plomin, 1994; Brown and Harris, 1993).

"Bottom-Up" studies: Age-adjusted lifetime prevalence rates of depression in the first-degree relatives of depressed children and adolescents have been estimated to range from 20% to 46% (Williamson et al., 1995c; Todd et al., 1993; Kutcher and Marton, 1991). However, there is an inverse relationship between age of onset and the density of familial aggregation of depression (Birmaher et al., 1996).

c) Growth Hormone studies: Like adults, depressed children have been found to hyposcrete growth hormone (GH) after various pharmacological challenges including insulin-induced hypoglycemia, oral clonidine, L-dopa, desmethylimipramine, and growth hormone-releasing hormone (e.g., Ryan et al., 1994; Jensen and Garfinkel, 1990) and this finding may be a “trait” or “scar” marker for MDD (Ryan et al., 1994). The dysregulation in GH secretion in depression may reflect changes in the central noradrenergic receptors, but also it may be secondary to changes in other neurotransmitters, somatedins, and somatostatin, which have been reported to be altered in some depressed patients (Ryan et al., 1994).

Findings as to the control of nocturnal secretion of GH without stimulation are conflicting. Earlier studies of children (e.g., Puig-Antich et al., 1984) and adolescents (Dahl et al., 1992b; Kutcher et al., 1991) suggested that there may be a relative hypersecretion during sleep, while a recent study (De Bellis et al., 1996) has failed to replicate this finding. A reexamination of nocturnal GH secretion in depressed children (e.g., Puig-Antich et al., 1984) suggested that stressful life events may contribute to elevated nocturnal GH secretion in this population (Williamson et al., in press).
d) **Neuroanatomy:** The areas of the brain appear to be most important for mood recognition and control are the prefrontal cortex and related areas of the striatum, and the thalamus, and the amygdale. The most consistent findings in depressed adults have been decreased frontal and prefrontal cortical volume and increased metabolic activity in the ventral medial prefrontal cortex and the anterior cingulated. There have been few studies of brain structures and functioning in depressed children and adolescents, but the few reports available are broadly compatible with those indicated previously (Shaffer, 2005).

e) **Serotonergic Studies:** Several biological investigations in adults with MDD have suggested that dysregulation of the central serotonergic function may be a vulnerability factor for the development of depression (Maes and Meltzer, 1995). To date, only one child study has shown that children with early-onset MDD have significantly lower cortisol levels than normal children after infusion of L-5-hydroxytryptophan (Ryan et al., 1992a). In addition, depressed female children appear to secrete significantly more prolactin compared with normal females and depressed and normal males (Birmaher et al., 1996; Ryan et al., 1992a).

f) **Hypothalamic-Pituitary-Adrenal Axis:** Evidence of hypothalamic-pituitary-adrenal axis dysregulation has been observed infrequently and inconsistently in studies of depressed children and adolescents, compared with studies of depressed adults. Investigations of baseline plasma cortisol secretion (24-hour or nocturnal sampling) have not found significant differences between depressed outpatients and normal control children and adolescents (Birmaher et al., 1992a, b; Dahl et al., 1991b; Kutcher et al., 1991; Puig-Antich et al., 1989a).

There have been numerous studies of the dexamethasone suppression test in depressed children (10 studies in inpatient settings and 4 outpatient studies) and depressed adolescents (11 inpatient samples and 2 outpatient studies) (Dahl et al., 1992a). The results indicated that the sensitivity was somewhat better in depressed children than in depressed adolescents (58% vs 44%) and the specificity compared to other psychiatric controls for child inpatients was approximately 60%, whereas for adolescent inpatients it was approximately 85%. A recent study found no significant
differences between prepubertal children with MDD and normal controls in baseline or post CRH stimulation values of either cortisol or ACTH (Birmaher et al., 1996). Abnormalities in ACTH secretion in response to CRH have also been observed in abused children, with the nature of the ACTH disturbances affected by both past history of abuse and current stressors (De Bellis et al., 1994; Kaufman et al., 1995, 1993).

g) Molecular Genetic Studies: The serotonin transporter gene (5HTT) plays a key role in the availability of serotonin. Polymorphism commonly affects this gene, so that only one-third of the population is homozygous for a (full) long allele (approximately 50 percent) or two short alleles (17 percent). An elegant longitudinal study of more than 1000 New Zealand children, during which data on external stresses were collected, has shown that early stress is strongly associated with later depression, but only in individuals who have one or two short alleles of the gene. The short form of the allele is known to be associated with less efficient transcriptions, and hence, the availability of the transporter gene is a good example of a vulnerability marker (Shaffer, 2005).

h) Sleep Studies: Despite frequent subjective complaints about disturbed sleep among depressed children and adolescents (Ryan et al., 1987), objective EEG studies have not found consistent sleep changes paralleling adult MDD studies. Among studies of depressed adolescents (Emslie et al., 1994; Kutcher et al., 1992; Dahl et al., 1990, 1996), five reported prolonged sleep latency, four reduced REM latency, and three decreased sleep efficiency in the MDD subjects. To date, no studies have found any differences in delta sleep.

Greater rates of sleep changes have been observed in inpatient adolescent samples and also in association with psychosis, suicidality, and endogenous MDD subtypes (Emslie et al., 1994; Kutcher et al., 1992; Dahl et al., 1990). A recent study showed that depressed adolescents without any stressful life events had significantly lower REM latencies compared with normal controls without stressful life events, suggesting the need to incorporate measures of environmental stress in EEG studies (Birmaher et al., 1996; Williamson et al., 1995b).
It is evident that depression is a serious psychological disturbance that is experienced by a very large number of children and adolescents. Surveys and studies of service utilization suggest that most of these youngsters do not receive treatment of any type (Keller et al., 1991). The high prevalence rate combined with the potential range of long-term negative consequences (Kovacs, 1996b) supports the need and requirement for effective treatment procedures. Treatment approaches for depression in children and adolescents are, for the most part, modifications or direct applications of procedures developed for use with adults. A significant finding from the majority of studies was the relative treatment efficacy of cognitive-behavioral therapies for the treatment of depression, with promising results reported for interpersonal psychotherapy. This suggests that brief, structured contemporary psychotherapies are potentially efficacious for the treatment of depression in children and adolescents. Given what is known regarding the clinical efficacy of pharmacotherapies, contemporary psychotherapies should be considered as a general first line of treatment, unless clinical data shows strong support for antidepressant treatment.