
### Treating Adolescent Depression: Providing a Sense of Hope

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“Good morning, Eeyore”, said Pooh.
“Good morning, Pooh Bear”, said Eeyore gloomily. “If it is a good morning, which I doubt”, said he.
“Why, what’s the matter?”
“Nothing, Pooh Bear, nothing. We can’t all, and some of us don’t. That’s all there is to it.”
“Can’t all what?” said Pooh, rubbing his nose.
“Gaiety. Song-and-dance. Here we go round the mulberry bush”.

*A. A. Milne*

*From the book Winnie the Pooh*

“I don’t want my whole teenage-hood to go on like this.”

*Client S*

### Overview of Adolescent Depression

Adolescence is a time of much change. Previously, adolescence was seen as a time of uncertainty and “angst”, which precluded the investigation of depressive disorders during this developmental period (Rudolph, Hammen, & Daley, 2007). The research focus had been on depression in childhood and to a much greater extent, adulthood. There has recently been a shift to recognising depression as a genuine occurrence of adolescence and consequently, an increase in effective treatments for this period of life (Rudolph et al., 2007). Nevertheless, many models and treatment of childhood and adolescent depression have been adapted from adult models and treatments (David-Ferdon & Kaslow, 2008; Rice & McLaughlin, 2001). The present review will give an overview of the epidemiology of adolescent depression, specifically Major Depressive Disorder (MDD), and then outline current models and treatments available. The efficacy and effectiveness of these treatments will then be discussed.

### Definitions and diagnosis

Adolescent depression, also known as early-onset depression (Fletcher, 2008) refers to depression as it occurs in the teenage years. Adolescent depression can have a serious impact on a youth’s life and functioning, including poorer health and growth outcomes and less meaningful relationships (Bhatia & Bhatia, 2007; Saluja et al., 2004). An issue that arises when reviewing the research on adolescent depression is the variation in ages of individuals included in studies. For the purposes of the present review the following terms will be used to refer to the different ages of adolescence except where specified otherwise: early adolescence (for those aged 12 to 14 years), middle adolescence (15 to 17 years), and older adolescence (18 to 21 years).

Depression as a syndrome occurs when several depressive symptoms combine and persist for a long time. Table 1 shows the first criterion for a Major Depressive Episode (MDE) and the differences that are seen in children and adolescents. For a diagnosis of MDD, two or more separate MDE must have been present (as described in the *Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, text revision (DSM-IV-TR)*, American Psychological Association, 2000). To diagnose adolescent depression one must first rule out depression due to a medical cause, bereavement, or physiological effects of a...
After these causes are excluded, there are several depressive diagnoses that can be considered. For the purposes of the present review, only Major Depressive Disorder (MDD) will be discussed. For a full description of other diagnoses see the *DSM-IV-TR*.

Table 1. The First Criterion of a DSM-IV-TR Diagnosis for a Major Depressive Episode and Differences in Symptomatology for Children, Adolescents, and Adults.

<table>
<thead>
<tr>
<th>Criterion A: Five (or more) of the following symptoms have been present during the same two–week period and represent a change from previous functioning; at least one the symptoms is (1) depressed mood or (2) loss of interest or pleasure.</th>
<th>In children and adolescents</th>
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<tr>
<td>Symptoms in adults</td>
<td>Mood can be irritable or depressed. Children with less developed cognitive abilities may not be able to describe inner mood states and therefore may present with vague physical complaints, sad facial expression or poor eye contact. Irritable mood can appear like ‘acting out’. Older adolescents may show disturbances similar to adults.</td>
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<td>Depressed mood most of the day, nearly every day, as indicated by subjective reports or observation made by others.</td>
<td>Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day.</td>
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<td>Significant weight loss when not dieting, or weight gain, decrease or increase in appetite nearly every day.</td>
<td>Loss of interest can be in peer play or school activities.</td>
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<td>Insomnia or hypersomnia nearly every day.</td>
<td>Children may fail to make expected weight gain rather than losing weight.</td>
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<td>Psychomotor agitation or retardation nearly every day.</td>
<td>Similar to adults.</td>
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<td>Fatigue or loss of energy nearly every day.</td>
<td>Concomitant with mood change, hyperactive behaviour may be observed.</td>
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<td>Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day.</td>
<td>Disengagement from peer play, school refusal, or frequent school absences may be symptoms of fatigue.</td>
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<tr>
<td>Diminished ability to think or concentrate, or indecisiveness, nearly every day.</td>
<td>Child may present with self-deprecation (e.g., “I’m stupid”, “I’m a retard”). Delusional guilt not usually present.</td>
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<tr>
<td>Recurrent thought of death, recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.</td>
<td>Problems with attention and concentration may be apparent as behavioural difficulties or poor performance in school.</td>
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Adapted from “Childhood and Adolescent Depression” by S. K. Bhatia and S. C. Bhatia, 2007, Depression and Anxiety, 100, p. 76.

The *DSM-IV-TR* criteria for depressive disorders do not distinguish greatly between childhood, adolescence, and adulthood. There are no specific criteria given for adolescent depression, however there are possible differences given for children (see Table 1 above). The main difference for diagnosis is that children tend to display an irritable mood rather than a depressed mood and may present with more physical symptoms (Bhatia & Bhatia, 2007). A young adolescent may display signs more similar to a child’s presentation of the disorder. However, there have been differences found in depressive symptomatology across different ages (Cantwell & Baker, 1991). These differences in symptomatology highlight the importance of applying a developmental psychopathology perspective (Rudolph et al., 2007). Treatment will also differ depending on the age of the adolescent and the individual’s mental abilities (Lewinsohn & Clarke, 1999). For example, some adolescents will be able to describe their mental states accurately and others may not (American Family Physician, 2010).

Adolescent depressive symptoms have been found to be similar to adult depressive symptoms in terms of the effects of recent events on depression; however, differences have also been found (Lewinsohn & Clarke, 1999). The most commonly found
difference is that adolescents are more likely to have a comorbid disorder (see comorbidity section), sleep disturbances, and have a higher degree of suicide attempts (Cantwell & Baker, 1991).

**Epidemiology of adolescent depression**

*Incidence and prevalence*

Lifetime prevalence rates of adolescent depression have been found to vary, in part due to the different age ranges included in research. It is thought that some depressed adolescents go unnoticed due to stereotypes about the changes that occur in that period of life (Rudolph et al., 2007). Lifetime prevalence has been estimated at 15%-20% (Rice & McLaughlin, 2001).

*Age of onset and risk factors*

It is thought that depressive disorders that start in adolescence are more difficult to treat than adult-onset mood disorders (Mondimore, 2002) and that early onset depression may reflect a more severe form of the disorder (Rice & McLaughlin, 2001). An episode of depression in adolescence increases the chances of the individual having a recurring episode later in life (Rudolph et al., 2007). In adolescents, stressors such as conflict with parents, breaking up with a partner, or death of a loved one can increase chances of depression (Bhatia & Bhatia, 2007). In particular, adolescents with low self-esteem are at risk of becoming depressed after stressful events (University of Maryland Medical Center, 2010). Having a family history of depression, being female, childhood abuse, neglect, stressful life events, and chronic illness are also risk factors (Bhatia & Bhatia).

In a study of over 2000 early adolescents (males and females), MacPhee and Andrews (2006) investigated risk factors to determine which were more likely to lead to depression in later years. They found that peer relationships, perceived parental rearing behaviour, self-esteem, hyperactivity/inattention, and conduct problems significantly contributed to adolescents’ depression. Self-esteem was the greatest predictor for both males and females. It is thought that body image may be a risk factor for females as they advance into middle and late adolescence (MacPhee & Andrews). Another interesting finding from this study was that parental rearing behaviour was linked to the development of low self-esteem.

*Comorbidity*

Comorbidity with other psychiatric disorders is more common in adolescents than adults (Lewinsohn, Clarke, Rohde, Hops, & Seeley, 1996). Results from a two-wave epidemiological study for 1993 indicated that the disorders most commonly comorbid with depression are the anxiety disorders (Lewinsohn et al., 1996). Other researchers have found similar results, with estimates of 21% of those with a MDE also suffering from a lifetime anxiety disorder (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993). The main concern about comorbid disorders is that when they are present, the prognosis for the adolescent is generally worse and depressive symptoms can often be more severe.

*Sex differences*

Depression in childhood affects males and females equally, but in adolescence the sex ratio begins to resemble that of depression in adulthood. Adult females are twice as likely to have depression than males (Lewinsohn & Clarke, 1999; Nolen-Hoeksema, Girgus, & Seligman, 1991). It is thought that the rate of depression in adolescent girls becomes significantly higher than that for in boys at about 12 to 14 years. One reason for the discrepancy has been thought to be that the challenges of adolescence, such as puberty and school changes, have a greater impact on girls than boys in early adolescence (Petersen, Sarigiani, & Kennedy, 1991).
Literature review: Models of adolescent depression and treatment outcome literature

Genetic and Biological Models
Genetic models postulate that depression is transmitted genetically; therefore people who are closely related to someone with MDD are more likely to become depressed (Rice & McLaughlin, 2001). Biological models hypothesise that changes that occur during adolescence (i.e., puberty) can increase the chances of an adolescent developing depression (Rudolph et al., 2007). It is thought that changes in chemicals in the brain, specifically regulation of the neurotransmitters norepinephrine and serotonin, result in an imbalance that underlies the behaviour changes seen in depressed adolescents (Rice & McLaughlin).

Biological models, particularly the biochemical view, indicate treatment of depression with medication. The use of pharmacological treatments with adolescents is not usually the first line of treatment (British Medical Journal, 2010). For a more comprehensive review of pharmacological treatments of depression in adolescents see Bhatia and Bhatia (2007). To summarise the review briefly, it has been recommended that tricyclic antidepressants are not used with adolescents, and if selective serotonin reuptake inhibitors are administered to children or adolescents, that they are monitored carefully for increases in suicidal thoughts and behaviours (Bhatia & Bhatia).

Socioenvironmental Models
The main premise of socioenvironmental models involves investigation of environmental stressors and adolescents’ reaction to negative life events (Rudolph et al., 2007). There are three main types of stress models but due to the paucity of treatment outcome research on stress models they will also only be briefly described. Between 1998 and 2008 there were 18 studies on the treatment of adolescent depression and the majority focused on CBT or IPT techniques, with the exception of a pilot study that incorporated a stress model with several other models (David-Ferdon & Kaslow, 2008). Diathesis stress models of depression suggest that it is the interaction of an individual with his or her environment that results in depression. There are also stress-exposure models that focus on elucidating the etiology of depression (Rudolph et al., 2007). These models propose that early life stress (e.g., socioeconomic disadvantage, family structure) increases adolescents’ risk of depression. Stress-generation models propose that a self-perpetuating cycle can develop in which an individual generates stressful events (Rudolph et al.). Stress-generation models have been linked particularly to interpersonal relationships.

Interpersonal models
Interpersonal models focus on four main interpersonal problem areas that have been linked to the beginnings of depression (Mellin & Beamish, 2002). Similar to other models of adolescent depression, these problem areas are the same as those thought to be associated with adults’ depression, including interpersonal deficits, role transitions, interpersonal role disputes, and grief (Mellin & Beamish). It is hypothesised that if an adolescent is having difficulty in these areas, it can lead to behavioural and cognitive deficits in relation to problem solving and coping in social relationships (Mellin & Beamish).

Interpersonal psychotherapy (IPT) for treatment of adult depression has been modified and is available as interpersonal psychotherapy for adolescents (IPT-A). The modifications focus on the different developmental stages of adolescents and also take into consideration the different contexts adolescents move in. For example, a fifth area of single parent families has been included (Mellin & Beamish, 2002). (For a full description of each of these areas see Mellin & Beamish). The goals of IPT-A are to reduce depressive symptomatology and improve interpersonal functioning. There are three phases within treatment (early, middle, and termination) to achieve these
goals (Mufson, Moreau, & Weissman, 1996). The initial phase includes full assessment and introduction to the therapy and identification of problem areas that will be addressed (Mufson et al.). The middle phase associates interpersonal problems with the client’s depression and targets specific problem areas decided on by the client and therapist. The termination phase helps the adolescent think about future problem solving and involves relapse prevention, including the recognition of signs of depression.

In a randomised control trial (RCT) of IPT-A with 48 adolescents (age range 12 to 18 years), 32 participants completed treatment and showed significantly fewer depressive symptoms in comparison to the control group (clinical monitoring only, with a review of symptoms and supportive listening). Adolescents in the treatment group also showed improvement in social functioning overall and with peers and in dating relationships. However, there were no differences between the two groups at a 12-week follow-up (Mufson et al., 1996).

An effectiveness study has been conducted with IPT-A to assess how the therapy translates into school-based clinics, an important step in helping research move into practice. IPT-A was compared with treatment as usual (TAU) at the school-based clinics (TAU procedures included mostly supportive listening techniques). Adolescents in the IPT-A condition had fewer depressive symptoms and improved overall functioning when compared with adolescents in the TAU condition (Mufson et al., 1996). It was concluded that IPT-A was an effective treatment.

**Family Models**

Family models postulate that families transmit not only a genetic vulnerability but can also affect the development and maintenance of depression in adolescents (Larner, 2003; Rudolph et al., 2007). In addition, a negative family environment (e.g., unresolved conflict, neglect, maternal depression) has been found to have a significant impact on the course of depression (Larner). There are two types of family therapies that will be briefly discussed here: attachment-based family therapy and systemic family therapy. Attachment-based family therapy hypothesises that depression results from difficulty with attachment occurring in a context of family conflict (Larner, 2003). It has been found to be efficacious in reducing depressive symptoms in adolescents but is considered experimental because there has been a lack of research into the therapy (David-Ferdon & Kaslow, 2008).

Systemic family therapy brings together the family, professionals, and the adolescent (Larner, 2003). It has recently been found to be highly effective at reducing moderate to severe depression (Trowell et al., 2007). However, the lack of control group and small sample size in this study need to be considered when interpreting these results.

It has been proposed that family therapies need to be integrated alongside other treatment approaches for adolescent depression (Larner, 2003). However, the evidence base is still limited due to the relatively new nature of the therapies. Larner conducted a systematic review of literature on family therapies used with adolescents and found that while research findings were often mixed there was support for the efficacy of family therapies in reducing depressive symptoms.

**Cognitive models**

Overall, cognitive theories propose that an individual’s thought processes and belief systems contribute to depression (Rudolph et al., 2007). There are two main theorists who have developed cognitive models of depression in adults; these models have also been applied to adolescents. The first model is based on Beck’s well-known cognitive theory of depression, which focuses on helping people become aware of and modify dysfunctional thinking styles, alongside developing good problem solving skills.
(Beck, 1995, Lewinsohn et al., 1996). There are three main aspects of Beck’s cognitive model: automatic thoughts, intermediate beliefs, and core beliefs (Beck, 1995). Another important aspect of the cognitive model is that people with depression can have distorted cognitions, which are negative and irrational thinking styles.

Evidence for Beck’s cognitive model comes from research findings showing that depressed adolescents exhibit negative thinking styles similar to those of adults, for example, maladaptive attributional styles, cognitive distortions, and negative self-concept (Reinecke, Ryan, & DuBois, 1998). While these factors do not cause depression they can contribute to a depressive episode.

The second theorist is Seligman, who developed a theory of learned helplessness (Abramson, Seligman, & Teasdale, 1978). The premise of Seligman’s model is that depressed individuals tend to make internal, stable, and global attributions about negative events and make external and specific attributions about positive events, leading to feelings of helplessness. Seligman's theory can also be seen from a behavioural perspective. If an individual is in an environment with inescapable punishment they are more likely to become depressed (Rice & McLaughlin, 2001).

One review of research including studies of the learned helplessness model found that there was evidence of the importance of taking comorbid disorders into account (anxiety and conduct disorders) (Craighead, 1991). Craighead concluded that the attributional model hypothesised by Seligman appeared to account for mild to moderate depression and not severe.

When considering cognitive treatments it is important to consider the developmental and mental age of the adolescent because cognitive therapy may be beyond the understanding of some adolescents, especially younger adolescents. One hypothesis is that older adolescents have better developed cognitive skills and get increased benefit from CBT techniques as these techniques are often more effective with adolescents than children (Michael & Crowley, 2002).

**Behavioural Models**

Original behavioural models of depression came from behaviourists such as Skinner and are based on the premise that people interact with and respond to their environment (Rice & McLaughlin, 2001; Rudolph et al., 2007). Depression results from a lack of ability or skills to continue to obtain positive reinforcement from the environment. In simplified terms, a loss of activity and positive reinforcement can lead to depression, as a cycle is created in which the individual reduces their positive behaviours and no longer responds to positive interactions (Lewinsohn et al., 1996). The goals of behavioural therapy are to increase behaviours that provide positive reinforcement for the individual and reduce behaviours that provide negative reinforcement (Lewinsohn et al.). Behavioural activation is one therapy based on behavioural models but has not received much attention as a stand-alone therapy for adolescent depression. Research is still developing on behavioural activation as a pure therapy for adult depression and some issues in terms of strategies and principles have been identified as needing to be addressed (Hopko, Lejuez, Ruggiero, & Eifert, 2003).

While models of depression generally come from cognitive and behavioural theories, most treatment for adolescent depression is based on a combination of the two approaches. CBTs in the literature tend to include different elements based on differing presenting problems but there are several commonalities in basic organisation (Lewinsohn & Clarke, 1999). All sessions are structured, with an agenda provided for the client and a limited number of sessions (ranging from 8 to 16 sessions) (Lewinsohn & Clarke). However, one difficulty that arises in the literature on CBT for
adolescent depression is that studies do not often fully describe the specific techniques used. One meta-analysis in the late 1990s provided a description of the common CBT techniques found in the literature and these appear to be still relevant today. These techniques can be seen in Table 2 below.

Table 2. Treatment Intervention Types Described in Adolescent Depression Treatment Literature

<table>
<thead>
<tr>
<th>CBT techniques found in the treatment literature</th>
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<tr>
<td>Cognitive techniques</td>
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<tr>
<td>Constructive thinking (rational emotive therapy,</td>
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<td>cognitive therapy)</td>
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<tr>
<td>Positive self talk</td>
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<td>Being your own coach</td>
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<tr>
<td>Self-change skills (self-monitoring, goal setting, self-reinforcement)</td>
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<td>Family context (FAM)</td>
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<td>Conflict resolution</td>
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<td>Communication skills</td>
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<tr>
<td>Parenting skills</td>
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<tr>
<td>Behavioral (BEH)</td>
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<tr>
<td>Problem-solving skills</td>
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<tr>
<td>Increasing pleasant activities</td>
</tr>
<tr>
<td>Social skills (assertiveness, making friendships, role modeling)</td>
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<tr>
<td>Affective education and management (AEM)</td>
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<tr>
<td>Relaxation</td>
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<tr>
<td>Anger Management</td>
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Adapted from “Psychosocial Treatments for Adolescent Depression” by P. Lewinsohn and G. Clarke, 1999, Clinical Psychology Review, 19, p. 332.

One CBT approach based on the cognitive theories of Beck and behavioural theories of Lewinsohn was used in the Oregon Adolescent Depression Project (OADP) epidemiologic study. The approach is known as Coping with Depression for Adolescents (CWD-A) and has been modified from an adult form of the treatment. The main goal of the treatment is to teach adolescents new coping strategies and skills to deal with depressive symptoms and any problems that come up in their lives (Lewinsohn et al., 1996). There are several components to the CWD-A treatment approach which include the following techniques: increasing social skills to provide a base for other skills such as communication, increasing pleasant activities to encourage positive reinforcement, decreasing anxiety with relaxation training, reducing depressogenic cognitions, resolving conflict with negotiations, and planning for the future (Lewinsohn et al.).

Research on the efficacy of CWD-A has been conducted in group settings with 16 two-hour sessions over 8 weeks in two clinical trials (Lewinsohn et al. 1996). After the first clinical trial, 46% of adolescents in the treatment condition no longer met DSM-IV criteria for an affective disorder compared to 5% of adolescents on a waiting list. At a six-month follow-up, this percentage increased to 83% in the treatment condition and at a two-year follow up, treatment gains were maintained. In the second clinical trial adolescents with either MDD or dysthymia were included. Recovery rates were superior in the treatment condition compared to the waitlist control: 67% vs. 48% no longer met criteria for an affective disorder. Rates of relapse did not differ across all follow-up conditions, suggesting that the inclusion of booster sessions did not impact on treatment outcomes (Lewinsohn et al.).

One important area of research is to see how CBT works in primary care settings, as this allows the connection to be made between research and practice and evidence-based treatments to be more readily available for consumers (Asarnow et al., 2005). One RCT study of a CBT programme compared usual care with a quality improvement condition in primary care settings for adolescents with depression aged 13 to 21 years. The quality improvement condition involved a manualised CBT programme based on the CWD-A course mentioned above. At a 6-month follow up, participants in the quality improved condition had significantly lower Center for Epidemiological Studies-Depression (CES-D) Scale scores than usual care participants. They also had lower rates of severe depression, and self-reported higher rates of mental health quality of life and satisfaction with mental health care.
While self-reported suicidal ideation, suicidal attempts, and self-harm declined in both groups, there were no significant effects for the treatment condition. However, after follow-up, a third of quality improvement participants still exhibited severe depressive symptoms. An interesting finding was that when pharmacological and psychotherapy treatments were both available, participants tended to choose the psychosocial treatment (Asarnow et al., 2005).

One study (n=107, age range 13 to 18) of adolescents with MDD compared three manual-based treatments, including a CBT programme (a combination of Beck's cognitive therapy, functional family therapy and problem solving), systemic behavioural family therapy and nondirective family therapy (Brent et al., 1998). It was found that CBT resulted in more rapid and complete relief of depressive symptoms during the acute phase of a MDD episode than either of the other two therapies. However, as all therapies included a family therapy component, it is hard to differentiate the effects of each therapy. In a 2 year follow up long term outcomes were the same for all three treatments (Brent et al.).

Summary of literature review

Treatment will provide the best outcomes when it is matched to the underlying reasons for the individual’s depression (Parker, 2010). It is also important for treatments to include maintenance and relapse plans (Lewinsohn & Clarke, 1999).

Generally, research has found that all psychosocial treatments are comparably effective at reducing depressive symptoms (David-Ferdon & Kaslow, 2008; Michael & Crowley, 2002). A suggestion has been that for early adolescents and those with a mild depression, supportive psychotherapy treatments may be more appropriate while for adolescents with severe depression and those who are able to understand more complex treatments, CBT and IPT-A might be more appropriate (Mufson et al., 2004).

A final reminder for therapists when treating adolescent depression has been summarised in the following quote: “treatment of adolescents with depression should be approached with the combination of education, hope and patience” (Birmaher et al., 2000, p. 34).

References


