

## Article 2

# **Major Depressive Disorder in Children and Adolescents: Etiologies, Course, Gender Differences, and Treatments**

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## **Abstract**

Within the last decade, research has supported the notion that Major Depressive Disorder (MDD) can occur in children and adolescents as well as in adults. This paper elaborates on the trends of the current literature concerning the etiologies, course, gender differences, and recommended treatment approaches for early onset MDD.

Can children and young adolescents be clinically depressed? This is a question that mental health professionals and researchers have answered very differently within the past few decades. In fact, although the older research states that children cannot be clinically depressed, the literature within the last 10 years maintains that youngsters

actually can experience and be diagnosed with Major Depressive Disorder (MDD; Costello, Erkanli & Angold, 2006).

MDD is a mood disorder characterized by “a clinical course of one or more Major Depressive Episodes without a history of Manic, Mixed, or Hypomanic Episodes” (*Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., text rev.; *DSM-IV-TR*; American Psychiatric Association, 2000, p. 369). These multiple episodes consist of a persistent change in mood with manifested symptoms of depressed or irritable moods, loss of interest and pleasure in once-loved activities, and other additional behaviors (4th ed., text rev.; *DSM-IV-TR*; American Psychiatric Association, 2000).

These are some of the characteristics that make up the diagnostic criteria for MDD, but recognizing the disorder in children and adolescents is different from doing so in adults. This discrepancy is due to the numerous differences in the developmental stages and makeup between child and adult populations. Due to the significant change in perspectives within the recent years concerning early onset MDD (Costello et al., 2006), it is important to understand and recognize the symptoms of MDD in youth so that these children will be able to receive appropriate treatment. It is also vital to understand the etiology and course of MDD in children and adolescents while still taking into account the effects that gender has on the manifestation of the disorder. Further, along with all of these aspects considered, it is imperative to know what the most recommended and supported treatments are for this population.

Although professionals typically use the same diagnostic criteria for MDD in children as they do for adults, it is more complicated when trying to assess whether or not a child has this disorder since youngsters may not be able to fully verbalize and explain their emotions and symptoms. As stated in the *DSM-IV-TR* (2000), a Major Depressive Episode in a child or adolescent may manifest itself through irritability rather than the typical sad mood experienced by adults. Children may also not be able to verbally express their feelings of depression and only behaviorally exhibit their depression (e.g., tantrums, frustration, somatic issues).

The somatic complaints that depressed children oftentimes have include stomachaches, headaches, various pains, etc. (Emslie & Mayes, 1999), and they may also suffer from low self-esteem, feelings of guilt, or persistent boredom; may speak of or have efforts to run away from home; may have decreased interest in previously favorite activities; and may exhibit frequent sadness, tearfulness, or crying (American Academy of Child and Adolescent Psychiatry, 2010). There can also be some atypical symptoms that appear, such as “increased reactivity to rejection, lethargy, increased appetite, craving for carbohydrates, and hypersomnia” (Cheng & Myers, 2010, p. 178). Overall, however, the recent literature consistently maintains the importance of considering irritability as a symptom of childhood MDD (Birmaher et al., 2007; Fava et al., 2010).

Some of the symptoms of depression in adolescents are similar to those seen in younger children, but there is usually more emphasis on interpersonal conflicts in the former age group. For instance, Meichenbaum (2010) states that some symptoms could be loss of friendships, trouble or breakup of relationships, not getting along with family members, and higher than normal levels of expressed emotions. In some cases, adolescents can show symptoms of a physical illness that may be a sign of clinical depression. Any of these situations can be both a cause and a result of depression

(Meichenbaum, 2010). As in children, a major symptom of MDD that is typically seen in adolescents is irritability (Fava et al., 2010).

The assessment for pediatric depression can be difficult, as it is not always clear-cut. As Cheng and Myers (2010) explain, the presentation of depression in this age group can vary depending on the child's unique development, experiences, and physical health. Clinicians must also ensure that symptoms such as fatigue, difficulty sleeping, or somatic complaints are not due to an underlying medical condition. Differential diagnoses must be kept in mind as well, as depression is oftentimes co-morbid with other behavioral disorders that require treatment. Concerning differential diagnoses, it is common for youth with eating disorders or anxiety to initially present with depressive symptoms (Cheng & Myers, 2010).

### **Etiology**

The etiology of MDD in children and adolescents is not straightforward either, as it is not attributed to or explained by a single, distinct factor. Rather, the disorder is usually brought about by the complex interaction of many factors. These etiologies include neurological, genetic, environmental, psychosocial, and psychological aspects of the child (Abela & Hankin, 2008). These realms interact in intricate and unique ways to produce the possible outcome of MDD.

Within the neurological domain, Goodyer (2008) explains that the roles of three neuroactive chemical components are particularly influential in the etiology of depressive disorders like MDD in youngsters. These components are serotonin, brain-derived neurotrophic factor (BDNF), and cortisol. Abnormal levels of these factors can lead to depressive symptoms within the first few years of life, especially as the neurological chemicals relate to the amygdala and ventral prefrontal cortex. This, in turn, can alter the regulation of mood and emotion in children and adolescents, thereby leading to depressive symptoms and disorders such as MDD. Goodyer goes on to explain that later disruptions in neural development, termed acquired neuroendangerment, can also lead to similar outcomes. This particular occurrence is "a pathological brain process leading to reduced synaptic plasticity, in particular in the hippocampus and perhaps the nucleus accumbens and ventral tegmentum" (Goodyer, 2008, p. 1239). At any point in life, especially within the presence of environmental stressors, deficits within the motivational, cognitive, and behavioral realms may result from this neural disruption.

Also within the neurological realm, recent research has found linkages between 5-*HTTLPR* (a serotonin transporter genotype) and MDD during adolescence. Specifically, differences in biochemistry and behavior have been observed in individuals with varying 5-*HTTLPR* genotypes. This discovery suggests that 5-*HTTLPR* "may be partially responsible for differential biological stress reactivity and that behavioral differences between those carrying the S versus L allele may be most prominent in stressful situations" (Petersen et al., 2012, p. 2). Stress and anxiety have been consistently shown to be predictors of subsequent depression, and since individuals react to stress differently, it is more likely that individuals with 5-*HTTLPR*-S alleles would be more prone than L-allele carriers to experience anxiety and develop depression following stress. These "findings demonstrate that the 5-*HTTLPR* genotype affects likelihood of experiencing anxious/depressed symptoms in situations of stress, particularly during late adolescence" (Petersen et al., 2012, pp. 13-14). If future findings support this 5-*HTTLPR* Gene  $\times$  Stress

interaction, more effective assessment tools could possibly be developed to identify and target at-risk individuals for treatment (Petersen et al., 2012).

Another neurological influence for depression in youth concerns the hypothalamic-pituitary-adrenal (HPA) axis, which triggers an increase in cortisol levels when a stressful or threatening event occurs (Guerry & Hastings, 2011). Specifically, Guerry and Hastings (2011) note that there is abundant evidence showing that dysregulation of this axis is a large contributor to MDD in adults. These researchers discuss multiple studies and conclude that there is evidence that the HPA axis plays a significant role in depressive disorders of children and adolescents as well. These influences from the HPA axis come into play through various means, including genetic inheritances (especially maternal depression) and cortisol-releasing responses to psychological stressors. Despite these possible evidences, Guerry and Hastings (2001) report there is a large need for more longitudinal research to determine whether developmental changes in HPA functioning could account in part for the dramatic increases in the rates of depression from childhood to adolescence. They further note that future research needs to determine if dysregulation of the HPA axis is a correlate, cause, or consequence of childhood MDD.

Genetic influences also play vital roles in the etiology of MDD in children and adolescents. Schreier, Höfler, Wittchen, and Lieb (2006) discuss several studies that have consistently demonstrated a familial pattern of MDD, and they also state that various twin studies have supported this notion of genetic influence. However, they add that the environmental factors within the family must also be taken into account when considering the role of genetics in childhood MDD. Similarly, in studying adolescent twin pairs, Ehringer, Rhee, Young, Corley, and Hewitt (2006) found that there was support for genetic, non-shared environmental influences and shared environmental influences for MDD. In view of these findings, the interaction between genetics and environment in the etiology of childhood MDD is apparent and vital to take into consideration.

As stated earlier regarding the influence of stress on later depression, research highly attributes the environmental and psychosocial influences of early-onset MDD to the child being reared in a stressful family environment or to the child experiencing multiple stressful life events, particularly when the mother suffers from depression herself (Wachs, Black, & Engle, 2009). Luby, Belden, and Spitznagel (2006) define and give examples of what such *stressful life events* can include. The events can be either mild/common or severe, the former including occurrences such as the birth of a new sibling or peer rejection, and the latter including experiences such as the death of a parent or a family history of mood disorders or suicide. The overall consensus among recent literature is that living in a stressful environment and/or experiencing many stressful life events is a major precursor to childhood and adolescent MDD (Luby, Belden, & Spitznagel, 2006; Wachs, Black, & Engle, 2009; Williamson, Birmaher, Dahl, & Ryan, 2005).

Regarding parental psychopathology, approximately 15 million children in the United States are reared in a household containing a parent who has experienced one or more episodes of a major depressive disorder (England & Sim, 2009). These children are at an increased risk of developing various disorders and symptoms themselves (England & Sim, 2009; Goodman et al., 2011). According to recent research, parenting is one of

the primary ways that the risk for psychopathology is transmitted in these families (Compas, Keller, & Forehand, 2011; Goodman & Gotlib, 1999; Goodman & Tully, 2006). Fortunately, it has also been found that changes made to the parenting habits of parents with a history of depression may result in changes in child emotional and behavioral problems (Compas et al., 2010).

The last of the intertwined etiological factors is the psychological makeup of the child or adolescent. One part of this realm is the child's cognitive representation of him/herself and of others (Hammen & Rudolph, 1996). For example, as Goodyer (2008) explains, the child may have negative cognitive schemas about the self, which are usually latent. These negative self-statements and beliefs, such as *I am no good, other people know I am no good*, etc., may develop from having negative social experiences, and they directly affect the child's mood after they enter consciousness. This destructive view of the self can also develop from the child's witnessing of their mother's own negative self-talk. Indeed, Wachs et al. (2009) state that maternal negativity and depression can put the child at risk for slower cognitive development, insecure attachment, learned negative cognitive schemas, and low activity—all of which put the child at the risk of developing MDD at an early age.

### **Course and Outcome**

The course and outcome of MDD in children and adolescents has also been discussed in the recent research. The adolescence era is an ideal period to investigate because many adolescents experience anxiety and depressive symptoms (Petersen et al., 2012). Regarding the course, Meichenbaum (2010) reported that the average age of onset of MDD is around 13 years old and that one third diagnosed with MDD (by 16 years old) have had previous episodes of depression. When MDD arises in childhood, usually the episode lasts an average of 7 to 11 months. Although 80-90% of youth will recover from this initial episode within 11 months, Meichenbaum (2010) stated that the remaining could have ongoing depression that can last longer than 18 months. Of those children with MDD, 40% will have remission within two years, and 70% within five years. Early recognition and treatment of childhood MDD is vital to aid in the reduction of future depressive episodes. Ultimately, about 10% of children will have extended depression that persists into adulthood. Children have a much higher chance of their depression lasting into adulthood when the onset is earlier in life. Further, the earlier the onset of MDD, the more likely it is for the child to have deficits in social and psychosocial areas of life (Meichenbaum, 2010).

Recent research on the rise in depression during middle to late adolescence has found a stronger Gene  $\times$  Environment ( $G \times E$ ) interaction effect in later adolescence, particularly at ages 16 and 17 (Petersen et al., 2012). This interaction may support the late-maturing prefrontal cortex theory of adolescent depression (Andersen & Teicher, 2008). According to this theory, the complex and continual development of the prefrontal cortex (PFC) may cause adolescents to be especially vulnerable to the effects of stress. One of the roles of the PFC is to modulate activity of limbic areas, including the amygdala, which is involved in processing emotion and fear. Due to the prefrontal cortex's significant role on the limbic system and emotions, researchers have suggested that adolescent depression may result from the developmental lag between the PFC and the earlier developing limbic areas (Davey, Yücel, & Allen, 2008). Concerning serotonin,

adolescents with lower (as opposed to higher) amounts may be more likely to develop depression or anxiety after a stressful incident due to the lower serotonin and lagging PFC (Friedel et al., 2009).

Fergusson and Woodward (2002) give some examples of the psychosocial problems that children and adolescents with early-onset MDD may experience later in life. These could include the later development of anxiety disorders, nicotine dependence, alcohol abuse or dependence, suicide attempts, educational underachievement, unemployment, and/or early parenthood. In this study, there was also a direct linkage found between early-onset depression and a later increased risk of MDD and anxiety disorders. Further, 30-70% of children who experience a major depressive episode will have one or more episodes during their childhood, adolescence, and adulthood. Those children particularly at risk for experiencing additional major depressive episodes are those with a “familial history of MDD, co-morbid psychiatric disorders, dysthymia, subsyndromal symptoms of depression, anxiety, negative cognitive style, and exposure to negative life events (e.g., family conflicts and abuse),” (Birmaher, Arbelaez, & Brent, 2002, p. 619).

Gordon, Tonge, and Melvin (2011) tested a number of psychological instruments to determine which ones were reliable in predicting the course of depressed adolescents’ mental health six months after treatment. They found that one of the more reliable self-report measures was The Self-Efficacy Questionnaire for Depressed Adolescents (SEQ-DA), which is a 12-item self-report scale questionnaire that rates adolescents’ perceived ability to cope with depressive symptoms (Tonge et al., 2005). Higher scores mean that the adolescent perceives him or herself as having a more adaptive way of coping with daily activities and feelings. Gordon et al. (2011) conclude that these findings signify the importance of depressed adolescents developing a sense of self-efficacy.

There are some differences between males and females in the manifestation of MDD, but the research on those differences in child and adolescents is limited (Weller, Kloos, Kang, & Weller, 2006). However, there are well-known gender differences in the prevalence rates of depression that emerge around adolescence (Petersen et al., 2012). Specifically, males and females experience equal rates of major depression until after puberty. After puberty, both sexes experience an increase in depressive symptoms, with the rate of depression in females doubling that of males. In fact, females are approximately two times more likely than males to develop depression over the lifetime (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). The general increase in depression at puberty could partly be due to typical adolescent stressors such as hormonal changes, identity crises, emerging sexuality, and stressors over academic and vocational choices. Adolescent females experience additional unique stressors such as menarche, breast development, and concern about body image. Future research should determine if these stressors are the cause of the higher depression rates in young females when they reach puberty (Weller et al., 2006).

Some gender variations in depression found in adults may apply to children and adolescents as well. For instance, adolescent females have some similarities to depressed adult females such as the occurrence of anxiety and eating disorders (Weller et al., 2006). Weller et al. (2006) also state that adolescent males typically have lower levels of self-reported depressive symptoms, and they oftentimes exhibit externalizing behaviors such as disruptive behavioral disorders and eventually substance abuse disorders. Further

related differences of depression between males and females adhere to gender-related stereotypes. For example, Weller et al. (2006) cite one study (Winkler, Pjrek, & Heiden, 2004) that found women cried more than men and men exhibited affective rigidity more than women.

### **Treatment Options**

There are a variety of treatment options available for children and adolescents diagnosed with MDD. When considering this range of opportunities, the particular treatment chosen is dependent on the child; there is not only one therapy or treatment that is best for all children. Individualized treatment incorporating the client's strengths and resources can be the most beneficial option (Emslie & Mayes, 1999). In all children and adolescents, treatment should seek to include interventions in various environments of the child's life, including both home and school, for example (Birmaher et al., 2007). Pharmacotherapy, psychotherapy, family therapy, and school interventions should also be utilized, and clinicians should consider employing both acute and long-term treatment approaches (Yu, Mooreville, Weller, & Weller, 2011). The treatments that are most often used and that have been repeatedly empirically supported are now discussed.

Without considering medications, the treatment method most often used and recommended today is cognitive-behavioral therapy (CBT). If medications are considered, however, it has repeatedly been shown that the combination of selective serotonin reuptake inhibitors with CBT accelerates the improvement of depression as compared to receiving just CBT alone (The TADS Team, 2007). One particular option is called the ACTION treatment program, which is based on a CBT model. Originally designed for only female clients, this treatment approach has recently also been designed for males (Meichenbaum, 2010). Allen (2008) describes this program as a group intervention that aims at individual needs through the usage of CBT concepts. Clients deemed appropriate for the ACTION model are between the ages of 9 to 13 years, and they meet in small groups for a total of 20 times in 11 weeks. A particularly impressive aspect about this treatment model is that parents are also included in the intervention. Specifically, the clients apply what they learn in their small groups about their distorted beliefs to their home life with their parents. This real-life application along with the implementation of skills monitoring, homework, and reward systems yields efficacious results for the clients involved in this treatment approach (Allen, 2008).

Yu et al. (2011) discuss the usage and outcomes of CBT approaches for both acute and long-term treatments of pediatric depression. These authors note that CBT is effective in the short-term, but that when it comes to long-term maintenance, those children and adolescents with severe depression are at increased risk of recurrences and chronic depression. However, Yu et al. (2011) also report that studies are limited on long-term treatment of MDD in children and adolescents, as the majority of studies to-date concern adult populations. Results with youth thus far have been inconclusive, as some studies have found benefits of CBT continuation treatment (e.g., Knoll et al., 1996), while others have found that the "combination of fluoxetine and CBT [is] not better than fluoxetine alone after 28 weeks of treatment in adolescents with moderate to severe depression" (Yu et al., 2011, p. 120). Hence, more studies are needed on the efficacy of long-term treatment of depression in children and adolescents.

Outside of the CBT realm are some other effective therapies for children and adolescents with MDD, such as interpersonal therapy (IPT). Interestingly, IPT for adolescent depression has been shown to be as effective as CBT (Birmaher et al., 2007). One version of this treatment approach is called interpersonal psychotherapy for adolescents, or IPT-A. Meichenbaum (2010) explains that IPT-A is offered in school-based health clinics and that it can be delivered on either a group or individual basis. Clients should be from 12-18 years of age, and the approach is 12 weeks long, covering a total of 12-15 sessions. The problem areas that this treatment modality focuses on are interpersonal role disputes, role transitions, interpersonal deficits, and grief (Meichenbaum, 2010). CBT and IPT have both been widely researched and supported, and they represent the strongest evidence-based therapeutic approaches to treating children and adolescents with depression. As with any therapeutic interventions with youth, and as the ACTION model does, it is vital for parents and family members to be involved in the treatment process as well. Additional treatment approaches also shown to be effective include group and family therapy (Cheng & Myers, 2010).

There are also many psychotherapeutic activities that can be used with younger clients who are not of adolescent age. For example, Asarnow et al. (2005) used a *hot seat* game based on CBT principles. When one group member is in the *hot seat*, the rest of the members call out negative thoughts to him/her, and the one in the *hot seat* immediately responds with a more positive thought. Another group member can act as a *coach* for the child in the *hot seat*. Cartoon-like characters can be used with even younger children. Some additional games and therapeutic strategies could include a Feelings Thermometer or Watch, role-playing, or the creation of a movie that features the children enacting the coping skills they have learned. These movies can be shown to the children's parents so that they can also be a part of the therapeutic process by helping their children use the coping skills at home (Asarnow, Scott, & Mintz, 2002).

Although the effectiveness of CBT in treating early-onset MDD has consistently been supported, some studies have found limitations. For example, Birmaher et al. (2007) state that CBT does not appear to perform as well when there is a history of sexual abuse or when one of the child/adolescent's parents is depressed. In order to maintain the beneficial effects, the child or adolescent should continue to receive therapy for 6 to 12 months. The authors elaborate by explaining that a longer period of maintenance is required for patients who take longer to recover or who have a higher number of depressive recurrences. Hence, how long a child receives treatment depends largely on their particular circumstance and number of depression recurrences. Other things that may cause prolonged episodes of depression and that therefore may require continuous treatment include external factors such as family dysfunction and divorce, family psychopathology, psychosis, suicidality, and lack of social support (Birmaher et al., 2007).

## **Conclusion**

In summary, although MDD is a disorder with its own set of diagnostic criteria, it gets much more complex when trying to diagnose a child or adolescent. Due to the developmental differences between children, adolescents, and adults, the symptoms of



MDD are exhibited differently amongst all of these populations. Nevertheless, research within the past decade has consistently supported the notion that MDD can and does exist in youngsters and that it can be recognized through a set of common manifestations. The etiology of MDD in the younger population is multifaceted in the biological, psychological, and environmental areas, with each causative realm affecting the others in an intricate and unique fashion for each individual. Further research should be done on the gender differences of MDD in children and adolescents, but some explanations have already been put forth in the recent literature. Although the earlier the onset of MDD, the more likely the individual is to suffer from additional psychological and psychosocial deficits in adulthood, there are a variety of effective treatment options available.

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