Depression - Causes, Diagnosis and Treatment Series

Depression in Children

Bernice T. Naylor

Editor

NOVA

Depression- Causes, Diagnosis and Treatment Series

DEPRESSION IN CHILDREN

DEPRESSION- CAUSES, DIAGNOSIS AND TREATMENT SERIES

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CONTENTS

PREFACE

Depressive disorders, which include major depressive disorder (unipolar depression), dysthymic disorder (chronic, mild depression), and bipolar disorder (manic-depression), can have far reaching effects on the functioning and adjustment of young people. Among both children and adolescents, depressive disorders confer an increased risk for illness and interpersonal and psychosocial difficulties that persist long after the depressive episode is resolved; in adolescents there is also an increased risk for substance abuse and suicidal behavior. Unfortunately, these disorders often go unrecognized by families and physicians alike. Signs of depressive disorders in young people often are viewed as normal mood swings typical of a particular developmental stage. In addition, health care professionals may be reluctant to prematurely "label" a young person with a mental illness diagnosis. Yet early diagnosis and treatment of depressive disorders are critical to healthy emotional, social, and behavioral development.This new book presents the latest research in the field across a wide spectrum of countries.

Chapter 1 - This is the second part of a study of dyslexia, children and depression, the first detailing empirical evidence. This reflective biographical study of N=29 dyslexic adults between the ages of 23 to 65 years olds, used a semi-structured investigative interview methodology to probe adult dyslexics for their childhood experiences. Dyslexic adults with depression, both with and without degrees were the largest group (N=21) recruited. Female dyslexics with depression was the largest subgroup (N=16) and this reflects the higher frequency of females developing depression than males in the general population, suggesting that females tend to cope more emotionally than males.

Fourteen themes were developed from the transcripts, which were investigated in this study. The themes were: What is dyslexia?; Difference; Humiliation; Schools and Teachers; Frustration and Anger; Avoiding and Being the Class Clown; Rebelling in the Classroom; Truanting and Running away from Home; Perfection; Bullying; Going into a Bubble and Regression; Why some Dyslexics get Depressed; Self-harm and Suicide; Post Traumatic Stress Disorder.

There seemed little difference between those who have recently left education, to those who left over forty years ago, suggesting that mainstream education has been slow to develop to screen mechanisms for children with disabilities to allow the access to the whole school curriculum.

The evidence presented in this chapter suggests that dyslexics commonly are humiliated in their class by their difficulties, which makes them frustrated by their inability to learn as

successfully as their peers. They perceive the classroom as a threatening environment rather than a place to gain new knowledge, so they begin to look for opportunities to reuse old knowledge rather than experimenting to learn new ones, thus they will use smaller words in essays and avoid writing tasks. Their avoidance camouflages their inabilities, and gets in the way of their teachers recognising that they are struggling, thus classroom humiliation and avoidance continues. Parents may also be to blame for covering up their child's difficulties by doing their homework for them.

The evidence in this study also suggests that avoidance is the key strategy for dyslexics to survive their school years, starting with avoiding putting up their hand to join the academic discourse in the classroom, to continual sharpening of pencils to reduce time spent on task in the classroom. At some point the child sees that the long hard effort they put into their assignments is not reflected in the marks they receive and this confirms to them that they are abnormal learner. They then look to either rebel against their classroom environment or aim to be invisible to reduce further their interaction with these feared stimuli. Avoidance turns to truancy, faking sickness to avoid school or running away from home. Depression begins to set in as they try to deal with the frustrations, anger and anxieties of their school day and a home that does not understand their difficulties, confirming some of their self-perceptions that their bodies are faulty, or that they are adopted or are aliens from outer space. Alternatively their self-blame could turn into self-harm to control their bodies, as they have little control over the rest of their world, as found in the case of cutting their bodies, anorexia, drug/alcohol abuse and binge-eating. These are cries for help when their other cries have not been heard. If they are still ignored then suicide, as seen in this study is an option to put an end to the long-term pain they are experiencing.

Chapter 2 - This is the first of two chapters to investigate dyslexia, children and depression. This chapter begins by defining what a child is, what depression is and lastly what dyslexia is. It is felt by the author that such a strategy is needed to clearly define the direction of the empirical investigation. Inclusion of the indicators of dyslexia, not only in pre-school but in primary and secondary school ages are necessary, so that a reader can assist in recognising unidentified dyslexia amongst those they meet, if necessary. This is necessary as it would seem from the evidence presented that either teachers are unable or unwilling to identify dyslexia in their pupils. Such a situation causes frustration and stress, with children therefore seeing the classroom as an environment that must be controlled and avoided. That learning is not the investigative process that it should be, but an opportunity where embarrassment and failure commonly takes place in front of their peers.

Dyslexics with high intelligence will use their intellect to carefully choose subject options that will limit their time doing feared tasks (e.g. reading out loud in class and essay writing) and choose science or design subjects. They will use their energies to avoid and choose easier to spell words. Dyslexics with normal or lower intelligence will have a harder time. They will have fewer strategies to avoid feared tasks and will begin to self-doubt and may either lash out or withdraw.

A large number of empirical studies in this area suggest that many dyslexics develop negative learning strategies, as a reaction to the frustrations and stresses they encounter, whilst this is an unhealthy attitude to learning, it is one option which will protect their self-esteem. Unfortunately evidence points to secondary manifestations developing which are both emotionally and psychological damaging. In some cases these can turn into physical manifestations with self-harm and suicide as the ultimate conclusion. All children hate to fail,

hate to be different and hate to be singled out as having special needs. Their segregation to the remedial or 'slow' table or taken out of the classroom for extra help may be a negative strategy as they are marked out as being different in the eyes of their class peers.

What is clear is that dyslexics do not feel integral to their family and society as a whole, and they look for ways to protect themselves from the harm that such a conclusion poses. Such a conclusion can, if taken to extremes, mean that young dyslexics can withdraw from society, self-harm to punish their perceived faulty bodies or limiting their burden on their parents and society by attempting suicide in extreme cases. Such a conclusion, especially in childhood suggests the support framework which children need to rely on from parents, peers, teachers, school and the medical profession is not working effectively. The Dyslexic Defence Mechanisms DDMs (Alexander-Passe, in press-2) suggest a means to understanding the processes involved in coping with an unidentified learning difficulty such as dyslexia.

Chapter 3 - This paper reviews recent literature on various aspects of depressive disorders in children and adolescents, including the epidemiology, clinical presentation, natural history, etiology and treatment. Depression is an important psychiatric disorder in youngsters that increases in frequency with age, often coexists with anxiety disorders and behavior disorders, and is associated with significant psychosocial impairment. Early depressive episodes often recur and persist into adult life along with long-term morbidity and mortality. The etiology of pediatric depression is complex, involving genetic, neurobiological, psychological and environmental processes. There is some evidence for short-term efficacy of both pharmacological and psychosocial interventions for the treatment of depression in children and adolescents. Given the relatively long duration of depressive episodes and high propensity for relapse and impaired psychosocial functioning, further exploration of the etiology, natural history and treatment of pediatric depression is warranted.

Chapter 4 - The World Health Organisation (WHO) estimates that major depression is the single greatest cause of years of life lost to disability worldwide (Global Health Forum for Research, 2008). In the United Kingdom, the cost of mental ill-health is currently estimated at £77 billion pounds, greater than heart disease and cancer combined. By 2020, the WHO predicts a 50% rise in child mental health disorders. The rise in mental health disorders may reflect the dramatic change in diet observed during the past century favouring mass food production coupled with a global decrease in fish and seafood consumption both during pregnancy and in children of school age. It is well established that polyunsaturated fatty acids are vital for the optimal development of both the brain and retina. They are abundant in the central nervous system and play a pivotal role in neurotransmission, serotonergic and dopaminergic function. The omega 3, docosahexaenoic acid (DHA; 22: 6n-3) and the omega-6, Arachidonic Acid (AA; 20:4n-6) are key components of cell membranes; they are required for normal brain structure and early visual development during pregnancy and infancy. Low maternal seafood consumption, that is less than 340 grams per week during pregnancy, is linked with higher scores of suboptimal outcome across a range of developmental measures in children. Furthermore, children with lower levels of omega-3 fatty acids are reported to have more temper tantrums, sleep disorders and behavioural problems than controls. This chapter discusses the role of nutrition in child and adolescent depression, with specific reference to essential fatty acids and brain function. An overview of specific comorbid neurodevelopmental disorders (such as attention deficit hyperactivity disorder; ADHD) is provided along with clinical trial evidence of the efficacy and safety of dietary

supplementation. The review concludes with an evaluation of nutritional trends as reflected in the blood profiles of patients with depression and ADHD.

Chapter 5 - Although childhood depression often co-occurs with other psychological conditions, there is little systematically organized knowledge as to how these co-occurring or comorbid conditions develop. This chapter presents several potential explanations for the co-occurrence of depression with other conditions, and illustrates these explanations using co-occurring depression and conduct problems. In the first section of the chapter, author present three potential explanations for co-occurring depression and conduct problems: (1) depression confers risk for conduct problems, (2) conduct problems confer risk for depression, and (3) shared risk factors account for their co-occurrence. In the second section, author describe a model that extends the shared risk factors explanation to include autonomic, prefrontal, and limbic system processes that may underlie co-occurring depression and conduct problems, and discuss how parent-child processes may interact with these child-specific factors to confer risk for this comorbid condition. In the final section, author identify several gaps in the literature and potential directions for future investigations involving depression comorbidity among children.

Chapter 6 - Major depressive disorder affects up to 10% of adolescents and is associated with substantial short-and long-term morbidity and mortality.

Depressive disorders are difficult to diagnose in children, because the symptoms are non-specific and due to the process of change at this stage of life. The picture varies enormously depending on the child's developmental stage.

This disorder affects the normal development of a child, school performance, social behavior and family relationships. Major depression in childhood or adolescence increases the risk of affective disorder in adulthood. The precise nature and course of the subsequent disorder remain unclear.

There is very little evidence for treatment in children. Most treatments are based on clinical experience or extrapolation from results of studies in adults. The increase over the last few years in the amount of available clinical research on the use of antidepressants to treat major depression in children and adolescents has substantially improved author knowledge of the safety and efficacy of these medications in the pediatric population. Many questions remain, however, that highlight the need to continue research in this patient population rather than relying on the extrapolation of data from trials involving adults.

Chapter 7 - Historically, psychoanalytic theories suggest that it is not possible for children to be depressed. Today, using criteria from the *Diagnostic and Statistical Manual of Mental Disorders* (APA, 2000), children and adolescents can be diagnosed with depression. Further, developmentally appropriate modifications of these criteria are being described and validated for use in young children (Luby, Heffelfinger, Mrakotsky, et al., 2002). When age-appropriate symptom states are assessed, it becomes clear that young children who are between the ages of 3- and 6-years can experience stable and specific depressive syndromes (Luby, 2007). Even with these advances, however, relatively little attention is paid to the diagnosis of depression in young children, particularly in comparison to the occurrence of disruptive behavior disorders in young children (e.g., externalizing disorders and conduct problems often are cited as the most common reason for preschool referrals for mental health services; Luby & Morgan, 1997; Renk, 2005). As a result, this chapter intends to draw attention to the occurrence of depression in young children by discussing the criteria of

available diagnostic systems, other considerations that may be helpful to mental health professionals, and available assessment measures and treatment interventions.

Chapter 8 - Childhood depression was once believed to be a rare or transitory developmental phenomenon (Shwartz, Gladstone, & Kaslow, 1998). Contrary to such assumptions, however, strong evidence has accumulated in recent years demonstrating the high prevalence and chronic course of childhood depression (Kessler, Avenevoli, & Merikangas, 2001). Epidemiological studies have shown that by age 14, approximately 9% of children have already experienced at least one episode of severe depression (Lewinsohn, Rohde, Seeley, & Fischer, 1993). In addition, a large proportion of children and adolescents with subsyndromal levels of depressive symptoms report significant functional impairment and seek treatment at the same or higher rates than their peers with clinically elevated levels of major depression (Kessler & Walters, 1998; Gonzalez-Tejera et al., 2005) Furthermore, the best predictor of depression in adulthood is childhood or adolescent depression (Kim-Cohen, Moffitt, Harrington, Milne, & Poulton, 2003) and youth onset of depressive episodes has been shown to be associated with chronic or recurrent episodes in adulthood (Weissman, Warner, Wichramaratne, Moureau, & Olfson, 1997). Such findings highlight the importance of identifying the factors that render certain youth vulnerable to the development of depression.

One factor that has been found to significantly increase the risk for depression in youth is parental depression (Beardslee, Versage, & Gladstone, 1998). More specifically, children with an affectively-ill parent are four to six times more likely than other children to develop clinically significant depressive episodes (Beardslee, Keller, Lavori, Staley, & Sacks, 1993; Hammen, Burge, & Adrian, 1991; Weissman et al., 1997). Parental depression has been hypothesized to influence child depression through multiple pathways including through the mediating role of higher levels of cognitive and interpersonal vulnerability factors. Cognitive and interpersonal vulnerability-stress theories posit that individuals with pre-existing maladaptive cognitive styles are more vulnerable to developing depression when they experience negative life events. Multiple cognitive and interpersonal vulnerability factors have been proposed including dysfunctional attitudes (Beck, 1963; 1972), low self-esteem (Brown & Harris, 1986), ruminative response style (Nolen-Hoeksema, 1987), depressogenic attributional style (Abramson, Metalsky, & Alloy, 1989), the personality predispositions of self-criticism and dependency (Blatt & Zuroff, 1992), and negative attachment cognitions (Bowlby, 1969; 1980). Providing support for the applicability of such theories to youth samples, cognitive and interpersonal vulnerability factors have been found to prospectively predict increases in depressive symptoms in youth following the occurrence of negative events (Abela & Hankin, 2008; Hankin & Abela, 2005).

Numerous studies have shown that high-risk children (i.e., those with a depressed parent) possess higher levels of cognitive and interpersonal vulnerabilities to depression than do low-risk children (i.e., those with non-depressed parent; Jaenicke et al., 1987; Taylor & Ingram, 1999). More specifically for cognitive vulnerabilities, children of mothers with a history of unipolar depression have been found to possess more negative cognitions about themselves than children of medically-ill parents or controls (Jaenicke et al., 1987). Similarly, high-risk children have been found to report lower levels of self-esteem (Hirsch, Moos, & Reischl, 1985) and more negative cognitive styles (Garber & Robinson, 1997) than normal controls. Such differences in cognitive vulnerabilities have been found to remain even after controlling for children's current levels of depressive symptoms (Garber et al., 1997). With respect to interpersonal vulnerabilities, children of depressed mothers have been found to exhibit

attachment styles characterized by greater insecurity as well as higher levels of interpersonal dependency (Abela, Hankin, Haigh, Adams, Vinokuroff, & Trayhern, 2005).

One way in which parental depression has been hypothesized to lead to higher levels of cognitive and interpersonal vulnerability to depression in youth is through the mediating role of parenting processes. Parental depression has been found to negatively impact parenting style (Cummings & Davies, 1994; Gerlsman, Emmelkamp, & Arrindell, 1990). For example, parental depression has been found to be associated with lower levels of warmth and acceptance as well as with higher levels of control in parents (Alloy, Abramson, Tashman, Berrebbi, Hogan, Whitehouse et al., 2001). Depressed mothers have been found to be less responsive to their children (Cox, Puckering, Pound, & Mills, 1987; Goodman & Brumley, 1990) and more critical (Conrad & Hammen, 1993) than non-depressed mothers. Depressed mothers have also been found to exhibit lower levels of involvement with their children (Weissman & Pykel, 1974). Last, greater severity of parental mental illness has been found to be related to more negative parenting practices (Kokes, Harder, Fisher, & Strauss, 1980).

Negative parenting practices, in turn, have been found to be associated with higher levels of depressive cognitions in children (Bruce, Cole, Dallaire, Jacquez, Pineda, & LaGrange, 2006; Garber & Flynn, 2001; Jaenicke et al., 1987; Mezulis, 2005; Whisman & Kwon, 1992). More specifically, lower levels of parental support and higher levels of parental rejection and over involvement, have been found to be associated with lower levels of self-esteem and more depressogenic attributional style in children (Tiggemann, Winefield, Goldney, & Winefeld, 1992). Similarly, higher levels of maternal restrictiveness and rejection have been found to prospectively predict increases in children's levels of self-criticism even after controlling for children's temperament (Koestner, Zuroff, & Powers, 1991). Lower parental care during childhood has been found to predict higher levels of depressive symptoms in young adulthood through the mediating role of depressotypic attitudes and attributions (Whisman et al., 1992). Last, Bruce and colleagues reported that negative parenting and negative life events were associated with higher levels of depressive cognitions in children, while positive parenting was associated with lower levels of depressive cognitions in children (Bruce et al., 2006). The relationship between negative parenting and negative cognitions was stronger for older children (Bruce et al., 2006). Thus, overall, there appears to be an association between negative parental practices and the development of vulnerabilities in children.

The goal of the present study was to examine whether parental depressive symptoms predict child cognitive and interpersonal vulnerability to depression through the mediating role of parenting. Although several studies have examined the relationships between (1) parental depressive symptoms and parenting processes, (2) parental depressive symptoms and child vulnerabilities, and (3) parenting processes and child vulnerabilities, to author knowledge, no studies have examined whether impaired parenting *mediates* the relationship between parental depressive symptoms and child cognitive and interpersonal vulnerabilities. Participants included 140 children between the ages of 6 and 14 with a parent with a history of major depressive episodes. During an initial assessment, parents completed measures assessing parenting styles and current depressive symptoms, while children completed measures assessing depressive symptoms, cognitive vulnerability factors (e.g., depressogenic attributional style, response style, dysfunctional attitudes, dependency and self-criticism) and interpersonal vulnerability factors (e.g., insecure attachment). The following hypotheses were examined: (1) higher levels of depressive symptoms in parents will be associated with higher

levels of cognitive and interpersonal vulnerabilities in children; (2) higher levels of depressive symptoms in parents will be associated with parenting styles characterized by lower levels of nurturance, consistency and responsiveness, and higher levels of restrictiveness; (3) lower levels of parental nurturance, consistency and responsiveness and higher levels of parental restrictiveness will be associated with higher levels of child cognitive and interpersonal vulnerabilities; (4) after controlling for the proportion of variance in child cognitive and interpersonal vulnerabilities accounted for by parenting style, parental depressive symptoms will no longer be significantly associated with children's cognitive and interpersonal vulnerabilities

Chapter 9 - Depression vulnerability in children seems to be connected with cognitive and interpersonal vulnerability, like pessimistic thinking and insecure attachment to their parents (DesJardin, 2003; Richaud, 2006a). Negative self-perceptions are believed to result from the negative competency evaluations of significant others, such as parents and teachers. A child's self-perception of competency may interact with others' appraisals to influence vulnerability to depression (Richaud, 2006a). According to DesJardin (2003) children who are vulnerable to depression have a tendency to make pessimistic remarks; have low self-esteem; poor coping strategies such as chewing over their problems without solving them; rigid and extreme personality traits that include self-criticism and over-dependency on their parents. At the same time, although clear gender differences in depression only appear after adolescence, developmental perspectives have placed its origins in childhood socialization. Relatively little is known, however, about continuities and discontinuities between childhood and adult behavior.

The aim of this chapter is to study vulnerability to depression in normal boys and girls in middle and late childhood, and its relation with cognitive vulnerability, as expressed in children's self-perception of academic and social competency, controllability of perceived stressors, and coping strategies.

Method Argentine adaptation of Harter's Self-Perception Profile for Children, Argentine adaptation of Harter and Nowakowski's Dimensions of Depression Profile for children and adolescents, and Coping Questionnaire for children were administered to 1,050 middle class children, aged 8 to 12, from four primary schools, two state-run and two public, residing in the city of Buenos Aires and various towns in the province of Buenos Aires.

Results Cognitive vulnerability expressed as negative self-perception of academic and social competence is a factor of risk that may offset depressive vulnerability in middle and late childhood. This cognitive vulnerability is also connected with threat appraisal related to close-family interpersonal difficulties. Coping with these difficulties follows a pattern characterized by non-problem focus, low approach, high avoidance and emotion-focused coping. Finally, greater gender differences in vulnerability to depression appear in middle childhood and diminish in late childhood, males being more vulnerable to depression than girls, probably due in part to changes in socialization practices.

Chapter 10 - Depression poses a significant risk to the mental health of children and adolescents, and with a prevalence rate of about 2-10% is not uncommon. While most pediatricians feel responsible for identifying depression in children and adolescents, successful recognition is estimated to be as low as 17%. Depression has been found to worsen health outcomes, as child patients with depression may be three times more likely to be medically noncompliant than their non-depressed counterparts. Fortunately, depression is also highly treatable with psychotherapy and psychotropic medication. Because depression causes

emotional suffering and is a risk factor for medical noncompliance and poor health outcome, the pressing need to recognize childhood depression in the medical setting is clear. This commentary discusses the importance of identifying and treating childhood depression in the medical setting and suggests future directions for research.

"The incidence of untreated child and adolescent mental health problems constitutes a public health crisis for our nation" (American Psychological Association Task Force on Psychology's Agenda for Child and Adolescent Mental Health, 2003, p. 3). This statement highlights the current dismal state of affairs for children with mental health needs. It is consistent with the perspective of a relatively recent report by the Surgeon General (U.S. Department of Health and Human Services, 1999). Some estimates indicate that as many as 75% of children with mental health needs remain untreated (Mash & Dozois, 2003). Unfortunately, depression is common in children of all ages. It is estimated that 2%-10% of children and adolescents experience a depressive disorder (Birmaher, Brent & Benson, 1998; Costello, Mustillo, Erkanli, Keeler & Angold, 2003; Dopheide, 2006). The adolescent lifetime prevalence rate for major depression is estimated to be even higher at 14% (Hammen & Rudolph, 2003).

Given this mental health "crisis," what role do medical professionals have? The medical setting provides both a unique opportunity and responsibility to recognize depressive symptoms and address them through treatment or referral. Young people suffering from "psychosocial dysfunction and/or chronic illness" see their primary care physician about four times a year (Bernal et al., 2000, pp.263-264). Many health care systems structure the primary care physician as the "gatekeeper" to specialty mental health care (Costello et. al, 1988). However, recognition of psychosocial problems in the pediatric setting is poor and has been estimated to be as low as 20% (Bernal et al., 2000). Other estimates find that childhood psychiatric illnesses are recognized about 17%-59% of the time in the medical setting (Goldman et al., 1999). In addition to psychological suffering, health care utilization and costs are elevated for children suffering from psychosocial problems (Bernal et al., 2000). Although numerous barriers to recognition exist, identification and diagnosis are crucial to obtaining treatment.

Depression in the medical setting may be complicated by other problems, such as the co-occurrence of chronic illness. Chronic childhood illness is an intensely stressful experience for the child as well as the family unit. The first author's clinical experience as a schoolteacher in a children's hospital has brought to her attention the unique stressors associated with seriously ill children and their families. The hospitalization and treatment process can create enormous stress, as family members can be separated by large distances while children are treated at specialty children's hospitals. Hospitalization or outpatient treatment may linger into months or years. With the family unit far from home, the comforting routine of school and work is absent. Chronic illness introduces a host of difficulties for young people that include body image problems, difficulties with peers, academic problems and stressful medical procedures (Jellinek & Snyder, 1998). In this environment, the first author has noted that depressive symptoms in pediatric inpatients and their siblings can go unrecognized and untreated. While the physical health of the child is imperative, mental health problems must be recognized as well.

In addition to this anecdotal experience, research has also established that chronic illness has a profound effect on the psychosocial functioning of children and adolescents. It is not surprising that depression has been found to be associated with chronic and serious illness

(Scott et al., 2007). Depression is not only a devastating mental illness, but also may have a substantial impact on medical treatment adherence and even survival. In their meta-analysis examining the relationship between depression and medical non-adherence, DiMatteo, Lepper and Croghan found that depressed patients were three times more likely than non-depressed patients to be medically non-adherent (2000). Although the meta-analysis focused mainly on adult studies due to the lack of pediatric studies, their findings were compelling. A recent study on treatment adherence in adolescent oncology patients also found that non-adherence is correlated to elevated depression levels. Sadly, medication non-adherence was also correlated with mortality. Although the majority of adolescents exhibited subclinical levels of depression, this study highlights the need to identify even minor mood disturbances in seriously ill adolescents (Kennard et al., 2004). The connections between depression, medical adherence, and survival make detection and treatment of depressive disorders essential.

Clinicians in the medical setting need to be attuned to depressive symptoms, as medication and treatment side effects can mimic depression. Symptoms characteristic of depression--namely fatigue, appetite problems and cognitive changes--might easily be misinterpreted by clinicians because they are also common side effects of cancer treatment (Dejong & Fombonne, 2005). Some research has suggested that adolescents may be less willing to tolerate intensive drug regimes, such as immunosuppressant drug therapy, due to side effects such as obesity, infection and depression (Griffin & Elkin, 2001). Numerous medical conditions can imitate depressive symptoms in young people, including infection, epilepsy, diabetes and hypothyroidism (Jellinek & Snyder, 1998). Pharmaceutical drugs such as benzodiazepines, corticosteroids and oral contraceptives can also produce depressive symptoms in children and adolescents (Jellinek & Snyder, 1998). Thus, it is important to keep in mind that treatments for medical problems may actually create subsequent depression through either the side effects of medication or the social impact of coping with medical illness.

Numerous barriers to identification of childhood depression exist even for those who are mental health professionals. Although the symptoms needed to meet diagnostic criteria are the same for adults and children, there are developmental considerations that may affect symptom presentation. For example, irritability seems to be more common in children with depression. In addition, children's capacity to report their symptoms may be influenced by cognitive development and language considerations. Furthermore, given the high rates of comorbidity with disruptive behavior disorders, depression may be missed or "masked" by other more readily recognized disruptions (Hammen & Rudolph, 2003).

This diagnostic issue is complicated in the medical setting with the additional barriers mentioned above; medical illness causing/exacerbating depression and treatment side effects that appear similar to depression. In a study surveying pediatricians' thoughts about their role in diagnosing and treating childhood depression, 90% of pediatricians considered themselves responsible for diagnosing depression in youth. However, only 46% of the pediatricians felt they had the necessary skills to diagnose depression. "Inadequate time to provide counseling or education" and "incomplete training to diagnose or counsel" were some of the most commonly perceived barriers (Olson et al., 2001, p. 93). Indeed, lack of time appears to be the most commonly perceived obstacle for "identification and management of children's psychosocial issues" in the medical setting (Horwitz et al., 2007, p. e208). With the current health care crisis, it is alarming, but not surprising, that psychosocial issues go unrecognized in the already overburdened medical setting. However, given the evidence that depression can

exacerbate illness and affect outcome, and that it can be successfully treated when identified, it is incumbent upon medical professionals to identify childhood depression.

The importance of diagnosing child and adolescent depression is particularly critical as successful therapeutic interventions and psychotropic medications are widely utilized. In fact, both intervention types can be helpful. Emslie and associates (2002) examined the effects of fluoxetine, a selective serotonin reuptake inhibitor (SSRI) in a randomized clinical trial with 122 children and 97 adolescents with major depressive disorder (MDD). They found that a protocol of 10 mg/day of fluoxetine for a week followed by 20 mg/day for eight weeks was associated with a significantly greater drop in depression scores compared to a placebo condition. For mild to moderate childhood depression, psychotherapy can be considered an alternative to psychotropic medication (Son & Kirchner, 2000). Although the evidence for various kinds of psychotherapy for children is limited and mostly based on studies with adults, a variety of approaches are used with children, which should be tailored to children's developmental level (Son & Kirchner, 2000). The most compelling evidence exists for the effectiveness of Cognitive-Behavioral Therapy (CBT) in reducing depressive symptoms. In an 8 week study, adolescents provided with CBT showed significantly greater reductions in depression compared to those assigned to a waitlist control condition (Clarke, Rohde, Lewinsohn, Hops & Seeley, 1999).

The effects of psychotropic medication may enhance the effects of psychotherapy--at least for adolescents--as suggested by a randomized controlled trial conducted by the Treatment for Adolescents with Depression Study Team (2004). They found that the combination of fluoxetine with CBT was significantly more effective in alleviating depression among adolescents than was either CBT alone or the placebo condition. The combination of fluoxetine with CBT also showed the greatest reduction in clinically significant suicidal thinking. Effective treatments for child and adolescent depression are prevalent; therefore, the identification and treatment of such a debilitating and costly disorder is vital.

Pediatricians are the gatekeepers in children's healthcare. They can function as a unique bridge between mental and physical healthcare for young people. It is imperative that significant mood changes and depressive symptoms are recognized in the medical setting, and action is taken. Untreated child and adolescent depression not only leads to unnecessary suffering, but also is associated with comorbid substance abuse and other psychological disorders (Hammen & Rudolph, 2003). Indeed, high levels of depressive symptoms in adolescence have been found to be predictive of suicidal acts in later adolescence and early adulthood, making prompt identification critical (Nrugham, Larsson & Sund, 2008). The barriers are numerous, but because the alternative is unthinkable, identification of childhood depressive disorders in the medical setting must take precedence. Further research is necessary to improve understanding of how depression and medical illness interact to exacerbate or complicate children's health. In addition, research is required on how to accurately diagnose medically ill children with depression. Furthermore, greater knowledge is needed about how to improve and deliver treatments to address the complicated issues that are presented by children with medical illnesses and depression.

Chapter 11 - The Gilles de la Tourette Syndrome (GTS) is a neurodevelopmental disorder characterised by the presence of multiple motor tics and at least one phonic tic. GTS is increasingly recognized as a relatively common neuropsychiatric disorder, usually diagnosed in early childhood. Comorbid behavioral problems occur in approximately 90% of patients,

with attention deficit hyperactivity disorder (ADHD) and obsessive-compulsive disorder (OCD) being the most common ones. Depression is also common, with a lifetime risk of 10% of patients. Moreover, converging evidence shows that children with GTS may have depression ratings that are higher than those in other school age children but lower than those found in children with primary depressive disorder. Whilst the association between GTS and depressive symptoms does not appear to be mediated genetically, social and environmental factors are thought to be of etiological importance. Children with GTS have involuntary movements and noises, which might amount to a chronic stigmatizing disorder in a school setting. This in turn could isolate the individuals, make them a subject for bullying, and ultimately lead to depression. The depression in children with GTS has been shown to result in a reduced health-related quality of life, and in severe cases it may lead to hospitalization and even suicide. Further research is needed to address factors of particular relevance to the etiology of depression in children with GTS and thus improve its recognition but also treatment and outcome.

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Chapter 1

DYSLEXIA, CHILDREN AND DEPRESSION: RESEARCH EVIDENCE

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ABSTRACT

This is the second part of a study of dyslexia, children and depression, the first detailing empirical evidence. This reflective biographical study of N=29 dyslexic adults between the ages of 23 to 65 years olds, used a semi-structured investigative interview methodology to probe adult dyslexics for their childhood experiences. Dyslexic adults with depression, both with and without degrees were the largest group (N=21) recruited. Female dyslexics with depression was the largest subgroup (N=16) and this reflects the higher frequency of females developing depression than males in the general population, suggesting that females tend to cope more emotionally than males.

Fourteen themes were developed from the transcripts, which were investigated in this study. The themes were: What is dyslexia?; Difference; Humiliation; Schools and Teachers; Frustration and Anger; Avoiding and Being the Class Clown; Rebelling in the Classroom; Truanting and Running away from Home; Perfection; Bullying; Going into a Bubble and Regression; Why some Dyslexics get Depressed; Self-harm and Suicide; Post Traumatic Stress Disorder.

There seemed little difference between those who have recently left education, to those who left over forty years ago, suggesting that mainstream education has been slow to develop to screen mechanisms for children with disabilities to allow the access to the whole school curriculum.

The evidence presented in this chapter suggests that dyslexics commonly are humiliated in their class by their difficulties, which makes them frustrated by their inability to learn as successfully as their peers. They perceive the classroom as a threatening environment rather than a place to gain new knowledge, so they begin to look for opportunities to reuse old knowledge rather than experimenting to learn new ones, thus they will use smaller words in essays and avoid writing tasks. Their avoidance camouflages their inabilities, and gets in the way of their teachers recognising that they

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are struggling, thus classroom humiliation and avoidance continues. Parents may also be to blame for covering up their child's difficulties by doing their homework for them.

The evidence in this study also suggests that avoidance is the key strategy for dyslexics to survive their school years, starting with avoiding putting up their hand to join the academic discourse in the classroom, to continual sharpening of pencils to reduce time spent on task in the classroom. At some point the child sees that the long hard effort they put into their assignments is not reflected in the marks they receive and this confirms to them that they are abnormal learner. They then look to either rebel against their classroom environment or aim to be invisible to reduce further their interaction with these feared stimuli. Avoidance turns to truancy, faking sickness to avoid school or running away from home. Depression begins to set in as they try to deal with the frustrations, anger and anxieties of their school day and a home that does not understand their difficulties, confirming some of their self-perceptions that their bodies are faulty, or that they are adopted or are aliens from outer space. Alternatively their self-blame could turn into self-harm to control their bodies, as they have little control over the rest of their world, as found in the case of cutting their bodies, anorexia, drug/alcohol abuse and binge-eating. These are cries for help when their other cries have not been heard. If they are still ignored then suicide, as seen in this study is an option to put an end to the long-term pain they are experiencing.

Key Words: Dyslexia, Children, Depression, Avoidance, Coping, Self-harm

INTRODUCTION

The first chapter of this study (Alexander-Passe, in press-3) was a detailed empirical review to investigate correlations between dyslexia, children and depression. It began by defining what a child was, what depression was and lastly what dyslexia was. It was felt by the author that such a strategy was needed to clearly define the direction of the empirical investigation. It clearly defined the indicators of dyslexia, not only in pre-school but in primary and secondary school age so that any reader could assist in recognising unidentified dyslexia amongst those they meet. This is necessary, as it would seem from the evidence presented that teachers are either unable or unwilling to identify dyslexia in their pupils. Such a situation causes frustrations and stress, with children therefore seeing the classroom as an environment that must be controlled and avoided. Moreover, learning is perceived as not the investigative process that it should be but an opportunity where embarrassment and failure commonly takes place in front of their peers.

Empirical evidence suggests dyslexics with high intelligence will use their intellect to carefully choose subject options that will limit their time doing feared tasks (e.g. reading out loud in class and essay writing) and choose more science or visual subjects. They will use their energies to avoid and choose easier to spell words. Whereas dyslexics with normal or lower intelligence will have a harder time. They will have fewer strategies to avoid feared tasks and will begin to self-doubt and with either lash out or withdraw.

A large number of empirical studies in this area suggest that many dyslexics develop negative learning strategies, as a reaction to the frustrations and stresses they encounter. It is hoped that in most cases dyslexics will just avoid reading and writing to get through school, whilst this is an unhealthy attitude to learning, it is one option which will protect their self-esteem. Unfortunately studies point to secondary manifestations developing which are both

emotionally and psychological damaging. In some cases these can turn into physical manifestations with self-harm and suicide as the ultimate conclusion.

As noted earlier by Burden (2005) all children hate to fail, hate to be different and hate to be singled out as having special needs. Their segregation to the remedial or 'slow' table or taken out of the classroom for extra help may be a negative strategy as they are marked out as being different in the eyes of their class peers.

What is clear is that dyslexics do not feel integral to their family and society as a whole, and they look for ways to protect them from the harm that such a conclusion poses. This idea is most damaging as it suggests that their attitude towards themselves is faulty, along with the perception that they have no place in the society around them. Such conclusions can if taken to the extremes, mean that those young dyslexics can withdraw from society or they may wish to take what they see as the ultimate sacrifice and rid society of their ills. Such a conclusion, especially in childhood suggests the support framework which children need to rely on from parents, peers, teachers, school and the medical profession is not working effectively.

The Dyslexic Defence Mechanisms DDMs (Alexander-Passe, in press-2) suggest a means to understanding the processes involved in coping with an unidentified learning difficulty such as dyslexia. In this chapter an investigation will be made using biographical semi-structured interview studies with a range of dyslexic adults.

METHODOLOGY

Aim of the Study

The aim of the study was to pose a semi-structured interview script to a range of UK adult dyslexics to gauge how they cope, their reactions to success/failure and a review of their childhoods to identify where any emotional damage originated. Adult dyslexics were chosen as they would have the ability to look back to their childhood for the origins of their coping strategies and can give a data rich explanation of their suffering. Four groups were identified and sourced: dyslexics with a clinical depression diagnosis, degree-educated dyslexics, non-degree-educated dyslexics and dyslexics with a criminal record. Four sub-groups were also found, as dyslexics with a clinical depression diagnosis along with ones with criminal records could also be divided by degree and non-degrees.

Sample

Participants were located from three places. Firstly, an email was sent via large UK dyslexia newsgroups, secondly an advert on the beingdyslexic.com website and thirdly approaching several dyslexia associations for adverts to be put onto their websites. Although four groups were requested, the largest group replying were dyslexic adults with depression.

All participants were required to provide evidence of a formal diagnosis of dyslexia (commonly found to be from an educational psychologist), however evidence of depression was not sought as this would be more difficult to attain from their physician or hospital.

The study noted in this chapter is part of a larger investigation for a book (Alexander-Passe, in press-6). It uses a select number of items from a qualitative study using N=24 items (see Table 1), based on a pilot study in Alexander-Passe (in press-1) investigating a group of N=7 diagnosed

In this study N=29 dyslexic adults were recruited, N=21 had a depression diagnosis (rated as either a clinical depression diagnosis or at least one courses of anti-depressants prescribed by their physician/GP). With the depressed dyslexics, there were N=15 females (mean 23yrs, SD 3.4) and N=6 males (mean 23yrs, SD 3.4). With the non-depressed dyslexics, there were N=3 females (mean 23yrs, SD 3.4) and N=5 males (mean 23yrs, SD 3.4).

Apparatus

A semi-structured interview script was used with N=24 items, however as it was an investigative interview. Interviews lasted between an hour and three hours.

Table 1. Semi-Structured Interview Script, based on Alexander-Passe (in press-1).

1.	Please describe how you are feeling today? (Are you taking any depression medication at present?)
2.	Please describe your life/yourself? (I need to create a description of you e.g. age, education, job, character, personality etc)
3.	Do you enjoy life?
4.	Please describe your childhood? Was it happy? (e.g. with your family)
5.	Do you have any siblings? Do you think you were treated fairly/unfairly to your siblings?
6.	Please describe your time at school? Was it enjoyable?
7.	Did you ever get frustrated from your learning difficulties?
8.	What does dyslexia mean to you?
9.	Is dyslexia something positive or negative?
10.	How does dyslexia affect your daily life?
11.	What classic dyslexia symptoms to you have?
12.	Do you think your hobbies help you? Giving you self-confidence?
13.	Do you ever blame your dyslexia for things?
14.	Do you/have you ever resented your teachers at school for not seeing your difficulties?
15.	Do you ever feel rejected? Please explain?
16.	How does failing or getting things wrong affect you?
17.	Do you ever say why me? Why am I dyslexic?
18.	Do/Did you self-harm? Why? What are the triggers?
19.	Have you ever thought about or tried to commit suicide? Why? What were the triggers?
20.	Do you think dyslexia and depression are

correlated (linked)?
21. Did you ever truant/run away from home?
22. How do you feel going into schools now, what triggers any negative emotions?
23. Do you enjoy being you? Please explain?
24. Would you call yourself a successful dyslexic?

The interview process, Confidentiality, Informed consent and Personal disclosure

The majority of the interviews took place over the phone (N=27) after email contact and basic details confirmed. N=2 took place in participant's homes. Participants were sent details of the study before the telephone interview took part. All confirmed on the phone before the start of each interview, specifically that they were happy to proceed. They were advised that they could avoid any questions that were too emotional to answer; fortunately, no participant took this option, although avoidance was noted in several interviews, thus further investigative questions were required, these pushed the number of items from N=24 to over N=100 at times. Each interview was audio-taped, then transcribed and then subject to analysis to isolate numerous themes.

Confidentiality was assured at several points in the investigation, firstly in the emailed advert, secondly emailed confirmation and request for basic details (name, age, education etc) and lastly at the start of each interview. At the start of the interview each participant was asked to confirm they were happy to proceed on tape. As the interviews were about participants disclosing emotionally painful or frustrating events it was felt best that the interviewer (the author) also disclosed that firstly he was diagnosed dyslexic at fourteen years old and secondly that he had been depressed as a young child and thus he understood the difficulties at school that they may have encountered.

Analysis

Each interview was recorded on audio tape, then transcribed. They were then computer spell checked with minimal grammar changes to improve readability. A second check was made by printing off the interview to check that it made sense. This was then emailed or posted to the volunteer for them to check and amend as they wished. They were also given the opportunity to add additional notes or post interview revelations, as such investigative interviews may also trigger post-interview thoughts. The results were then read through once and then at the second time a copy was cut up to create a range of themes to describe the data.

RESULTS

The data produced by this study totalled approximately 250,000 words and was highly detailed. Analysis to write this chapter and the authors proposed book, suggested that both IPA and theme analysis was the most suitable way to present the data. Therefore fourteen main themes were highlighted for this chapter and one quote out of the many is given below to summarise what was found. These will be discussed afterwards. IPA will be used for the author's book (Alexander-Passe, in press-4). Each quote used to describe the theme or sub-theme notes the participants name and whether as an adult they could be rated as depressed

according to the study criteria. This criteria is based on them personally seeking medical intervention for their trauma. However the comments from non-depressed participants are also shown. The aim of the study was to indicate whether depressive symptoms were present amongst all the dyslexics questioned.

Are those rated as non-depressed suffered any less traumatised by their experiences? As the evidence suggests there is little difference the trauma encountered, why have they not seeked medical intervention? The discussion section of this chapter will aim to shed light on this phenomena.

What is Dyslexia?

What does it Mean to You

What does dyslexia mean to you? It is, what it means to me is my brain is wired differently, it's basically a different brain to a non-dyslexic and so therefore I see the world differently, I handle problems differently and it means I sort things out differently and for me it doesn't mean that it's wrong….I see it as an ability. (Milly, depressed)

Society's Reaction

Do you think the problem is us (dyslexics) or the world around us? No, I do not believe there is a problem. To hear some people say [things, or] to receive certain reactions, if it can be agreed that we have difficulties in learning, which doesn't make us inferior or worse than others. I do not think it is a problem, I do not see it as a problem with them or us, and it is just a lack of understanding. If they understood dyslexia and the implications of dyslexia, there would be fewer problems encountered. (Jordan, non-depressed)

The Label

What does dyslexia mean to you? To me, to be honest, it basically is a label that is given to people whose strengths are not made for today's society. That is all it is. If you give things to people visually, they seem a lot happier. To me it is a label that's says I am not the best at reading and writing and I never will be. **Is it something positive or negative?** I think now it is a positive, but not at school. I am very happy with who I am and where I am, so it is a positive. I think for any child, who is dyslexic, it is a negative. (Kirsty, depressed)

Difference

Feeling Different

How was your time at primary school? I always felt left out, on the outside. **By your teachers or peers?** I felt I did not fit, I felt they were doing things I could not do. So it was like a two way process, if I felt I could join in, I would, and thus if I did not join in they just left me alone. Teachers generally tended to be rather supportive without really understanding what the problem was. (George, Depressed).

Being Unique

Growing up do you think you felt normal? [Pause] Strangely enough, I have not thought about that. I always thought I was a bit unique. I have never thought about it. I always thought I was some case study for some higher being and that they were keeping an eye on me. Again, it was the little world I escaped to. I always thought I was not 'run of the mill' [something different/special]**Did that make you feel good, being a little bit different** Yes, I would say, I used it to my advantage in my own little world. I did think I was different, not stupid, but different from everybody else. (Peter, non-depressed).

It Defines Me

Would you say it is something you would like to get rid of or something you like to have? It describes me and makes me different from everybody else and because I am different, I see things differently. It means I am able to explain things, because of the way I am. I can research things and see it from different angles and then explain it well to people who do not understand it. It means I can find different ways to explain it. **Do you like being different?** Yes [laugh]. (George, depressed).

Like an Alien in a Different Planet

You remind me of another volunteer who is also a massive science fiction fan; he said that being dyslexic is like being 'Spock' from Star Trek, as he was different and didn't quite fit in. Yes I can see that, sci-fi are full of characters who are 'other', you have like 'Doctor Who' who is different from all the people around him, he goes on all these adventures but in the end he is on his own. He can deal with it, I really hate being like everybody else, I cannot stand it. It does cause you pain, annoyance, and things, you know. There is very much a sci-fi community. Do you think growing up that sci-fi was your hobby, to get your self-confidence, knowing all the detail? Yes, I had very little interest in the other things (like make-up), I was not sporty that was not something I could identify with. I was not girly, so again I could not identify with that. But with sci-fi, when you are different you are celebrated. 'Spock' is different and really smart, which is like me…I love learning new things. (Kirsty, depressed)

Sibling Comparison

You were talking about being compared to your sister, was that more in the school, school reports or generally. Everything. I am the black sheep I am, 'I never do anything right'. I did not do…yes I think, it is because I did not or do not now like to cause any sort of hassle or anything. There was never any issue with her that 'she could work harder' and that I had turned into a 'petulant girl' growing up. With my sister she did okay, did her A'levels and pottered on, it was all right for her. But for me I was struggling all the time, they said 'I could do better or why can't I do this', you know I remember them saying 'I just told you that, why can't you do it, I just showed you how to do that', I said back 'I can't, I don't know'. **Did feel that you fitted into your family?** No. **Did you feel normal growing up?** No. **It sounds similar to attempting suicide, the feelings of no being wanted?** Yes. I used to write help signs and stick them on the window, for people to come and rescue me. **Growing up did you ever feel that you might be adopted?** Yes, well. I had an aunty, my dad's younger sister and she was brilliant, we always got on until she died. And I always thought, as she never got married or had kids, we look similar and I always used to think I was hers but my mum and

dad had to look after me. I sifted through my parents love letters to find proof, evidence that I was my aunt's really. (Rachel, depressed).

Thinking you were Adopted

Many dyslexics I talked to in this study, felt they did not fit into their family and felt like they were adopted. Oh yes, I feel like that all the time. Because everybody else in your family fits together in a puzzle and you do not. Especially the first time I came back from university, I found it difficult and went back the same day and said I wouldn't do it again, it was so weird being back with people you hadn't been with and felt crowded with. I had spent most of my time (at home) growing up in my room; I did feel 'other' in my family. I knew from a psychological place that they loved you and that was important then being part of it, as sometimes you feel quite apart from the family, as they seem fine without you. When you were stuck in your room, they carried on perfectly well without you. Then you turn and it is weird and you don't know where you fit in. (Kirsty, depressed).

Being Labelled Early

I guess you are first person I know who was diagnosed at five year old stage. It is fairly interesting how you view the situation. Do you know what it is, I can't remember a single time in my life when I haven't been told that I'm dyslexic. It has been a constant word in my life. I can only vaguely remember the test, at the time I wasn't sure why I being tested, being taken out of class for it. I don't remember ever doing badly and everyone around me blaming my dyslexia. It has always been this word, I'm angry that in my whole life I have been labelled, just because the educational system didn't fit into my strengths, that I didn't fit into a mould, my brain isn't like yours, we are all different, you know. I guess if you test my whole class, most would have a similar IQ, a few would have a high IQ, and others might have an IQ a bit lower. We all have our strengths and weaknesses, it's a spectrum. I now know what my strengths are; I have proven I'm good at things. (Izzy, non-depressed)

Humiliation, Schools and Teachers

Humiliation

I didn't pick up [improve in reading/writing] as fast as I should and I was labelled "lazy/stupid" I was quite a boisterous lad and I had an elder brother who looked out for me so that I didn't come into it [get into trouble], but when I was put into the remedial class the humiliation I felt still stays with me today. I am working with disabled people now and I feel guilty about having these feelings [the way they do] but the reality is that at that time, in those days it was called that "spass" [spastic] class. I lived in a small community, and one of the things that I [now] would really get distraught about was [is], trying to hide my education and where I actually had it, because I felt shamed of it, attending what was an approved [delinquent] school without breaking the law in anyway. (Adrian, Depressed).

How Schools and Teachers React

When I got into secondary school, by this time I was diagnosed, I was diagnosed when I was 7 or 8 years old. When my parents had that sort of information, they were able to,

basically started to get the proper education for me and to get me sent to a school that recognised my condition. The primary school I was going to at the time, the headmaster was of the opinion that dyslexia did not exist and was some made up term to excuse stupidity. This actual headmaster at my primary school told my parents that I was basically backward and that I would never be educated and never have a job, never have a career and so my parents basically wanted me to go to a different school and tried to get the educational authority to acknowledge that something was wrong. Well they finally managed to do that and I was sent for the last year of my primary school [education] to a different school where I was properly supported, received five hours of intensive English teaching a week from a special teacher who came into my school. Then when I went to my secondary school it again supported me. My parents bought books for the school library for the teachers and I received education from a specific specialist trained teacher - I received three hours a week through the whole of my years of secondary education. **Do you feel you were lucky at the level of support you got?** When I know how other dyslexic children and people…when I have met dyslexics my own age and younger I know that I was very lucky. I was lucky because my parents found out what it was, they knew something was wrong and they knew I wasn't stupid and it was grandmother who read a newspaper about the dyslexia institute [now dyslexia action] and she paid for me to go to the institute and be assessed. So I was very lucky. My family basically weren't willing to accept what the teachers were saying. My dad is a very stubborn person and he basically fought…both my father and mother fought, basically when I was diagnosed that was when my father realised he was dyslexic, and up to then he always knew he had a problem with school and with reading and writing, but he didn't know what it was. He is an incredibly intelligent person and I think he realised that that was what he had [dyslexia] and now he has been treated and his underlying angers came out. He opened up a lot [Pandora's Box] and made sure I got what I needed [educationally] because he never got any form of support or recognition when he was growing up. (Milly, depressed).

Denying there was a Problem

How early did your parents recognised there were problems? They did something about it, I do not know the answer, but they did something about it at an open evening at my school and there was work displayed and they faked all my work. **They faked it, really.** Yes and my dad turned round to them and said 'he didn't do that' and they said 'we couldn't show the real work as it was crap', you know he did not say crap but they could not show it. So that was the moment. **How old were you?** I was around nine or ten. (Malcolm, non-depressed).

Not Fair

Do you/did you ever say 'why me, why am I dyslexic'? Um yes I think that's fair, I do feel, because the education [my schooling] was hard, doing essays was hard, doing examinations was hard. I did think why this can't be easier, why my brother can do this much easier than me and I would see other people who could just turn… [Do work easily]. I love English [the subject] and loved writing stories, creative writing, as I had good stories to tell and other people could just write reams and reams of paper and I just wished I could do that. So yes I did [say why me]. (Milly, depressed)

The Wrong Support

I have always said about teachers with me, it is about them being reluctant. You get the ones who do not like you and they tell that you are 'not trying' and are not going help you, there are those who let you get on with what ever you want, they do not dislike you or like you. Then you get the other kind that try and help you, they believe they will save you and they are the worst kind as I said before. I would rather for the whole of my school life to have been left alone, left to do the lessons, if I was struggling I would have rather wait for help, then I will ask for help. My mother was very good, she helped me more than anyone else, I think she taught me to read and write. There were so many 'so-called' helpers, but as I said its not help, taking a child out of their classroom, in front of your so-called friends for special lessons is the worst thing you could possibly do. I think that that is more destructive than anything else, humiliating a child is destructive and I got that a lot, I am angrier about that than anything else. (Izzy, non-depressed)

School Reports

How were your school reports, what sort of comments did they say? 'Lack of effort, could try harder, lack of attention, messy', occasionally there were some positive comments, as generally I was not doing as well as I could. I did not start reading properly until I was about eight or nine years old, but I managed to catch up and over took everybody else. (Norman, depressed).

Good Teachers

I was very lucky to have a really good remedial maths teacher, Mr. Anon, he was really amazing. Like I said, I was put in a class with children with behavioural problems, if there was a kickoff in his class they would not try, as he could give as well as he got. He really was good at making you have faith in yourself, in my case, he was one of the only teachers that tried different ways to teach me, he would try one thing and if it did not work, he would go home and think of a new strategy to try. He would very much try to focus you. I took every opportunity to put up my hand and try and relay the information, even if I got really bad grades on a paper, they could say 'she puts her hands up, she is communicating how she feels, she can relay the information back to us' but there was my discrepancy, they would note that 'I could say it in class verbally but not in writing…we don't understand why'. (Kirsty, depressed)

Resenting Teachers

You earlier talked about resenting your teachers for not seeing your problems earlier. How bitter do you feel towards them? I feel very angry, because of what I had done, I was a senior staff nurse before I even 'came out of the closet' officially and I told people. I never had it formally assessed at 15yrs old and nobody properly knew what it was. I now think if I had got that far without absolutely no additional assistance made, the struggles I went through in my adolescents, what could I could have done without the traumas, without the negative effect on my self-esteem and being actually helped rather than being a hindrance on the academic staff, I could have done…and been a different person. (Anita, depressed)

Supportive Dyslexic Parents

Were your parents helpful and supportive? My dad just kind of wasn't that helpful with spellings or handwriting and things, he just told me that my handwriting was a mess, and I asked him to spell things, and we now know why he wasn't able to tell me, he would get me to work out what letter it started with to look it up in a dictionary, he obviously as he now realises now, that he didn't know himself, but other than that, any questions I had, if dad could show me how to do things he would do. My mum would get me to read words out from the paper, she said she knew the words but couldn't pronounce them, but I could and [but] I'm sure it was some of the disguise, I'm sure she admits now, was to get me to do the reading, to read stories and stuff. She was supportive, I never felt embarrassed about how I did things. (Emma, depressed).

Parents Helping with Homework

How was homework? I mean, mum would help with homework for me; she used to do it, as I used to run out of time, rather than say to the school that there is a problem, saying 'my child can't keep up'. **Was that a form of covering up for you?** I don't know what mum thought she was doing, but I know my mum still carries a lot of guilt, if she would have done his, if she would that? Mum carries loads of that, more so since I passed my certificate of education (teaching qualification) and they realised that actually I wasn't the 'thick one', its done a lot [of damage], there is quite a lot of ricochet in the family, that actual fact I was the first one [of her children] to go to university and qualify, but I'm the only one to not have any GCSE's [school qualifications]. (Natasha, depressed)

Frustration and Anger

Frustration

Could dyslexia be linked to your triggers? Absolutely, yeh I remember as a child I lay in bed at night just feeling which I can now identify as frustration, it was like a nagging pain in my feet and abdomen which I'm now clenching now thinking about it, unable to sleep, insomnia, I was an insomniac as a child, I did not know what it was but I dreaded school and I knew I did not fit in at all, I was different in someway, I knew I wasn't thick, I didn't have a lesser intelligence than my peers, but just knew I felt alienated in school, particularly in my primary school, that again was reflected in when I started school it was never a problem, at home…I wasn't playing up…I felt so anxious and frightened as a child but couldn't put my foot on it…I was unable to articulate why I behaved like I did at school. (Adrian, Depressed).

Dealing with Intense Frustration

So when you get things wrong and get frustrated, how do you deal with it emotionally? I get very upset indeed, I can be reduced to intense anger and I'm not proud of it but I used to hit my head in intense frustration and will tear things up. **I'm looking to gauge your intense anger, I would imagine you are quite high up with that.** It would arise quite quickly, I would get into that space and it is like someone had just flicked a switch [in me]. (Trixie, depressed)

It Feels so Unfair

Have you ever been or are you now frustrated from your learning difficulties? Yes. You see other people study, they seem to put in half the effort and get better grades, it feels so unfair, I put all the effort in, I manage my time better than them, I've got to plan more, such as I've got to cope with different strategies and stuff, think of it myself before I can commit it to paper, its where most people would just read it, know what they read and then want to put it down [on paper] quite quickly, I have to do more work on what is explained, more….**You just must put in more work?** Yes, I must put more effort in. When someone can read some text and know what it means, not so much learn it and keep it in their memory permanently, but keep it in their short term memory and fire it back and get the grades for that. I must digest it more, I must know more about what is going on in the paper. **How was it copying the blackboard at school?** It would take three times as long, cause I was always looking up, for every word, looking back down and then looking back up again, I can't even concentrate now [on such things]. I can type fast if I know what I'm typing, if it's coming out of me. (Ronnie, depressed).

Anger

Have you ever said why me? Why am I dyslexic? Yes. **Did you ever get angry from it?** Its not often I get angry. **How do you feel when you get things wrong?** I say 'stupid me', especially when I use a spell checker, it flags things up like a great big balloon it shouts it out at you angrily with the letters and lines under it. **How do you react to that?** Frustration. I get frustrated and then anxious and that builds up and then the anxiety kicks off more. **But you have never got angry about it?** No. but I used to….. Have you ever watched 'Star Trek?' the first and original version? The Vulcan 'Spock', I kind of relate to him, because I felt in a way that maybe, he lived in a world of humans, and angry, he used to control it, I know this sounds sad but I kind of related to that. I used that to control my emotions. **You were controlling it within your body; do you know how you managed that?** I don't really know how he did it, he never got angry, and he never lost his temper. (Ronnie, depressed).

Why Should I Bother?

I did try and at times, they just wrote me off. So I asked myself why I should bother. (Anita, depressed)

Labelled as Lazy

Do you think your parents were supportive and helpful with your school homework? I did recon there was something from the start, as my brother is dyslexic, but my mother would not have it as it was on a different level [symptoms seemed different]. I was described as lazy but they said 'she is top of the middle group', I was put down as quite bright and my difficulties were put down to a very disruptive education. **Did your mother believe the teachers more than you, that there was a problem?** They just thought I was lazy; often kids were described as lazy rather than dyslexic. I just accepted the title of 'lazy'. But there were things I loved to do. **The word 'lazy', did it come from the school or your mother?** My mother mainly, and my father. I used to play up a lot. **You said you dreaded secondary school reports.** Yes I think I thought 'oh my god what have they said about me', because I remember the headmistress said I had a 'cuckoo brain', what a thing to tell parents. **Cuckoo**

brain, why do you think she said that? I do not know. **Do you think your teachers saw you as lazy?** It was accepted that I was labelled as lazy, so I became lazy I guess [laugh]. (Karen, depressed)

Why Me

Have you ever said why me, why am I dyslexic'? It is not fair. Yes, I suppose everyone does. When I was younger, it was more of 'why am I dyslexic', we used to, I used to look around at people. No one in my life has called me 'stupid', I never thought I was stupid. However, I used to wonder in class when I tried to do work, I used to look around at my peers and think 'how do you know that? How can you do this and I can't? It was more questioning that than 'why am I dyslexic'. I never really asked 'why am I ugly', it is how you are made. (Izzy, non-depressed).

Avoiding and Being the Class Clown

Avoiding

Do you think you were avoiding things inside of the classroom? Oh gosh, yes, I would never have wanted to read out aloud, I very much tried to keep or hide, into the background with things like that, I just tried me make myself as inconspicuous as possible. (Anita, depressed).

Being the Teachers Helper

I would avoid doing the written side of stuff by being the teacher's helper, helping people. **Really?** Yes, I got out of a lot of work that way. **Do you think it was a form of avoidance?** Yes, definitely. **What else did you do to avoid schoolwork?** I would help people, I would chatter, I would go and take messages and books, and do things, sharpen pencils, anything I could do really. **Everything to avoid writing?** Yes, if I could do on the positives. I would never misbehave or get in to trouble as I would not want to disappoint my parents, but I would do, whatever strategies I could to kind of avoid doing [written] work. (Emma, depressed)

Clever Coping Strategies

Do you think you avoided things at school like reading aloud? Oh yes, I avoided it. I would do anything to not read aloud. The worst thing for me was a foreign language, because they were introducing tape-recorded lessons, oh god…I cannot process my own language, without even considering someone else's. **So what sort of things did you do to get out of reading aloud?** I do not know, faking falling asleep, anything really. When you are put on the spot, you sort of mumble your way through it. I think the teachers realised there was a problem so they tended to avoid me after a while. So they left me out, my turn always seemed to happen at the end of the lesson and that sort of thing. They knew there was a problem, rather than make it an issue they said I would read next time around, then someone else started and I was forgotten about. **How about in the classroom, did you place yourself in the back row so you were not seen?** Its even my coping strategy now, I will always go to the back of any room, so I can see what it going on, it's a visual coping strategy, so I can see

how others are reacting. Yes, I probably did it at school to avoid being seen. **Did you avoid written work?** Not really, it used to be a struggle doing it. I think basically, like many others, I managed to develop coping strategies to get me over the basic bits. I was one of those who took my English Language O'level eight times before passing it [laugh]. **You passed it, so that the main thing. You avoided reading out aloud, you moved to the back of the classroom to be avoided being picked on. Were you also the class clown or played up to be sent out of the room?** Not really, I was too quiet. I was the quiet one in the back. **So you tried to be invisible.** Yes, I tried to avoid being seen, to blend in but also not to be seen making mistakes. (George, depressed).

Getting others to do the Work

Do you think you would ever try to avoid school work, like reading out aloud Yes, at all costs **What work did you try to avoid?** I used to copy other people's homework. Just generally try and avoid stuff, like I was terrible in French. So for the exam I just did not revise. In the end, my mother got me an English tutor to help with my English and the tutor basically wrote all my essays for me. We wrote such detailed notes that in the end my mother was paying her to do my work for GCSE, she [the tutor] knew what she was doing and I knew what she was doing, she was being paid to do it. I also copied my science work, so really I did not do much in the end. But I did the subjects I enjoyed like craft and technology. (Izzy, non-depressed).

Class-Clown

You were talking about getting into trouble at school because you were bored. At the time I was just unhappy really, it was more to get a laugh out of people, I just did not want people to ignore me… a lot of the time,…I do not think I allowed myself to be bored. **So it sounds like you were trying to gain approval from your peers by acting up.** Yes, that is what it was really about. I think most people do. I think people always want to be liked and if people think you are funny, you will do things to get that, 'you would rather people laugh at you for doing stupid things than laugh at you because you are stupid'. After a while people suss you out, plus if people tell you are good you will behave well. If people expect you to be a certain way and they like that, to get a reputation to be like that, you are that. In the beginning, you are acting out to be noticed, but in the end, its how you are or you think you are. It was mainly for people to like me. (Izzy, non-depressed).

Putting Off Homework

At home, how was homework time at home? A battle of wills? I am the best procrastinator in the whole wide world, I would put it off, put it off, then it would be 'in an hour I will do it, then in another hour, or after the TV show' that sort of thing. **So you would try to avoid doing the writing.** Yes, but there were times when you were younger when you had to do it. They would sit down with me. When parents have time, especially with my maths as I could not do them, when they would try and explain it to me, it wasn't her (my mum's) fault but she would do it for me, because your saying that you understand it 'yes, yes, yes', when they ask 'do you know how to do this?', so they do it for you, whilst you are not learning a thing, its getting done anyway. **Do you think you used that often to get your work done?** Yes, but only for maths, as I really could not do it. I'm quite a weird dyslexic, as

I love English, I love doing it, I hated doing essays, so I only did them when I had to, I got used to, quite quickly getting really bad marks for things. So by the time I was sixteen or seventeen I told myself that I did not want to do them anymore (essays), as I just cannot do them. For maths, I was quite prepared for someone else to do the work for me, as I really could not understand it. (Kirsty, depressed).

Physical Symptoms

Did you ever fake being sick to avoid tests? I didn't have to fake it, I was always sick. **Did you make yourself sick?** It was just the nerves [from the tests], I didn't even get as far as the door, I would literally be running up and down to the loo and to a point what even now, examinations and me just don't got on with one another. But yeh its still there [exam nerves], it's not something I can do easily. (Natasha, depressed).

Rebelling in the Classroom

Thinking back to your primary school years, were they happy times? I remember coming downstairs and telling my parents that 'I don't feel well', I think it was a way of trying to get out of school, but my mum worked so there was no option. I went; I had a few fights, confrontations so I walked off. My time at school was not particularly happy, but I was quite a stroppy kid. There was a story that I wanted to go to the loo (toilet) and they said no, so I peed there and then, that was my attitude. **So it sounds like you were contrary.** Yes. **You were cheeky in the class; do you think it was a way of being sent out?** A combination of that and because I couldn't impress my peers with reading and writing. **So you made friends by being cheeky.** Yes. I made friends and found a niche. **A niche status with your peers?** Yes. **Do you think you were rebelling to be expelled?** No. I did not know what expelled was. **So why do you think you were rebelling?** It is this whole authority thing. My time at secondary school was quite complicated. **Were you ever called names by the teachers?** I used to fight with teachers; I was punched in the face by one. I do not remember name-calling. It was French, I did not want to do it, I said something, and he got annoyed. He stood me on a chair, he wacked me in the face, drew blood. **How did you react to that?** I think I went for him. I did, and then I dropped French. The irony of the next school I went to was that they did not teach French at that school. It was the excuse my parents gave to go into a mixed school. It was like 'he cannot do French as he beat the last French teacher up'. I seem to remember it happened when I was twelve years old. **How was secondary school?** What happened at secondary school was the aptitude for using electricity geared in. There were two issues, there was a pain of a kid that teachers didn't want in their class as he just messed about and didn't achieve a lot, me and there was a boy who was very competent with electrical things, me again. You were talking about thirty years ago. **Technology was computer cards and things like that** Well in my school it was projectors, VHS videos, TV's, lighting, school plays, so the most competent person to use this stuff was me, and the person you least wanted to be in the class was also me. So what they did is I had a title of 'Audio Visual Official' and I used to show the films, videos, do the lighting and sound for the plays. I was also made a prefect. **That is alien to normal school rules?** But that is where….I was not bullied, I was not depressed. Because probably when you ask me 'are you happy with your life' and I explained what I did with my business, if you listen back I did exactly the same with my

schooling. And the other positive thing I had was that we were adjacent to a grammar school, the grammar kids went to the right [path] and we went to the left. The work at the school is not all of what you go there for, of course, I remember the geography lessons, but it was secondary to social side and the audiovisual work. Those sixteen millimetre projectors are heavy things, setting them up, all the school plays, the sound. **So do you think your parents realised you weren't learning?** It was peaceful. I think you probably had two options, either you had it all confrontational or you can have this peaceful solution. Lazily going into school, sit in a geography lesson, then going to show a film, show this, do that, see you later. You know so…most of the time I remember I sat in the front due to my reputation. Don't forget you have this kid who was kicking off, who turned into this good boy, helpful good boy, so he didn't disrupt the school day. My parents saw that I was enthusiastic to go to school if I had a film to show. Remember I wanted to be an electrician so it was work experience. (Malcolm, non-depressed).

Truanting and Running Away from Home

Not Truanting but Running Away from Home

Growing up do you ever recall ever truanting or running away from home? I ran away from home, but I never truanted. It is really weird but I came from the family values of 'you have to go to school', no matter what. I'm sure I would have saved myself a lot of pain if I hadn't, but I kind of felt I had to do this and me being stubborn I did. **Do you think you ever faked being sick to avoid tests?** Constantly I did that. **Do you think your parents knew?** Yes, because my mum and dad knew there was stuff going on in the school that I was not telling them about. **Were they happy or okay for you to do that?** No. **Did they allow you to?** Sometimes. If I had worked myself up in a state, physically, then they were more than willing for me to be off school. **You were talking about running away from home. Often?** I planned to do it more than I did. I only went down the street. **I remember having a packed bag under my bed, just in case I couldn't cope and needed to leave fast, how about you?** I also did that, but I was aware that I had nowhere to go. I was aware that it was a dangerous world out there. Where I was running to might have been just as dangerous as where I was running from, and at least if I was at home I was under my mum and dad's protection. I knew they loved me, so it was quite fleeting ideas about running away from home.

Running Away

Growing up were there any occasions of running away, truanting from school or self-harming as a child? I used to truant from school, yes, on several occasions in secondary school, from like being 11yrs old to 16yrs. I [also] ran away from home on two occasions in between those years, I did that because everything was getting on top of me with school and the bullying, struggling [with school work], I was trying to escape. (Samuel, depressed).

Getting Pregnant

Did you get pregnant on purpose to avoid school? I know I did. I knew that the educational system would not allow a female pregnant girl into school in a building with

concrete stairs, even back then I wasn't daft. I couldn't survive school and the only way of getting out was to make sure I stayed out and they couldn't be forced to go back. (Natasha, depressed).

Faking being Ill

Growing up, do you recall ever truanting? Rarely, I did sometimes Do you think you ever truanted to avoid tests or even faking being sick to avoid tests? Yes, probably I did fake being ill to avoid tests sometimes. I think, looking back I hated maths and French tests... (Norman, depressed)

Perfection

How does failing or getting things wrong affect you? Everything has to be perfect; I can't [deal with getting things wrong]. I beat myself up a lot of the time if things are not completely perfect. When I write an assignment now I have to write it word perfect, I will go back, even if it takes me twice as long. It must be word perfect from the start. If I get a bad mark or grade I feel like a complete failure, I pretty much withdraw for up to a week. (Norma, depressed).

Bullying

Bullying by Teachers

Did you feel your teachers were supportive? We had a mixed bag of teachers actually, we had one….we had a mixed class, I think it was one of the first times they did a mixed year, and we had one [teacher] who was the head [master], I kind of….he didn't like me, or it kind of felt that, I think looking back now, because he didn't understand me, he could talk to me and I would answer him, yet when it came to my written work I exasperated him. Yet I had a teacher, another lady, who taught me italics and helped with me with my handwriting and had all the time in the world for me. They would all ask me to do things and help me do things, yet there were some you could see didn't, I didn't want to work with them because they scared me, because I could see they were disappointed. **Did you ever feel picked on by them?** I don't know about picked on, so much as scared by a couple of them, they would be so frustrated that I couldn't understand what they were trying to explain to me. **Were you ever called lazy by your teachers?** [Laugh] um, you know I can't actually remember, I don't think I was called lazy, I think it was the whole 'you need to apply yourself' more. **Were you ever kept in during break times to catch-up?** Yes, to finish off work and stuff. (Emma, depressed)

Bullied by Peers

Please describe your time at school? Was it enjoyable? Not at all enjoyable. I was bullied in the playground and when I went out [socially and to and from school], out and

about. I don't know why they bullied me. In the classroom I felt that teachers didn't understand me, I found it hard to learn and get on, understand what was being taught and so these children made fun of me as well [as I couldn't understand what was required of me]. I was put into a remedial class on several occasions, I felt singled out [by that], it felt like I was being bullied as well [in the classroom/school], [I felt that] people didn't understand me. I wasn't supported at all [for my learning difficulties]. I used to dread my school reports, it was always 'could try better, doesn't try, underachieves, doesn't take care' not taking notice, lack of attention' that sort of thing. My parents reached the conclusion that I wouldn't achieve, I was just an under-achiever, and I would never be as good as my brothers, who were not dyslexic. (Samuel, depressed).

Going into a Bubble and Regression

Bubble

You were talking about closing in growing up, sitting in your room and reading rather than being social, do you think you were doing that as a protective bubble? Yes. You do find, I do that sometimes, a put a protection there, a bubble to keep people away. I do feel very self-conscious, I always feel self-conscious. Some of it is due to my dyspraxia, as I try to look at where the dyspraxia problems combine into my dyslexia. (Norman, depressed).

Sucking your Thumb

Did you ever suck your thumb? [Big laugh] yeh, till about 12 years or older. Definitely a long-term sucker. Could it be comfort thing? Oh yes definitely. Did it allow you to go into your 'comfort and protected' shell? Yes. Did it feel safer there? I suppose it did as I did it for so long. Wetting your bed? Yes, till a late age, I do remember that. All of these show early forms of anxiety about the world; do you think that is true? Yes. You were saying you first felt depressed at 6yrs old? Yes, I never realised that those were signs of anxiety. Yes, they are regressive forms, a return to the womb. Yes I knew sucking thumb was, as also my curling up into a ball as in the foetal position, I like to curl up into bed. (Ronnie, depressed).

Wetting Your Bed

Do you recall wetting your bed until a late age? **I wet myself up to secondary school.** Do you think that was related to feeling nervous about school? **Yes. I used to wet myself at school as well.** So it was the panic then. **Yes. (Kirsty, depressed).**

Why some Dyslexics get Depressed

Depression and Dyslexia

Do you think dyslexia and depression are linked? Oh, yes definitely. With the children, I work with and the people I work with, I definitely think it is. Children I have seen both privately and in the mainstream. **Do you see depressed dyslexic children a lot?** Yes, I do, I see the affects it has on them and how they are down on themselves and how they think they are stupid or thick or they are not proper because [they think] they are not the same as

everybody else. One of the kids I work with has self-harmed, a couple of them have problems eating and I look at my [dyslexic] son and think how lucky I am, he actually copes very well [emotionally said]. (Emma, depressed, NHS Physio-therapist).

Why Dyslexics get Depressed

Do you think you also had similar depressive feelings when you were at school? Yes. At school you just have to do essays, I really couldn't understand why I couldn't do as well as everybody around me, it was going in and I was learning the things they were telling me, but when it came to doing an essay it wasn't coming out and I didn't really understand why, because, it made me look like I was being lazy or stupid, and when you get told that a lot you begin to believe it and say, yes [that's me]. I was not allowed to get anti-depressants then, so just had to ride over it [deal with it]. I was at nineteen years old at college as I wanted to be there, but I also thought since I left school that there would be other ways for me to do things like that (essays).**How young do you think it was when you first felt depressed?** I think it was when I was quite young, at primary school. I was always aware that I could not do things that other people could but did not know why, so it kind of ostracised you. You kind of thought 'well everybody else can do it'. I had problems communicating with people and describing how I felt, so could sound a bit 'stand-offish' (stubborn and blunt). So all through school, like at five or six years old, my ability to be part of a group made me feel excluded, made me an 'other', that sort of thing really hurt and I didn't know why, that other people could do things and I couldn't. I felt strange, as I could not communicate or I would take literally what someone said, I would get upset with people taking a certain tone of voice at me. (Kirsty, depressed)

Internalising

Do you think dyslexia and depression could be linked? Yes, I think they probably are. I think it is something connected with stress along with identity and self-esteem, but that you take on board a lot of feelings and messages; well at least I did growing up. You tend to internalise things, as a lot of the messages are to do with blame and blaming yourself, as 'you're not doing something' or 'you are not putting in enough effort', as you do tend to internalise these things. (Norman, depressed).

Rejected by Society (Positive or Negative)

Do you think dyslexia and depression are linked? I think dyslexia is a man-made problem and so depression is a result of those man-made problems. I think the depression comes from others not accepting the dyslexia. I think if they did, I would be happy coping with my dyslexia/APD, that's not a problem for me, in fact I see it as a bonus as it allows me to do things that other people can't do. But because other people are not prepared to see that, then I get depressed, as I am not allowed to be me. **So you see the depression as a secondary side of how society handles you and perceives you.** Yes. **So this is the core problem?** I do not see it as a problem so much. I know it is a problem [laugh]. If people would be willing to understand my and other people's problems more readily, we would not be in the problems we are now in. (George, depressed)

Self-Harm and Suicide

Anorexia

Have you ever said 'why me, why am I the one with the problems'? Yes, you look at everyone, you think if everybody else can do, so there must be something wrong with me. If you can't do this and they all can do it, you then look at yourself and think 'they must be right about me, I must be thick, I must be stupid, as I can't do what everybody else can do' obviously that must make you think 'it must be me'. **Have you ever self-harmed as a reaction to these frustrations, feeling low or not feeling worthy?** Yes, I have been pretty much anorexic for most of my life, I'm now only six and a half stone [as an adult] and I'm five foot high, so I'm under-weight. I guess that was self-harming. I did cutting a few times but quite frankly I'm not that brave, so the sight of blood kinds of freaks me out, so I understand the process of it as it's a kind of release, when everything in this world you can't control but this thing you can, with me it was my eating, I can control what I put into my mouth or I can control how much blood I can spill – I can understand that that. I would be lying if I said I did not understand why I did these things. **How young when you first controlled your food intake?** Pretty young as I have always had problems with food as I've always been tiny, I think at primary school I wasn't putting on weight as I wasn't eating. I was always delicate looking and quite small. **Do you think back then it was about control or about attention seeking?** I do not think it was for attention, as all the attention you got was negative, not from your parents. From my parents I always thought I disappointed them, as obviously I couldn't do what everybody else could and that it was my fault, I wasn't the perfect child that I should have been, no matter how many times your parents tell you they love you, in the end you don't believe it, as everybody else says you are different. I did not do it for attention, there are lots of layers [clothes] you can put on, and so no one knew. When you are small, you do not get a lot of attention, If I was not getting any attention from my peers, then I was getting negative attention from them in the form of bullying, so maybe I just wanted people to notice me, notice the pain I was going through **Have you ever thought about or tried to commit suicide?** Yes, but like the blood thing, I am really, really, really not brave…so I do not know if I could have gone through with it. There are loads of times when you think about it, when you plan it, but I do not know if I could go through with it. **How young do you think you were when these thoughts first came into your mind?** Probably quite young, although I wouldn't see death as a way out, because I didn't have that concept, I just wanted this to stop, the inability to do everyday things and you just think 'I want to be like everybody else, I want to be normal and I'm not allowed to be normal', so you want it to stop, to fall into a hole and not be there anymore, as obviously your not contributing anything to the community around you as your not doing it right. So obviously, if you are not doing it right you are not being productive and you are not worth anything. (Kirsty, depressed).

Cutting

Do/Did you self-harm? Why? What are the triggers? Yes. I do still self harm and have done since about the age of four. My triggers are anger and frustration. I cut my arms and belly. As a kid I would scratch my hands or bang parts of my body like my head. I am currently having therapy which is addressing this issue. **Have you ever thought about or**

tried to commit suicide? Why? What were the triggers? Yes. I took an overdose this summer and more recently drank so much I ended up tearing my oesophagus. These were both very scary occasions. I get very low sometimes and this is what triggers my negative thoughts. **Do you think dyslexia and depression are correlated (linked)?** Maybe. (Susan, depressed).

Have you ever blamed your learning difficulties for things? Yes I did. Have you ever self-harmed yourself because of your difficulties? Yes. Was that with food, drink or something else? I used to self-harm, I slashed my wrists open and stuff. I used to eat loads of food and [do] all sorts [of things] really. Did you ever attempt suicide? Not as such. So when you slashed your wrists, how old were you? About 13yrs old. Do you know what the triggers were for that? It was depression. Could it be linked to anything related to the school, like tests? No .Bullying? I suppose you could link it with the bullying. Do you still have those thoughts? Yes. Do you know what triggers that now? Just a lot of stress, which is pretty much my really big trigger. (Jean, non-depressed *see note at the start of the results section).

Alcohol and Binge-Eating

Have you ever self-harmed due to the frustrations that dyslexia brings? Drugs? I do drink too much. **Do you drink to drown the pain?** Yes, it gets me out of, out of it all really. It is like…it helps you sleep so you are not up all night worrying, going over things sixty times. **So it slows down your brain so it allows you to sleep, it numbs out the fears and self anger.** Yes **How about with food, self-harm can be comfort eating?** Yes, I over-eat [laugh].**Do you think as a child you used food as a comfort?** Yes, I always have **Do you think it is form of control?** [Pause] I suppose yes. (Ronnie, depressed).

Planning Suicide

Personally, I had those thoughts when I was 8yrs old and felt it would be easier if I wasn't here, a burden on my family. During my school years, I would quite often wish I had never been born, but actually I do remember as an early teenager kind of not wanting to wait till I could drive, because I couldn't wait to get into a car and just drive it into a brick wall. **Why the car?** Unsure but I wanted to do that so it would all be over, dealing with it all. (Emma, depressed, NHS Physiotherapist)

Over-Dosing

Please describe your time at school? Was it enjoyable? I quite enjoyed school until I went to secondary school, then I found it really difficult, I wasn't diagnosed dyslexic till I was 17yrs old I think when, no, when I was diagnosed at university, I was told I was probably dyslexic when I was 16-17yrs but wasn't diagnosed till I was at university a few years later. I used to hate school because I couldn't understand why I couldn't do things that others could do. I used to hate school and now I teach in them. I'm a very vocational person and I went to a very academic school, judging by results, it was a technical college and it was full of technology which is definitely something I'm not good at. My primary school was fine, it was just secondary school that things weren't good, I took an overdose when I was 14yrs I just couldn't cope with it, it was an academic school I was made to take 13 GCSEs which was far too much work. A lot of coursework, I just felt there was too much pressure, I just can't work under so much pressure, some types yes and I can work under deadlines [now], but I can't do

it if I have too many things on and doing 13 GCSEs, well I just couldn't do it. There was always too much to do and it takes so much longer to do it as well [due to the unidentified dyslexia]. They expected me to go in during my holiday to catch-up! My overdose was due to school, I didn't know what to do, and I couldn't tell anyone I couldn't cope with the work [load]. They never sent me for a dyslexia assessment. I was only diagnosed at college. They never picked-up on it at school, in fact rather than put me into remedial classes for my learning difficulties, I was put into higher classes because, I don't know, it was mainly the coursework I struggled with, apparently I was above average intelligence or average intelligence, it wasn't I couldn't start the work, I just couldn't understand it or in the maths, I would see one question and I could do it, but when it came to the next one [question] being the same type I just couldn't do it. At school, because I didn't know I was dyslexic I just felt stupid and I got accused of attention-seeking and being difficult because I just couldn't do the work, obviously I now know I'm dyslexic but at the time it felt everyone was out to get me [pressuring me]. Back then to cope I self-harmed, I just didn't know how to cope with it. I didn't really avoid writing, okay some of the time, I would go through phases, so particularly at year 10 I was bordering the average [standard] I could, and by year 11 it got better. I'm still like that now. My time at primary school was better than at secondary school. I think I still struggled at primary school, but I think when I went to secondary school, it was a very academic school where there was such pressure to, and there was no concept of 'you can't do it'. I suffered a bit from bullying at school, it was everywhere really [playground and socially] but it was really linked into my depression, I just didn't interact with anybody and then someone found out I had been in hospital [for my suicide attempt] and then it was tough, it became public knowledge. I think they saw me as easy prey/easy target. It never really bothered me. (Lara, depressed).

Post Traumatic Stress Disorder

With your child, how do you feel going into schools? Are you affected by it? Yes. The smell of the school, I hate it, the small chairs, and the paintings in the corridors. No disrespect to the artists, but it reminds me of being outside the headmaster's office, in trouble again. (Adrian, Depressed).

How do you feel going into schools now, do you ever need to? No. I get crabby and hot and I don't like it at all. **Is it the smell, layout?** Its not anything in particular, I just hate it. I now really suffer real problems with my nerves; I have to be in control. I get carsick and motion sickness, I have to be in control of the situation. (Jean, non-depressed)

How are at going into schools now, is it difficult? Yes, as all my old memories come back. Because you do not have any great memories of school, so you sort of travel back to your own days at school. **What is it triggering that? The smell, layout, the chairs, the teachers?** It is just that when you walk through the doors you feel like a different person, you kind of feel like that again [a small child]. You forget that you are twenty-three-years old and have done all these things. You think that you are twelve or thirteen again and you have to face a school where no one likes you. **However, the smell doesn't trigger things?** It can do. You get the sense of being there again. **Noise of the children?** Its everything, you just feel your back there drowning again. (Kirsty, depressed)

Feeling Inferior

How is it going into school now for your children? I work in social services and some of my foster kids have gone to my old school. It was really weird going into there, as when I first started, the same teachers were around and I felt strange going in there, especially when one of the came up to me and talked to me as he remembered me. I remember thinking 'you stand there being so nice to me and the traumas that you're participated in'. **Did you feel belittled by them? Inferior?** I am having an attack just thinking about it now. I did not feel that as an adult, but I did as a child. I felt very angry towards them and I am very much of the thought that one should not be two faced. **Did the smell or layout trigger things for you?** When I walked in there, I felt tense and I sort of panicked as I began to look a round for people who would know me. I began to get flashbacks to my time at the school and the traumas, being told off and being belittled. (Anita, Depressed).

Crying

I know that you get annoyed going into schools now for your children, are there any triggers there? Smell **Layout of chairs?** Anything really. I am a school parent-governor, so that is not so bad, but I must have mature conversations with the head master, but saying that, it is a different environment to my own school. When I go to my daughter's primary school, it is the smell, the chair layout, chair scraping, and noise from the kids. The other thing is my eldest son is at a new school, newly built so being a governor is okay. But my daughter's primary school is old; it has the old look, old smell, old chairs etc. **How about dealing with the teachers, do you have this old resentment towards them?** I cry. **Cry?** Yes as I can see everything, I went through, as a child, which I am still fighting against. I am getting better. **Do you think your wife would prefer not taking you with her to teacher meetings?** No. As she knows that I know what I am talking about. Subject to work commitments, I go, but she is as passionate about it as I am. She does not have dyslexia, but she has seen it, she knows. **Do you feel you know more about dyslexia than the teachers?** Yes. **How do the teachers react to you knowing more about it than them?** [Pause] each teacher is different and some actually come out and say they are dyslexic as well; some however are too stupid to deal with it. **Do some get defensive, the more you get angry it?** Oh yes, yes. (Malcolm, non-depressed *see note at the start of the results section)

DISCUSSION

The results suggest that twelve themes came out of the analysis, although only one quote per theme/sub-theme has been listed in the results section, due to space. These will now be discussed in more detail.

What is Dyslexia?

The body of evidence from this study suggests that dyslexia is perceived by those with it, as something akin to faulty wiring within the brain. They think their brains are not working as they should do and compared to their peers, they just can't compete in the classroom. Whilst

many thought it was negative as a child, as an adult or as late teenager they note the advantages that dyslexia brings to their lives. As adults they have awareness that they are different and can do different things.

The evidence suggests that three definitions of disability are evident (Kaplan, 2008). Firstly the 'medical' definition that dyslexia is a fault within their body that needs to be fixed. The second is the 'rehabilitation' definition that comes to the fore in that until such a fix is made with medical intervention they are not a complete person without the medical fix. The third is the 'disability' definition is that dyslexia is a difference within society and that society is made up of different people and that dyslexics are just one type.

Evidence that all three exist, sometimes within the same person suggests that whilst they may accept their dyslexia as something positive, those around them do not and that they are fighting within themselves and at work for their self-worth. This leads onto the finding that dyslexia is believed to be something man-made. It isn't the dyslexia which is disabling them, but the reaction of others to their dyslexia which is. They note that they frequently experience misunderstanding of their difficulties and that allowances are not being made for them to do their jobs or to develop their careers as per their peers. One participant noted that an interviewer for a vacancy she applied for openly said 'if it wasn't for you ticking the dyslexia box on the application form, I wouldn't have interviewed you'. Is this positive discrimination? Is it helpful? Discrimination legislation in the US and in the UK suggest that strides are being made and that some form of protection is being put into place, but is it enough and is it working? Whilst not noted in the results section of this chapter, adults in the wider study note that before allowances can be made via discrimination legislation, they need to disclose their dyslexia to their employee. Evidence suggests that this is fraught with difficulties with one participant in this study feeling they were bullied by their boss to then disclose this information to their colleagues. Another felt that coming out with their dyslexia could be compared to 'coming' out as gay, to work colleagues, as it shared many of the same concepts of being different and abnormal.

For a child, as soon as are diagnosed as dyslexic in school, you are labelled as such and can be treated differently to your peers. In the 1970's many UK schools were of the opinion that you do not give a child a label as this label would be with them for the rest of their lives. Dyslexia was perceived back then to be an abnormality by the public at large. Today the label is more acceptable and may be more advantageous to have; in fact in some circles it is perceived as socially advantageous for certain careers such as art and design. However this chapter is about children, and it is clear that being dyslexic in mainstream education is disadvantageous, especially for those who are undiagnosed and unrecognised as struggling due to the condition. It is not just a case of being short-sighted and needing glasses, it is a more of an issue and much harder to remedy.

Difference

Difference came out to be a huge issue with the dyslexics in this study; they felt that they were different and that this affected their self-esteem and self-concept. At school, especially primary school, difference is not always rewarded. Children want to be 'normal' like their peers, but what is normal? This huge concept is difficult to assess as it is dependant on the society and environment they are in. My favourite example of this is in the film 'One flew

over the Cuckoo nest', where Jack Nicolson was put into a mental asylum and rebelled against the nurses and doctors. He managed to convince the other patients that they were in fact the normal ones and that the nurses were the abnormal ones in their micro-society. It is the environment and majority who decide what is normal, in school normality is firstly defined by the teacher and secondly by the children. Normality is having the 'in' or 'cool' trainers, clothes or being able to play football. Children want to be normal, to be accepted. So anything that gets in the way of this can create stress in those who are deemed to be outside of the normality circle. The evidence in this study suggests that very early young dyslexics notice that they are different, they learn differently, they aren't able to read and write as their peers, in short that they are lacking. Teachers also early on label children as either normal or outside of normality, and will place them in appropriate group learning tables in classes, to be helped by more able classmates. Whilst the research found that teachers are generally supportive, they are unaware of the core difficulty due to lack of confidence in screening for dyslexia, which is due to either inadequate initial teacher training or inadequate continuous professional development training.

The research found that starting in secondary school and possibly through experiencing puberty, children begin to want to be different and dyslexia is one of the factors that allows them to celebrate such a difference. The unique learning style is a difference which can either be celebrated to raise self-esteem or hidden to lower self-esteem. Interestingly dyslexics in this study felt that their difference was akin to being from outer space and similarities with sci-fi, aliens and superheroes were made to describe how their difference was a strength but it separated them from their peers. They felt such a difference defined them as to who they were, but is this a reaction? Which came first? Their dyslexia or their personality? So is their personality a by product of their disability and the hardship they perceive they have faced?

One of the questions posed was 'did you know many dyslexics growing up?' in the majority of cases they didn't know any or many dyslexics amongst their peers or family. Thus they had little concept whether they were normal. They were similar to the 'Spock' character from 'Star Trek', an alien being among humans.

Not only at school, but in the home the dyslexic growing up can see differences. Siblings and parents who are not dyslexic can result in the dyslexic being made to feel even more of an outsider amongst the people whom he or she should be closest to. If your parents can't relate to you what hope have you? It is normal for children to be compared with peers in school using school grades, but at home parents compare school reports and sibling comparison is rife. It is not unusual for a dyslexic to be unfairly compared to their non-dyslexic sibling 'why can't you be like your brother David?' A large part of the problem comes from parents, like teachers being unable to recognise dyslexia. Parents are frustrated by their own efforts helping them with homework, thus they can see that something isn't right and labelling them 'lazy' doesn't help, but is a natural knee-jerk reaction.

All of this makes the dyslexic feel unwelcome at home and at least two participants noted that they felt they were adopted, with one making 'help' signs from their bedroom windows as they felt totally alien in their own family. They felt they didn't fit in. Several ran away from home and this will be covered in the 'Truanting' section, but it should be noted from the evidence that unhappiness, helplessness at home and school means that dyslexics will turn to extreme measures to either gain the attention they feel they need or will withdraw and internalise their anxieties and frustrations.

Where they are diagnosed early (as dyslexic), which is a positive step, the labelling is not always welcome as noted in this study. The labelling can be a hindrance if the diagnosis does not result in positive teaching interventions in the classroom, thus the positivity of diagnosis turns into a negative 'poison sword or chalice' if it limits choice in the curriculum.

Humiliation, Schools and Teachers

The research goes on to note that undiagnosed dyslexics can suffer from humiliation at school. This would normally start in the classroom by teachers asking dyslexics to read, when they are unable to do this as per their peers they feel embarrassed and humiliated (e.g. reading the wrong word out aloud from a book). Where the teacher does not deal with this difficulty the child will then see the classroom environment as one that is to be feared. This aspect was one of the most frequent experiences of dyslexics in this study, and the main blame was put with teachers for not understanding their problems with learning.

Part of the problem comes with the dyslexic being orally intelligent, so in class discussions the dyslexic will shine. Thus the teacher is unable to understand why this intelligent child is unable or unwilling to transfer to paper. Many parents are proactive when they see their child failing to learn within normal levels. They ask friends, the teachers and family what they should do. With the school slow to action, based often on a wait and see attitude due to very limited SEN (special educational needs) budgets, many parents pay for private educational psychologist assessments for dyslexia, thinking that this means at last that their child will be helped in school to learn, but unfortunately when informing schools of a private diagnosis they face hostility. This is due to parents trying to bypass the special educational needs procedure maintained by the school and local educational authority (as is the case in the UK). Many are faced with hostility as a private diagnosis is seen as biased, as it was paid for by the parents. However, more recently in the UK such a diagnosis can now be used as evidence to gain extra help and can form part of the procedure for assessment of special educational needs for a child, but will not replace a formal diagnosis paid by the school. So the private assessment puts pressure on the school to start action.

There is evidence that the teachers and head teachers still are suspect about dyslexia and don't understand the condition and its affects on learning. There is generally an incorrect perception that dyslexia only affects reading and writing, but its affects are much broader and touches every aspect of learning in every subject of the curriculum. Whilst this is down to inadequate teacher training, it is their inaction due to school budget restrictions which delays diagnosis and screening programs. It is easier to deny there is a problem and exclude children from national examinations than it is to spend money from limited budgets to buy in external help. In the majority of cases they try and help within existing resources, even though it may be inadequate and piecemeal.

In this study we see instances of teachers covering up for children's abilities to do school work by faking work for public presentation and parents covering up for their child's difficulties by doing their homework for them, rather than owning up to the school that their child isn't able to keep up. In the case of the parents it's 'pride', with the school it is due to a lack of training. It is this covering up which denies the dyslexic child the help they badly need.

Whilst this may put parents, schools and teachers in a bad light, teacher training is changing and many dyslexics do experience good teaching practices, with teachers trying different methods with dyslexics, however much of this has been done without specific dyslexic training and so the methods used may not be the most effective available. Whilst dyslexics may encounter some good teachers, it is more common for them to receive teaching which is perceived as traumatic, with the vast majority of participants in the study resenting their school teachers for the 'traumas they caused' and this subject will be discussed later with the evidence of post traumatic stress disorder.

Frustration and Anger

As a result of feeling different and the humiliation that dyslexics feel from school, frustration has been a common feature in this study. The participants have noted different ways they have manifested their frustrations ranging from nagging pains in feet, insomnia and dreading school, to intense anger leading to hitting ones own head against walls to try and get it to work. The reactions of being told you are lazy, even though you have spent hours trying to do the work is intensely frustrating and getting told your work is worthless is enough for some to turn round and give up on learning altogether. Again there are references to being like 'Spock' (a character in the television program 'Star Trek') where he was able to control his anger at those around him, a dyslexic growing up deals with intense emotions from dealing with daily school tasks such as reading and writing.

The problem comes from teachers misunderstanding how long it takes a dyslexic to do certain tasks, such as being unable to read a book and retain knowledge long enough to process and write an essay, or even remembering what the last paragraph they read meant. Again it comes down to teachers not understanding the students in their care.

Many dyslexics I have encountered in this study have asked themselves at some point in their lives 'why me?' why are they the ones with the problems, why can't they be normal and learn normally like their friends and peers? There is an intense feeling that life is not fair and they are helpless to do anything about it.

Avoiding and being the Class Clown

So far we know dyslexic children feel different, humiliated, frustrated and angry with their difficulties. However they must legally still go to school, they must still go to a place where they feel threatened on a daily basis and where they feel like an alien, as no one understands them. So how do they cope?

Avoidance was the most common coping strategy amongst this sample and other studies (Alexander-Passe, 2004a, b, 2006, 2007, 2008, in press-1; Riddick, 1996; Hales, 2001, 2004; Edwards, 1994). It is avoidance in all tasks to do with reading and writing, which is the basic tool of learning in mainstream education. Whilst the dyslexic will shine orally in class, they will look for any means possible to avoiding reading aloud. Evidence in this study suggests they will take a message for the teacher, sharpen pencils etc. to avoid hands on learning, but it is evidence of them hiding in the classroom by sitting in the back rows or avoiding putting up their hands in class which is most disturbing. It is the withdrawal from interactive learning in

the classroom which is the most damaging. They are hiding from their disability for fear of being humiliated in front of their peers.

Evidence suggests that boys will tend to act up in class to gain attention from their peers and teachers; they are less willing to be passive learners. They get bored in class and as they lack peer approval from their academic results they aim for peer approval from being the class clown. If they don't achieve the help they feel they need, they will then aim to be sent out of the class, again to avoid certain tasks like reading out loud or taking a test. Girls on the other hand tend to avoid being noticed in class.

But what happens when avoiding in class is not enough? How do you avoid feared tests where you might be humiliated? Participants in this study either developed physical symptoms of feeling sick due to the panic they were feeling internally or in more cases they would fake being ill to avoid lessons or tests. This aspect will be discussed further in the section on 'Truancy'.

When it came to homework there seemed to be evidence that homework that required reading and writing was put off time and time again until either they ran out of time or they weren't able to avoid it any more. However one participant noted that they panicked as soon as they got a writing assignment and began work straight away otherwise they thought they wouldn't have any chance in finishing it. It is natural to avoid tasks which are painful and frustrating and if at home, it is easy to get distracted by television and other hobbies rather than do homework. One participant had an interesting concept about homework, h felt that the only person homework tested was the teacher, as it was a gauge to whether they had done a good job or not in teaching their subject, so he didn't do any at secondary school. The same participant found their school had a novel way to deal with his rebelling. He was found to be highly useful as an electrician and was given the job title of 'Audio Visual Official'. He used to go round the school showing films and videos, along with setting up lighting stages for school plays and other activities. The school allowed him to miss lessons on an unofficial basis to show these films which meant he looked forward to going to school rather than rebelling in the classroom, however this was to the detriment of his education. Was it the right choice made by the school? As an adult he now runs his own electrician business, so views his schooling as on the job training. However he is still has negative and emotional memories of his school days.

Rebelling in the Classroom

The story so far describes unidentified dyslexics at school as feeling different to their peers, getting frustrated at not being normal and learning normally. They feel humiliated in the classroom and this causes frustration, anxiety and anger which could be taken internally or displayed externally. To cope with their daily school life they find avoidance of reading and writing tasks allows them to maintain their self-esteem. If they are not being helped in the classroom by their teachers (and in some cases they are humiliated by their teachers) they will begin to turn off academically. If this happens they ask themselves why they are going to school each day.

Interestingly, whilst overall the sample found school humiliating and frustrating there was no major evidence of truanting. There was more evidence of careful subject choices to

reduce negative attention of their difficulties and to reduce their reading and writing workload.

Other means at their disposal was to be a nuisance in the classroom to be sent out of class for tests which could be classed as rebelling in the classroom or being the class clown. As noted in the evidence it was more about dealing with the boredom in class, than being nasty towards a teacher. Dyslexia frustrates teachers as it shows up their inability to teach children in their care, which is why it was noted earlier that teachers had faked students work rather than show nothing to highlight their own insecurities about their teaching abilities.

Teaching dyslexics foreign languages was noted by several as a bone of contention, with many noting that it was illogical that their the school was teaching them French or German when they were having enough problems with their own language. The frustration of one French teacher led to them punching one dyslexic in the face, from this sample. Whilst such teaching methods are abhorrent, this example does highlight how frustrated teachers can get in extreme cases towards dyslexic children.

Truanting and Running away from Home

Truanting did not feature in the dyslexics in this study, as there seemed to be more fear of parents finding out than the actual act. This seemed to be a common theme suggesting that dyslexics are more akin to 'school phobics' than 'truants'. They were in a dilemma that they felt they should go to school but they found it emotionally painful and given the choice they would not go.

In the sample it was more common for dyslexics to fake being ill to avoid going to school, especially for days where there were dreaded tests. In the majority of cases parents did not put two and two together and work out why their child was ill on certain days or in fewer cases were aware of their child's difficulties with tests and turned a blind eye. One participant even knew the only way to avoid school long-term was to get pregnant, so she set out to do that before the age of fourteen or fifteen (this girl later achieved a degree and is now a teacher, thus such avoidance was not due to low intelligence but unsuitable teaching methods).

As noted earlier, where dyslexics feel home and school environments are hostile (e.g. the 'help' signs in bedroom windows) and they feel misunderstood there, they can look to run away from home. Such an activity commonly found in this suggests that parents were unable to understand their child's learning difficulties, even when symptoms of early dyslexia are easy to see (e.g. being clumsy, getting left and right mixed up, inability to tie shoe-laces, short term memory loss). Several mentioned having a packed bag in their bedrooms ready for them if they couldn't face it anymore, suggesting the planning of such action This suggests a long-term problem they felt they would never be able to overcome.

Perfection

Perfection was found by several in this sample as a means to control negative academic feedback. They would draft and redraft their work so much to try and avoid bad marks that they would be exhausted. Whilst such a strategy might be suitable for primary school

homework, when the demands of secondary school appeared, they were pushed to the limits to the detriment of their health, with one taking an overdose of tablets as they felt they had no one to talk to about their problems and school workload.

When dyslexics who have spent hours and hours doing a piece of homework (that many of their peers could do in half an hour) and it comes back with red lines through it the effect can be devastating and the effects of feeling a failure can take days or longer to deal with.

Bullying

As noted earlier, dyslexics feel humiliated in the classroom. Any public show of their abilities can cause anxiety and frustration. It is no surprise that many dyslexics feel the teachers who misunderstand them are hostile to their efforts, in many cases this was perceived to be by bullying from their teachers. The frustrated teacher can often openly air their frustration in class, both verbally and in actions which can easily be picked up by the other students in the class. So when a teacher tells a dyslexic or any child that they are lazy, stupid or thick, then it is probable that they will then be ridiculed in the playground with the same words. The dyslexic who commonly suffers low self-esteem, withdrawal and a poor self-image, turn out to be easy targets for playground bullies.

Going into a Bubble and Regression

Already there is a pattern forming as to how children at school cope, when they are undiagnosed dyslexics or are diagnosed but receive unsuitable help at school. They feel frustrated, anxious and different. To protect themselves they try and avoid all tasks that would show themselves up as lacking. However, that leaves them possibly emotionally damaged on a daily basis.

The evidence in this study suggests that many turn to a place of safety; this might be their bedroom or some emotional bubble where they feel safe. This could take the form of daydreaming in class or sucking a thumb or finger. Each gives comfort at times of stress and anxiety.

The concept of a safe bubble comes from Bendelow and Williams (1998) and is to do with a return to the safe comfort of the womb, in that they feel protected from harm in this place or activity of safety. Sucking your thumb or a finger is a classic sign of regressive behaviour (Vaillant, 1992) and indicates a return to infant behaviour, or a time when they relied upon their mother for everything. It is common for children to do this until approximately six to nine years old, depending on the child, after that time it begins to be a concern as the child is not developing properly to deal with life's stresses. The sucking of a thumb or finger is about helplessness (Seligman, 1975, 1991) and the feelings that they are unable to deal with the emotions and stresses of the world around them.

Bed-wetting is also a sign of regressive behaviours and is another classic symptom of helplessness. It most commonly happens during the night when the child replays their panic from their day in their dreams, but it can happen during the day as well, as found in this study. The wetting during the day came from sheer panic at the idea of having a test. However it might be another avoidance technique to instead them having to take the test and face the

public humiliation of failure. As noted in this study, participants would rather act out and do things to look stupid than be seen to be stupid from getting a test wrong, this may also be the case with wetting yourself before a test. Scott (2004, p. 258) notes that with dyslexics she has found 'sucking of fingers and clothing, curling into balls under desks, bed-wetting and soiling, rocking, holding of genitals and the constant company of furry toys'.

Why some Dyslexics get Depressed

This is the heart of the study. Do dyslexics get depressed and what causes them to get depressed? Why are some dyslexics able to remain resilient to depression whilst others are not?

The evidence from this study found that depression and dyslexia are correlated and that from the sample of N=29, N=21 were depressed as defined by either clinical diagnosis or by two or more courses of SSRIs (e.g. anti-depressants) by their physician or doctor. Evidence suggests that they recognise that their depression was evident as children and some even note from ages as early as seven or eight years old that they felt a similar feeling, but were unable to do anything about it, they just felt this was how a normal person felt. The evidence also suggests that the frequency of females with depression was higher than males, N=15 compared to N=6.

The question needs to be asked why females are more susceptible to depression. Earlier it was noted that in the classroom, dyslexic males tend to act out their frustrations by being the class-clown or disruptive, whereas dyslexic females tended to hide in class and this could be classed as withdrawal, such withdrawal along with isolation of having fewer or no friends could be the roots of depression.

So how does the dyslexic child cope? Depression is a collection of symptoms which suggests a helplessness to cope with their environment and is normally termed as lasting for at least two weeks. In the case of a dyslexic child it could be the majority of their school life, which amounts to at least ten years of feeling isolated, different and abnormal

The evidence presented concerning dyslexia and depression suggests that children with dyslexia get depressed and a main cause could be lack of understanding by teachers, peers and parents. Therefore they feel isolated and misunderstood. The Sci fi concept of feeling life 'Spock' or 'Doctor Who' is a useful analogy to understand how they feel. One participant felt they were an experiment from outer space, in that they felt like aliens, without the language or skills to be a complete member of society.

The pressure to perform as per siblings or peers is a huge burden for children, even more so with those unable to cope with the workload given at school. In the majority of cases they ask themselves 'why me, why do I have these problems?' it is a difficult question to answer even for a parent. Again we return to the three definitions of disability (Kaplan, 2008) which suggests that being different means being abnormal and needs to be fixed, rather than society celebrating difference. In schools, difference is not celebrated and therefore there is a huge pressure to conform to standards of ability. Such pressure to conform means that children who are either above or below this normal level are disabled by the system. Imagine forcing a bright child, in fact a chess genius to sit through a class according to his age to do basic algebra. He would get bored and turn off thinking 'I can do this standing on my head, I can do so much more' and gets frustrated. Well the same happens with the dyslexic who can't keep

up and the teacher carries on without caring that they can't keep up. Again they will turn off and get disillusioned. In both cases, the child sees the problem being themselves, so they internalize the feelings and begin to self-blame. They feel rejected by society as their abilities or disabilities are being ignored, in other words they are not feeling valued by those around them, and this can include parents as well as they will at first believe the professional educator (their child's teachers) that their child is being lazy and not paying attention.

Self-Harm and Suicide

What do dyslexic children do when they feel unloved, alienated at home and at school, and frustrated by difficulties that they don't understand? Evidence suggests they self-blame thinking 'why am I like this, why can't I be normal'. To begin with they try their best to achieve at school to please both their parents and teachers but when they don't gain the results they feel their effort deserves, they begin to feel helpless and under valued.

These feelings make them vulnerable and their self-esteem can drop lower with failure after failure. They ask themselves what choice do they have, can they empower themselves in this hopeless situation? What control can they regain?

Evidence in this study suggests that anorexia, cutting, alcohol and food abuse are means by which dyslexic children can control themselves. The questions posed asked if they were doing this to gain attention as any attention even negative attention is a means they could feel to get people to listen to their pain. It should be noted that all of these are cries for help, after they have exhausted other means to gain help. They are extreme measures where they feel they have no hope left.

What is anorexia and why is it attractive to dyslexics? Anorexia according to the evidence included in this study was a means to control their body. Was it a means to punish their body, as many dyslexics view their brains as having faulty wiring? One participant used to bang his head against walls, could this have been to try and get their brains to work properly, in the same way you would bang a toy to get it to work, or was it frustration and self-harm in revenge? The participant with anorexia said it was not to get attention as she would wear layers to disguise her weight loss, so was it therefore self-punishment and bodily control? As she also admitted that she avoided being noticed in class to avoid reading out aloud, to be invisible, so an alternative hypothesis could be that she was trying to reduce her size to be even more invisible.

What is cutting and why is it attractive to dyslexics? Again this carries on the theme of self-control, controlling how much blood you spill. Cutting is also a means to cause pain to oneself for things they perceive are their fault, as a form of self-blame. In the dyslexic it could be that they perceive that their body is not working properly, or not working as their peers bodies do.

Binge eating is related to comfort eating, a means to reduce stress as food is commonly seen as a reward in children. Sweet foods like chocolate raise the body's blood sugar and trigger a chemical reaction to calm the body. Binge eating is a faulty and uncontrollable means to rebalance self-esteem and treats the symptoms rather than cause. Sugar (methylanthines) cravings can be as powerful as drug addiction cravings, with sugar being more easily available and legal. The secondary side to binge eating is a conscious attempt to

change body size, to put off people from getting close to them, along with a conscious attempt to reject society and society's values.

What is alcohol and drug abuse and why is it attractive to dyslexics? One participant in this study noted that when they were drunk, they didn't feel dyslexic anymore and that they used drink to numb the pain and frustrations they felt. Another said that they used to drink when their parents went to school for parents evenings, when he was in primary school. What does this tell us? It suggests that such abuse is to gain an out of body experience which removes them from their current painful and frustrating life. They feel there is a need to take risks to gain such an experience, a 'time-out' from their normal lives.

No two dyslexics have similar combination of difficulties (Miles & Miles, 1999), thus no two dyslexics deal with the emotional side of their difficulties the same. Whilst some may self-harm, others wish to rid the world of themselves and their burden to others, especially family who they care for. This is therefore in the form of suicide or attempting suicide. Such a reaction to their emotions suggests that they have no way out of their daily hell and feel they just want out.

According to this study, such attempts are planned and come after they perceive that all other options have been exhausted. Evidence suggests that they wished they had never been born and they looked for ways to get out of this life. Children may not be able to vocalise how they feel and related planning suicide as 'I just wanted to jump into a black hole'. Others planning to learn to drive to 'just drive into a brick wall'. When they felt that their other cries for help were not heard they would turn to actual suicidal attempts. The cases noted in this study were with over-dosing with pills and slashing of wrists as a means to get out of the pressured hell that they felt school had become. The pressures to perform to other people's ideals and without others understanding their problems lead them to want out and taking an over-dose or cutting were their only way out. They felt isolated at home, in class and in the playground.

Post Traumatic Stress Disorder

According to According to The National Institute of Mental Health (2008b) Post-traumatic stress disorder (PTSD) is an anxiety disorder that some people develop after seeing or living through an event that caused or threatened serious harm or death. Symptoms include flashbacks or bad dreams, emotional numbness, intense guilt or worry, angry outbursts, feeling "on edge," or avoiding thoughts and situations that remind them of the trauma. In PTSD, these symptoms last at least one month and can be a contributing condition to depression.

The advantages of using a biographical study such as this, is the ability to ask adults about their childhood and their long-term reactions. PTSD is one such expression of reactions and denotes emotional responses to feared stimulus. In this study, school and returning to school for ones child's education is the feared stimulus and suggests that such a fear is a long-term effect of trauma from their own time at school, from trauma delivered by teachers, especially in mainstream education.

The evidence presented suggests that the smell, small chairs and layout of schools trigger emotional responses which cause panic, sweating and anger. Also in one subject it causes crying. It was felt that they experienced belittlement feelings from teachers and that even

though they were fully grown adults with degrees and careers, as soon as they entered into school they felt like a small helpless child again. Feelings of inferiority were also encountered as they dealt with multi-sensory triggers of anxiety.

Reactions were also caused by dealing with teachers for their own children's education; this causes them to get flashbacks to their own childhood and feelings of anger towards the educational system for their own children now facing similar difficulties. It was felt that educationally the treatment of dyslexics had changed very little over the last thirty to forty years, causing them to get emotional about their child reliving their own frustrations. These feelings can get in the way of the normal parent-teacher relationship and can even cause tension between parents where one is dyslexic and another is not. Several of the participants in this study noted that they or their parents were diagnosed as a by product of having their child diagnosed.

Such PTSD feelings are noted in this study to cause the participants to avoid going into school, with one noting that he had only been into school two or three times in his children's school careers. Such avoidance should be noted.

CONCLUSIONS

This reflective biographical study of N=29 dyslexic adults between the ages of 23 to 65 year olds, used a semi-structured investigative interview methodology to probe adult dyslexics for their childhood experiences. Whilst the study searched for several types of adult dyslexics, it was found that dyslexic adults with depression both with and without degrees were the largest group (N=21). Female dyslexics with depression were the largest subgroup (N=16) and this may reflect higher frequency of females developing depression than males in the general population (Cyranowski, Frank, Young and Shear, 2000), suggesting that females tend to cope more emotionally than males.

Thirteen child related themes were developed from transcripts and it should be noted that the whole transcripts are the basis for a book project by the author planned for 2009 (Alexander-Passe, in press-4). The themes were: What is dyslexia?; Difference; Humiliation, Schools and Teachers; Frustration and Anger; Avoiding and being the Class Clown; Rebelling in the Classroom; Truanting and running away from home; Perfection; Bullying; Going into a Bubble and Regression; Why some Dyslexics get Depressed; Self-harm and Suicide; and Post Traumatic Stress Disorder.

These themes were developed and sample quotes were presented for the results section of this chapter, along with a number of sub-theme quotes. The body of evidence included in this chapter gives the reader a flavour of the emotions a dyslexic (child) goes through. There seemed little difference between those who have recently left education, to those who left over forty years ago, suggesting that mainstream education has been slow in developing. It is still limiting children with disabilities to gain full access to the curriculum, although educators would argue that those with severe learning disabilities have gained (e.g. those with physical disabilities) from wheelchair access to classroom buildings. However the author questions how well mixed-ability classrooms have helped dyslexic or other non-average learners.

The evidence presented in this chapter suggests that dyslexics are commonly humiliated in class, with their difficulties making them frustrated, as it highlights their inability to learn as per their peers. They perceive the classroom as a threatening environment rather than a place to gain new knowledge.

They look for opportunities to reuse old knowledge rather than experimenting to learn new ones, illustrated in their use of smaller words in writing tasks and avoiding reading tasks. Thus they are using their intelligence to avoid rather than using it to learn. However they are unable to avoid all feared activities as reading and writing are core skills used in mainstream education. Their avoidance camouflages their inabilities and gets in the way of their teachers recognising that they are struggling in class. Parents are also to blame for covering up their child's difficulties, with some parents even doing their child's homework to lessen their own pride of having what they and their child's teachers perceive their child as 'lazy or slow'. There is a perceived stigma concerning having a child who learns abnormally or is unable to keep with their peers.

The evidence in this study suggests that avoidance is the key strategy for dyslexics to survive their years at school, starting with avoiding putting up their hand to join the academic discourse in the classroom, to continually sharpening pencils to avoid time on tasks in the class. Their teachers do not recognise this reluctance, so classroom humiliation and avoidance continues. At some point the child recognises that the long hard effort they put into their assignments is not reflected in the marks they receive and this confirms to them that they are abnormal learners or just plain 'stupid'. They then look to either rebel against their classroom environment or aim to be invisible to reduce further their interaction with these feared stimuli. Avoidance turns to truancy, faking sickness to avoid school or running away from home.

Depression also begins to set in, as they try and deal with the frustrations, anger and anxieties from their school day and a home life that commonly is not understanding of their unique set of difficulties. This is made worse by their being non-other family members or friends with similar difficulties, as these again confirm to them that their bodies are faulty or that they are adopted or in fact aliens from outer space. Alternatively their self-blame could turn into self-harm to either control their bodies, as they have little control over the rest of their world, as found in the case of cutting their bodies, anorexia, drug/alcohol abuse and binge-eating. These are cries for help when their other cries have not been heard. If they are still ignored then suicide, as seen in this study is an option to put an end to the long-term pain they are experiencing.

What is needed?

From the evidence presented in this study, it seems that to avoid dyslexic children developing depression, certain aspects of their school and home life needs changing: (1) Parents and teachers need to be made aware that reluctant learners might have problems and that they should refer to symptom checklists of dyslexia (see Appendix 1); (2) Calling a child lazy or stupid doesn't help and just undermines the child's self-esteem and gets in the way of learning; (3) Teachers need to look out for reluctant learners in the classroom and ask themselves why they are being reluctant; (4) If a child is bright orally but not on paper, ask yourself why? Investigate possible reasons; and (5) Teachers need to be less defensive about children in their care. Some children just may not be suited to particular teaching

methodologies. Try alternative ones and see if it makes a difference. It should not be seen as a threat to a teachers professionalism to ask for help with certain children who are reluctant to learn.

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APPENDIX 1. IDENTIFYING DYSLEXIA IN CHILDREN (BDA, 2008)

Persisting Factors

There are many persisting factors in dyslexia, which can appear from an early age. They will still be noticeable when the dyslexic child leaves school. These include:

- Obvious 'good' and 'bad' days, for no apparent reason,
- Confusion between directional words, e.g. up/down, in/out,
- Difficulty with sequence, e.g. coloured bead sequence, later with days of the week or numbers,
- A family history of dyslexia/reading difficulties.

Pre-School

- Has persistent jumbled phrases, e.g. 'cobbler's club' for 'toddler's club'
- Use of substitute words e.g. 'lampshade' for 'lamppost'.
- Inability to remember the label for known objects, e.g. 'table, chair'.
- Difficulty learning nursery rhymes and rhyming words, e.g. 'cat, mat, sat'.
- Later than expected speech development.

Pre-School Non-Language Indicators

- May have walked early but did not crawl - was a 'bottom shuffler' or 'tummy wriggler'.
- Persistent difficulties in getting dressed efficiently and putting shoes on the correct feet.
- Enjoys being read to but shows no interest in letters or words.
- Is often accused of not listening or paying attention.

- Excessive tripping, bumping into things and falling over.
- Difficulty with catching, kicking or throwing a ball; with hopping and/or skipping.
- Difficulty with clapping a simple rhythm.

Primary School Age

- Has particular difficulty with reading and spelling.
- Puts letters and figures the wrong way round.
- Has difficulty remembering tables, alphabet, formulae etc.
- Leaves letters out of words or puts them in the wrong order.
- Still occasionally confuses 'b' and 'd' and words such as 'no/on'.
- Still needs to use fingers or marks on paper to make simple calculations.
- Poor concentration.
- Has problems understanding what he/she has read.
- Takes longer than average to do written work.
- Problems processing language at speed.

Primary School Age Non-Language Indicators:

- Has difficulty with tying shoe laces, tie, dressing.
- Has difficulty telling left from right, order of days of the week, months of the year etc?
- Surprises you because in other ways he/she is bright and alert.
- Has a poor sense of direction and still confuses left and right.
- Lacks confidence and has a poor self image.

Aged 12 or Over

As for primary schools, plus:

- Still reads inaccurately.
- Still have difficulties in spelling.
- Needs to have instructions and telephone numbers repeated.
- Gets 'tied up' using long words, e.g. 'preliminary', 'philosophical'.
- Confuses places, times, dates.
- Has difficulty with planning and writing essays.
- Has difficulty processing complex language or long series of instructions at speed.

Aged 12 or Over Non-Language Indicators

- Has poor confidence and self-esteem.
- Have areas of strength as well as weakness.

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Chapter 2

DYSLEXIC CHILDREN AND DEPRESSION: EMPIRICAL EVIDENCE

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ABSTRACT

This is the first of two chapters to investigate dyslexia, children and depression. This chapter begins by defining what a child is, what depression is and lastly what dyslexia is. It is felt by the author that such a strategy is needed to clearly define the direction of the empirical investigation. Inclusion of the indicators of dyslexia, not only in pre-school but in primary and secondary school ages are necessary, so that a reader can assist in recognising unidentified dyslexia amongst those they meet, if necessary. This is necessary as it would seem from the evidence presented that either teachers are unable or unwilling to identify dyslexia in their pupils. Such a situation causes frustration and stress, with children therefore seeing the classroom as an environment that must be controlled and avoided. That learning is not the investigative process that it should be, but an opportunity where embarrassment and failure commonly takes place in front of their peers.

Dyslexics with high intelligence will use their intellect to carefully choose subject options that will limit their time doing feared tasks (e.g. reading out loud in class and essay writing) and choose science or design subjects. They will use their energies to avoid and choose easier to spell words. Dyslexics with normal or lower intelligence will have a harder time. They will have fewer strategies to avoid feared tasks and will begin to self-doubt and may either lash out or withdraw.

A large number of empirical studies in this area suggest that many dyslexics develop negative learning strategies, as a reaction to the frustrations and stresses they encounter, whilst this is an unhealthy attitude to learning, it is one option which will protect their self-esteem. Unfortunately evidence points to secondary manifestations developing which are both emotionally and psychological damaging. In some cases these can turn into physical manifestations with self-harm and suicide as the ultimate conclusion. All children hate to fail, hate to be different and hate to be singled out as having special needs. Their segregation to the remedial or 'slow' table or taken out of the classroom for

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extra help may be a negative strategy as they are marked out as being different in the eyes of their class peers.

What is clear is that dyslexics do not feel integral to their family and society as a whole, and they look for ways to protect themselves from the harm that such a conclusion poses. Such a conclusion can, if taken to extremes, mean that young dyslexics can withdraw from society, self-harm to punish their perceived faulty bodies or limiting their burden on their parents and society by attempting suicide in extreme cases. Such a conclusion, especially in childhood suggests the support framework which children need to rely on from parents, peers, teachers, school and the medical profession is not working effectively. The Dyslexic Defence Mechanisms DDMs (Alexander-Passe, in press-2) suggest a means to understanding the processes involved in coping with an unidentified learning difficulty such as dyslexia.

Key Words: Dyslexia, Children, Depression, Avoidance, Coping, Self-harm

DEFINITIONS

Defining a Child

A child is a human being between birth and puberty; a boy or girl. The legal definition of 'child' generally refers to a minor, otherwise known as a person younger than the age of majority (18yrs). 'Child' may also describe a relationship with a parent or authority figure, or signify group membership in a clan, tribe, or religion; it can also signify being strongly affected by a specific time, place, or circumstance, as in 'a child of nature' or 'a child of the Sixties'. (Bartleby, 2007). The United Nations Convention on the Rights of the Child defines a child as 'every human being below the age of 18 years unless under the law applicable to the child, majority is attained earlier' (United Nations, 1990).

Defining Depression

According to The National Institute of Mental Health (2008a) everyone occasionally feels blue or sad, but these feelings are usually fleeting and pass within a couple of days. When a person has a depressive disorder, it interferes with daily life, normal functioning, and causes pain for both the person with the disorder and those who care about him or her. Depression is a common but serious illness, and most who experience it need treatment (either medical, psychological or counselling) to get better, however many of those who suffer with a depressive illness never seek treatment. Whilst the vast majority, even those with the most severe depression, can improve with treatment, subsequent depressive episodes are common. Intensive research into the illness has resulted in the development of medications, psychotherapies, and other methods to treat people with this disabling disorder.

What are the Symptoms of Depression?

People with depressive illnesses do not all experience the same symptoms. The severity, frequency and duration of symptoms will vary depending on the individual and his or her

particular illness. According to The National Institute of Mental Health (2008a) the symptoms will include:

- Persistent sad, anxious or 'empty' feelings
- Feelings of hopelessness and/or pessimism
- Feelings of guilt, worthlessness and/or helplessness
- Irritability, restlessness
- Loss of interest in activities or hobbies once pleasurable, including sex
- Fatigue and decreased energy
- Difficulty concentrating, remembering details and making decisions
- Insomnia, early–morning wakefulness, or excessive sleeping
- Overeating, or appetite loss
- Thoughts of suicide, suicide attempts
- Persistent aches or pains, headaches, cramps or digestive problems that do not ease even with treatment

What Causes Depression?

There is no single known cause of depression. Rather, it likely results from a combination of genetic, biochemical, environmental, and psychological factors.

Research indicates that depressive illnesses are disorders of the brain. Brain-imaging technologies, such as magnetic resonance imaging (MRI), have shown that the brains of people who have depression act differently to those without depression. The parts of the brain responsible for regulating mood, thinking, sleep, appetite and behaviour appear to function abnormally. In addition, important neurotransmitters–chemicals that brain cells use to communicate–appear to be out of balance. But these do not explain why the depression has occurred in the first place.

Some types of depression tend to run in families, suggesting a genetic link. However, depression can occur in people without family histories of depression as well (Tsuang and Faraone, 1990). Genetics research indicates that risk for depression results from the influence of multiple genes acting together with environmental or other factors (Tsuang, Bar, Stone and Faraone, 2004). In addition, trauma, loss of a loved one, a difficult relationship, or any stressful situation may trigger a depressive episode. Subsequent depressive episodes may occur with or without an obvious trigger. This chapter is interested in depression triggered by stressful situations in childhood, such as school and perceptions of social difference brought about by dyslexia.

How do Children and Adolescents Experience Depression?

Until the 1970s it was believed that depressive disorders resembling adult depression were uncommon amongst the young. Pre-adolescent children were thought incapable of experiencing depression and were often seen as a normal feature of development, so called 'adolescent turmoil'.

In the 1970's and early 1980's researchers began to diagnose depression in children using adult criteria (Weinberg et al, 1973; Pearce, 1978; Puig-Antich, 1982). These studies showed

that even young children could experience depression resembling that of adults. In 1980 the American Psychiatric Association's criteria was changed to include both children and Adolescents with adult depressive disorder. The next 15yrs of research led to a reappraisal of the concept of childhood depression, and found that there seemed to be important difference between child and adolescent depression. In comparison with adolescent depression, pre-adolescent depression:

- Is less likely to lead to adult depression (Weissman et al, 1999a)
- Has more overlap with other disorders (Alpert et al, 1999)
- Is less prevalent (Angold et al 1998)
- Shows a male preponderance (Angold et al, 1998)
- Is more strongly associated with family dysfunction (Harrington et al, 1997)
- This pre-adolescent depression may therefore be a different diagnostic entity (Harrington, 2000).

Depression can be conceptualized both as a dimension and as a category. Epidemiological studies suggest that juvenile depression is a continuum that is associated with problems at most levels of severity (Pickles et al, 2001). Indeed it seems that there is no 'good' level of depression, it is better to have no symptoms of depression at all than to be averagely depressed (Harrington & Clark, 1998). The Oregon Adolescent Depression Project found that the level of psychosocial impairment increased as a direct function of the number of depressive symptoms (Lewinsohn et al, 1998). Moreover, in line with studies of adults (Angst et al, 1997), much of the morbidity associated with depression occurred in the 'milder' but more numerous cases with minor depression. Even mild forms of adolescent depression are a risk factor for depression in early adulthood (Pine et al, 1999). Thus depression should be regarded as a continuum. Clinicians do not generally prescribe a little bit of anti-depressants for a little bit of depression, depression usually means a diagnosis, something a patient has or has not.

The presence of childhood depression also tends to be a predictor of more severe illnesses in adulthood (Weissman, Wolk, Goldstein, Moreau, Adams, Greenwald, Klier, Ryan, Dahl and Wichramaratne, 1999). According to The National Institute of Mental Health (2008a) child with depression may pretend to be sick, refuse to go to school, cling to a parent, or worry that a parent may die. Older children may sulk, get into trouble at school, be negative and irritable, and feel misunderstood. Because these signs may be viewed as normal mood swings typical of children as they move through developmental stages, it may be difficult to accurately diagnose a young person with depression. Research suggests that before puberty, boys and girls are equally likely to develop depressive disorders. By age 15, however, girls are twice as likely as boys to have experienced a major depressive episode (Cyranowski, Frank, Young and Shear, 2000). Depression in adolescence comes at a time of great personal change–when boys and girls are forming an identity distinct from their parents, grappling with gender issues and emerging sexuality, and making decisions for the first time in their lives. Depression in adolescence frequently co–occurs with other disorders such as anxiety, disruptive behaviour, eating disorders or substance abuse. It can also lead to increased risk of suicide. (Shaffer, Gould, Fisher, Trautman, Moreau, Kleinman and Flory, 1996). Lastly, a recent clinical trial of N=439 adolescents with major depression found that a combination of

medication and psychotherapy was the most effective treatment option (March, Silva, Petrycki, Curry, Wells, Fairbank, Burns, Domino, McNulty, Vitiello and Severe, 2004).

For more than a decade clinicians use either one of the major schemes for diagnosis: ICD-10 (World Health Organisation, 1992) or DSM-IV (American Psychiatric Association, 1994). Each differ in many ways but the core concept 'an episodic disorder of varying degrees of severity that is characterized by depressed mood or loss of enjoyment that persists for several weeks'. The individual must also experience other symptoms during the episode, these include depressive thinking - such as pessimism about the future or suicidal ideas, and

biological symptoms - such as early waking, reduced appetite and weight loss.

DSM-IV states that in children irritable mood may substitute for depressed mood and that 'dysthymia' need only last for 1yr (2yrs in adults). Also that somatic complaints and social withdrawal are particularly common in children, whilst delusions are rare. ICD-10 does not mention age differences.

Both the DSM-IV & ICD-10 distinguish between mild, moderate and severe episodes of depression but differ in their definitions of severity. ICD-10 define severity by symptoms and DSM-IW define severity by symptoms and functional impairment

Differential Diagnosis

A problem with categorical approaches to diagnosis lies in differentiation from 'normality'. There is no obvious point of infection in the dimension of depressive symptoms, it is therefore not suprising that the DSM-IV & ICD-10 diagnoses are based on numbers of symptoms give implausibly high rates of so-called depressive disorder. Depressive disorder should therefore only be diagnosed when there is impairment of social role functioning, here symptoms of unequivocal psychopathological significance are present (such as severe suicidality) or when symptoms lead to significant suffering. A further problem arises from the fact that all epidemiological studies conducted to date found that depressive disorder very commonly occurs in conjunction with other psychiatric (physical) disorders and children with no psychiatric disorder: Restlessness is seen in agitated depression, hypomania and hyperkinetic syndrome, and Depression as the result of physical disorders (Leinsohn et al, 1988 found that adolescents who reported that they had a physical illness causing functional impairment (e.g. glandular fever) were at increased risk of subsequent depression.

Persistence

Studies of clinical samples have reported that young people with a depressive disorder have a high risk of re-occurrence or persistence (Emslie et al, 1997b; Goodyer et al, 1997a). Lewinsohn et al (1993) found that the 1yr relapse rate for unipolar depression (18.4%) was much higher than the relapse rate found in most other disorders.

Adolescent depressive disorder shows significant continuity into adult life. Harrington et al (1990) followed up 63 depressed children and adolescents on average 18yrs after their initial contact. The depressed group had a substantially greater risk of depression after the age of 17yrs than a control group, matched on a large number of variables. Other studies have found high rates of recurrence of major depression in clinical samples of depressed adolescents who have been followed through to adulthood (Rao et al, 1995).

Although follow-up studies suggest that depressive disorders tend to reoccur, it should be noted that the prospects of recovery from episodes are high. The majority of young people with major depression recover within two years, Kovacs et al (1984b) reported the cumulative

probability of recovery from major depression by one year after onset was 74% and by two years was 92%, and the median time to recovery was about 28 weeks. Similar results were reported by Keller et al (1988) in a retrospective study of recovery from first episode of major treatment and by Warner et al (1992) in a study of the children of depressed parents. Garrison et al (1997) found that only 20% of those with major depression at baseline continued to have it one year later. Harrington et al (1998b) found that most cases of major depression following deliberate self-poisoning lessen within 6 weeks. Recovery can be quite rapid.

Adolescent depression is a relapsing and emitting condition. Most adolescents will recover from major depression within a year, but a significant proportion will relapse. Persistence of depression is more likely if symptoms are severe (Pickles et al, 2001) or if the young person experiences persisting adversity e.g. family problems (Birmaher et al, 2000). Depressed adolescents are at risk of a variety of other problems later in life, including poor social functioning and suicide. However, the risks stem in part by other conditions associated with depression e.g. behavioural problems and drug abuse.

Defining Dyslexia

According to the National Institute of Child Health and Human Development, using IDA (2002), Dyslexia is characterized by difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities. These difficulties typically result from a deficit in the phonological component of language that is often unexpected in relation to other cognitive abilities and the provision of effective classroom instruction. Secondary consequences may include problems in reading comprehension and reduced reading experience that can impede growth of vocabulary and background knowledge.

Dyslexia is a widespread condition, with an estimated frequency of one dyslexic child in each mainstream school classroom (Miles, 1994). Professor Berlin of Stuttgart (1872) coined the term ‘dyslexia’, based on the case histories of adults who could read only three to five words, but were of high intelligence. Use of the term has continued to this day, with dyslexia likened to conditions with neurological abnormalities. Initially, it was thought to be purely an acquired condition from accidental brain damage, until Kussmaul (1878) found developmental cases of word blindness. Orton (1937) first noted the main aspects of developmental dyslexia as pronounced reversals (b/d, p/q, on/no and was/saw), orientation difficulties/strong left-handedness and conflicting lateral preference, which forms the basis for many definitions (see Thomson, 1996; Miles, 1994). This study uses the definition from the World Federation of Neurology (1968), which defines dyslexia as ‘a disorder manifested by difficulty in learning to read despite conventional instruction, adequate intelligence and socio-cultural opportunity’.

In the UK and many countries in Europe, terms such as ‘dyslexia’ or ‘Specific Learning Disabilities (SpLD)’ are used, whilst worldwide and especially in the US, ‘Learning Disabled (LD)’ and ‘Reading Difficulties (RD)’ are commonly used terms. ‘Dyslexia’ and ‘SpLD’ are more specifically concerned with difficulties that affect most situations (not just reading, e.g., co-ordination and balance) with neurological and phonological epidemiology. Reviews on dyslexia can be found in Thomson (1995) and Miles (1994).

Dyslexia is a negative disorder that affects many life skills (reading, writing, arithmetic) as well as balance and co-ordination, with Miles (1994, p. 189) suggesting that dyslexics

show ‘an unusual balance of skills’. Individuals with dyslexia can be affected emotionally by being unable to learn as well as their peers, commonly resulting in low self-image, low self-concept and even depression (Alexander-Passe 2004a, b, 2008a, b, in press-1, in press-2; Riddick, 1996; Edwards, 1994; Ryan, 1994). For more than a decade, research has been carried out to identify positive attributes of this disorder; these investigations began with biographical and neurological studies. West (1991) located famous and influential individuals who had school learning difficulties, yet had found alternative ways of learning and succeeding in life (e.g., Albert Einstein, Leonardo da Vinci), making correlations between these factors and dyslexia, and creativity. Thus, public perception of creativity amongst dyslexics has grown (e.g., 712,000 records on Google). Since West, the use of famous names with dyslexia (e.g., Charles Schwab, Richard Branson, Tom Cruise, Richard Rogers and so on.) has become widespread (Being Dyslexic 2006; Roehampton University, 2006; British Dyslexia Association, 2006; British Broadcasting Corporation, 2004; International Dyslexia Association, 2006; General Communication Headquarters, 2006, McLoughlin, Fitzgibbon and Young, 1994) to illustrate the career heights that dyslexics can reach. However, this could be misleading and could give false hopes to parents, as a high proportion of dyslexics leave full-time education with few or no qualifications (Grant, 2001)

Neurological investigations started by Geschwind and Galaburda (1985) noting clinical and post-mortem studies indicated atypical symmetrical brains, suggesting dyslexics have larger right (visual processing) brains; however, this conclusion was based on a very small sample of brains. Galaburda (1989) also suggests an ‘alternative wiring hypothesis’ that some abilities may be strengthened at the expense of others in the re-organisation in the brains of dyslexics. The classic measurement of dyslexia uses the WISC-R (Wechsler, 1974) measures, and the ACID profile (Thomson, 1996) indicates dyslexics show superior performance, rather than verbal skills, in WISC-R subscales. Thus, investigations have since been made into the possibility of superior visual-spatial abilities amongst dyslexics, with many correlating such skills to creativity (Padgett and Steffert, 1999; Wolff and Lundberg, 2002).

Nicolson and Fawcett (1993) note that a skill that would take a normal child 400hrs to develop and learn would take a dyslexic child 20 times longer to achieve. It is interesting that Fawcett and Nicolson (1994, 1996) found in their studies that 17yr old dyslexics achieved no better performance in a range of literacy skills than their 8yr old controls (in tests of psychometric, phonological and working memory, balance, motor skills and speed of processing). Fawcett (1995, p. 27) gave an analogy of driving through a foreign country, one can do it but at the expense of constant vigilance and an unacceptable cost in resources. Thus dyslexics can go through mainstream schools without high literacy skills but the emotional cost can be unacceptably high.

Identifying Dyslexia in Children (British Dyslexia Association, 2008)

Persisting Factors

There are many persisting factors in dyslexia, which can appear from an early age. They will still be noticeable when the dyslexic child leaves school. These include:

- Obvious 'good' and 'bad' days, for no apparent reason,

- Confusion between directional words, e.g. up/down, in/out,
- Difficulty with sequence, e.g. coloured bead sequence, later with days of the week or numbers,
- A family history of dyslexia/reading difficulties.

Pre-School

- Has persistent jumbled phrases, e.g. 'cobbler's club' for 'toddler's club'
- Use of substitute words e.g. 'lampshade' for 'lamppost'.
- Inability to remember the label for known objects, e.g. 'table, chair'.
- Difficulty learning nursery rhymes and rhyming words, e.g. 'cat, mat, sat'.
- Later than expected speech development.

Pre-School Non-Language Indicator

- May have walked early but did not crawl - was a 'bottom shuffler' or 'tummy wriggler'.
- Persistent difficulties in getting dressed efficiently and putting shoes on the correct feet.
- Enjoys being read to but shows no interest in letters or words.
- Is often accused of not listening or paying attention.
- Excessive tripping, bumping into things and falling over.
- Difficulty with catching, kicking or throwing a ball; with hopping and/or skipping.
- Difficulty with clapping a simple rhythm.

Primary School Age

- Has particular difficulty with reading and spelling.
- Puts letters and figures the wrong way round.
- Has difficulty remembering tables, alphabet, formulae etc.
- Leaves letters out of words or puts them in the wrong order.
- Still occasionally confuses 'b' and 'd' and words such as 'no/on'.
- Still needs to use fingers or marks on paper to make simple calculations.
- Poor concentration.
- Has problems understanding what he/she has read.
- Takes longer than average to do written work.
- Problems processing language at speed.

Primary School Age Non-Language Indicators

- Has difficulty with tying shoe laces, tie, dressing.
- Has difficulty telling left from right, order of days of the week, months of the year etc.
- Surprises you because in other ways he/she is bright and alert.
- Has a poor sense of direction and still confuses left and right.
- Lacks confidence and has a poor self image.

Aged 12 or Over

As for primary schools, plus:

- Still reads inaccurately.
- Still has difficulties in spelling.
- Needs to have instructions and telephone numbers repeated.
- Gets 'tied up' using long words, e.g. 'preliminary', 'philosophical'.
- Confuses places, times, dates.
- Has difficulty with planning and writing essays.
- Has difficulty processing complex language or long series of instructions at speed.

Aged 12 or Over Non-Language Indicators

- Has poor confidence and self-esteem.
- Has areas of strength as well as weakness.

THE EFFECTS OF SCHOOL

Normality and School

All children when entering school or nursery perceive themselves as normal. They have had many years of encouragement by parents from activities on a one to one basis, on entering school they begin to compare themselves to others as others compare themselves to them. They enter school thinking the world is a happy place and very quickly learn that there are winners and losers in life, children who can do things and children who can't. Life changes from a safe and friendly environment to one that they must be on edge to defend themselves not only in the classroom, but in the playground as well. In primary and early secondary school, all children wish to be the same and not stick out, as experienced dyslexia teachers noted 'dyslexic children – all children – hate to fail, hate to be different, hate to be singled out as having 'special needs' (Burden, 2005).

The undiagnosed dyslexic child sticks out easily as different and their segregation to the slow table or the remedial class does not help the situation. As the world of comparison has put them on edge, due to failure they become hypersensitive to criticism which is perceived to be a logical reaction to frequent failure, more frequent than their peers (as found by Edwards, 1994 and Riddick, 1996). Cutting and Dunn (2002, p. 856) found that generally 'five year olds…were sensitive to criticism; they reacted significantly more negatively in response to criticised failure than in response to non-criticised failure'. Such a reaction causes the production of anxiety and lower self-esteem, indicating how fast school failure affects children.

Erikson (1959) notes that our early childhood experiences play a significant part in our attitudes towards ourselves and our place in the world. This shaping is called self-concept and is easily damaged. Rogers (1959, p. 138) discusses self-concept to be 'composed of such elements of the perceptions of one's characteristics and abilities: the perceptions and concepts of the self in relation to others and to the environments; the value qualities which are perceived as associated with experiences and objects; and the goals and ideas which are

perceived as having positive or negative valence'. Or as Burns suggests (1982, p. 7) 'self-concept is best regarded as a dynamic complex of attitudes held towards themselves by each person' and has two parts: self-image and self-evaluation. However, Hansford and Hattie (1982) point out that self-concept, self-image and self-esteem are often used interchangeably without adequate definitions and applied without valid and reliable measurement techniques. This comes in part from the hypothesis that self-concept is based on a highly subjective set of constructed attributes and feelings which take on meaning for the individual through evaluation compared to their particular society (Burns, 1982), as also found with self-efficacy.

Morgan and Klein (2001, p. 53) suggests that dyslexics have strong awareness in comparing themselves with their peers and recognise intuitively their undefined and unacknowledged learning difficulties, with many adults noting that they felt this way from an early age. Gilroy (1995, p. 66) notes in her experience that dyslexic students tend to compare themselves unfavourably with their peer group and that Griffiths (1975) found that dyslexic students saw themselves as less intelligent than their peers. Thus they recognise their abnormality, as also seen by Alexander-Passe (in press-2) with dyslexic adults.

The Dyslexic Child at School

When life goes wrong for a dyslexic at school, it has a knock-on affect to life after school, thus the influence of school is profound (Scott 2004, p. 53). At school there are two curriculums: academic and social, failure in either or both will have lifelong effect on the individual and their success, and can be powerful predictors of later outcomes as intelligence is commonly correlated to academic achievement (Sylva, 1994). Whether you choose to or not in the UK, children must legally attend school for at least ten years and parents are legally required to send them to a recognised institution, whilst home-study is an alternative, it is not encouraged and difficult to gain formal approval for such a choice. Each year at school can be unpredictable and is highly complex for both parents and children e.g. homework, class timetables etc. Integration with others (teachers and peers) can be fraught with danger with bad/good experiences and friendships having lasting impressions.

Several commentators (Fawcett, 1995; Edwards, 1994; Riddick, 1996, Saunders, 1995) view schools as legalised abusive institutions, where children are kept against their will and subjected to the will of teachers. Whilst in the majority of cases children and parents are accepting of such a regime, it does not suit all children and parents, so many choose to transfer out their child's education to the private sector. However, in the private sector more abuse may exist to motivate pupils, with some private schools requiring parents to sign forms to permit caning and other forms of corporal punishment, in the name of school ethos and learning.

Scott (2004, p. 54) notes 'all dyslexic children – and this is not a loose phrase – experience some form of damage from school'. Like all children, some excel in the school environment and others do not. In the case of dyslexics, larger proportions do not excel, resulting in high levels of stress and anxiety. Dyslexics find school an unpredictable battleground in which they are unskilled to exist. Scott (2004, p. 55) notes that 'for the vast majority of dyslexic children and adults, school has been a place of psychological and often physical torture…School for them was destructive and humiliating, nasty, degrading

experience, sometimes of raw brutality, of which modern society should be deeply ashamed [of]', comments that others echo (Fawcett, 1995; Edwards, 1994, Riddick, 1996).

Dyslexics fail at school in numerous ways: failure to make friends, failure in literacy, failure to be attractive (to their peers as friends and teachers as receptive to help) and failure to be normal.

As school promotes both a social and academic curriculum, failing to make friends and failure to gain literacy are two basic stepping stones needed for development. Due to their learning difficulties they do not come across to others as normal e.g. clumsy and not knowing their left and right which affects play with others.

At school, failure in literacy has huge knock-on effects for the whole academic curriculum and with all subjects. Failing to read and write is a very public failure (Scott, 2004) and begins to propagate the idea that dyslexics are abnormal. This sets off a chain reaction that puts the child in a defensive state of mind and makes them fearful of all learning situations. School becomes a dangerous place and sticking out in class continues to the playground where teasing begins and can lead to unhealthy bullying. Children are very quick to pick-up who is the brightest and dumbest in the class and until a dyslexia diagnosis is made, the child begins to self-doubt and believe they are stupid, thinking 'if everyone says I'm slow/stupid I must be'. Lastly, teachers prefer easy children, children who do not create problems for them, as this confirms their effectiveness as teachers

Congdon (1995, p. 91) notes the reactions to failure at school 'can vary. Some children lose interest and adopt negative and avoidance attitudes. Others may try harder, spurred on by their teacher and parents, only to discover that the greater effort does not produce the longed for results'. Feelings of disillusionment and mystification sets in, as up to this point the child may have been told he was clever by his parents which he believed until now, thus he is now confused and develops self-doubts.

Stress

Stress is the personal perception of how one communicates with the environment around us and is highly subjective. It is normal for pupils to be anxious when taking tests as people commonly wish to do their best. According to Thomson (1995, p. 33), 'stress appears threatening only when it becomes pervasive and invasive, when it affects too many areas of our lives and when we have neither the strategies nor the energy to cope with it'.

A combination of factors (e.g. lack of strategies or lack of skills) can result in: 'an overwhelming sense of apprehension, incompetence and confusion, sleeplessness and fatigue. It also invites the victim to resort to escape mechanisms which indirectly relieve pressure, to fantasise, obsess, rebel or withdraw' in dyslexics at school (Thomson, 1995, p. 33).

Whilst one can't reasonably protect children from all stress, as some measure of it is useful, parents and teachers should monitor stress levels carefully. Thomson (1995, p. 34) notes that 'if stress levels become intolerably high, many dyslexic children develop their own inappropriate strategies, becoming disruptive, aggressive, withdrawn or school phobic'. Some even also enter primary school vulnerable, as they have already learnt in pre-school that are unable to learn as easily as their peers.

As Fawcett (1995, p. 12) notes 'it is hardly surprising that life is stressful for a dyslexic child who is failing within the school system….a useful analogy here might be that of the dyslexic constantly running on a treadmill, just to stay in one place in a range of skills that others acquire with ease'.

Hales (1994) notes using the 16PF (Cattell, Eber and Tatsuoka, 1970) with N=300 dyslexics of mixed age, found that infant aged dyslexic children had scores which indicated that they were tense and frustrated, the primary school, low motivation and high anxiety and at secondary school a desire to keep in the background. Also in the middle school he found there was a noticeable drop in confidence and optimism, especially among girls. He also found an overall inverse relationship between anxiety and IQ, with middle school children with low IQ have the higher levels of anxiety.

Alexander-Passe (2008a, b) investigated the sources and manifestations of stress in N=78 school-aged dyslexics compared to N=77 sibling controls. Results suggest significant differences between the groups, with dyslexics in primary school experiencing the highest stress levels, specifically in interactions with teachers, worries over academic examinations (SATs) and performance testing, causing emotional (fear, shyness and loneliness) and physiological (nausea, tremors, or rapid heart beat) manifestations. Results also suggest that dyslexics in larger families (3 to 4 sibling families) experience greater stress in interactions with their peers, than those in smaller families (2 sibling families) – possibly from greater unfair sibling comparison.

Anxiety

Grigorenko (2001), Hales (1994) and Scott (2004) suggest there is clear evidence that dyslexics suffer from anxiety disorders. In addition, Legrand, McGue and Iacono (1996) suggest that approximately 20% of all adolescents suffer from anxiety disorders which can exhibit itself in numerous ways: panic attacks; irritability; restlessness; poor concentration; incoherence in speaking; fear; and the inability to move. Physical features include: dizziness; faintness; sweating; tremor; tension in neck; chest or stomach pain; nausea; shortness of breath; diarrhoea; increased urination; palpitations; hyperventilation; and insomnia (Gomez, 1991). It has been linked with alcoholism and drug taking, with Scott (2004) suggesting that such abuse is higher amongst dyslexics than non-dyslexics.

As Gomez (1991, p. 68) states ‘everyone is anxious some of the time - survival depends upon alertness to danger’ and one should distinguish between rational human response to real threat, as opposed to the irrational, neurotic and often learned response of excessive anxiety that have become an end in itself. This is particularly relevant for dyslexics who may be experiencing a very real threat to them[selves]’ (Scott, 2004, p. 169). In studies of children’s anxieties (Winkley, 1996; Jacobs, 1986) the following basic anxieties were found: fear of isolation; abandonment; not understanding; not knowing; being disoriented in new circumstances; and fear of the notion of emptiness. Scott (2004, p. 170) says ‘I am struck by how almost every one of these childhood anxieties are particularized in the dyslexic condition’.

The DSM-IV (APA, 1994) defines excessive anxiety or phobia as when an individual’s reactions are out of proportion to the demands of the situation, cannot be explained or reasoned away, is beyond voluntary control or lead to an avoidance of the feared situation. Other important signs are that such fears persist over an extended period of time and are unadaptive or are not age or stage-specific. Scott (2004, p. 170) a counsellor to dyslexics notes ‘excessive anxiety occurs a lot in dyslexics, and it is a serious and disabling condition…anxiety often leads to panic attacks in both dyslexic children and adults’. Thus it could be suggested that dyslexia as a condition is disabling and the excessive anxiety resulting from the condition is also disabling, thus a double whammy!

Self-Esteem

Morgan's (1997) study of delinquent/criminal dyslexics found that, when dyslexic children fail to keep up at school, their self-esteem drops as they begin to question their academic abilities (developing inferiority complexes). There are suggestions that both unrecognised and recognised dyslexics receiving insufficient or inappropriate support can feel devalued at school and turn to deviant behaviour. This is a response to their sense of low self-esteem induced by school and as a way of gaining recognition from their peers (Kirk & Reid, 2001 & Scott, 2004). Riddick, Sterling, Farmer and Morgan (1999) and Peer & Reid (2001, p. 5) suggests 'frustration leads very often to antisocial or deviant behaviour' amongst dyslexics, especially those with low self-esteem.

Some pupils might disrupt a class because they interpret the class work as threatening, and use attention seeking to protect self-esteem, according to Molnar and Lindquist (1989). They suggest that if the teacher, in class with pupils, can help re-interpret the nature and purpose of class work (keeping the child's self-esteem), the child's long-term behaviour will change. But most teachers, as Molnar and Lindquist found, hand out reprimands, as this is the only skill teachers know to quickly influence a child's present behaviour – a fire-fighting technique. Low self-esteem will also mean the development of a poor or negative self-image. Such beliefs can become self-fulfilling prophecy of expecting to fail (Riddick, 1996). Morgan and Klein (2001) note that childhood experiences of being labelled 'thick' and public humiliation caused by failing often results in choices which reinforce low self-esteem.

Studies of dyslexics suggest that low or poor self-esteem is commonly encountered (Hales, 1994, Riddick, 1996; Humphrey, 2002; Alexander-Passe, 2004a, b, 2008a, b, inpress1, 2). As Barret and Jones (1996) note 'it would be naive to assume that dyslexics would have good self-esteem given their learning difficulties' As dyslexics are often bullied, there are strong correlations between bullying and poor self-esteem, with particular strong relationships in children with special educational needs (O'Moore and Hillery, 1992, p. 64). Specialist schools for dyslexics have been found to improve self-esteem, especially social and academic self-esteem (Thomson and Hartley, 1980), and Scott (2004) suggests the best improvements in self-esteem comes from literacy and the improvement of literacy breaks the difference between them and their peers, as 'difference' is the core of the problem.

Empirical studies note correlations between low self-esteem/anxiety and academic failure (Burns, 1982), more so with dyslexics, as Humphrey and Mullins (2002, p. 199) notes 'the experience of dyslexics at school has clear and demonstrable negative effects on the self-concept and self-esteem of children'. Riddick et al. (1999, p. 241) indicated 'the powerful mediating effect of literacy performance on how individuals perceive themselves and are perceived by others', suggesting literacy failure can distort the dyslexic's self-perception. As Thomson and Hartley (1980) and Humphrey and Mullins (2002) note, dyslexics acquire a belief that being a good reader is significantly correlated with both happiness and intelligence, with the implication that as they are not good readers they are unhappy and unintelligent. Which leads dyslexics to believe they are out of control in their own learning and destiny, which Burns (1982) found to be the single biggest factor a child needs to progress at school (above that of good teachers, facilities and curriculum). The lack of control can be seen as a predictor of academic success, with striking parallels found between learned helplessness and children with reading difficulties (Butkowsky and Willows, 1980, p. 410) with Hiebert, Winograd and Danner (1984, p. 1139) finding that dyslexic children 'attribute[d] their success

to factors beyond their control'. Pumfrey and Reason (1991) also suggest that there are many correlations between learned helplessness and how dyslexics cope at school.

Gilroy (1995, p. 66) notes 'it is obvious that past experiences [of failure] leave a deep scar and that many [adult] dyslexic students have a poor self-concept and suffer from low self-esteem. Gilroy also details an interesting observation that in a spontaneous, undirected, general conversation lasting 20 minutes between five adult dyslexic students, the following words and phases were observed: hopeless at (seven times); useless at (five times); could never (three times); mess (twice); typical me (twice); never been any good at (twice). She points to 'typical' and 'never' suggesting deep-rooted poor self-image stretching back to childhood. Post-observation conversation noted four out of the five students 'often felt that they were thick'.

The failing reader must deal with self doubt which becomes far from being a secret shame, often becomes a public failure (Gaines, 1989). Osmond (1994, p. 31) found one boy saying 'I know inside I'm not stupid, but I look stupid to everyone else because all the things that I can't do are the things that you have to do at school'. Another young adult dyslexic noted 'the last person to be convinced I was dyslexic was me. I just thought I was thick at school and that it was my fault. I can remember the anger and frustration I felt, especially earlier on, and I still do I suppose, though not as much. I just felt uptight all the time'. This person had grown up thinking he was thick and stupid!

Riddick (1996, p. 32) notes one mother about her dyslexic son 'it was traumatic for him, incredibly traumatic, every morning I had to pull him up screaming 'I don't want to go to school' and then I had to pull him all the way down to school'. Riddick (1996) indicates there is general empirical consensus that children with reading difficulties are more likely to have behavioural and emotional difficulties (Tansley & Panckhurst, 1981; Gentile & Macmillan, 1987; Hinshaw, 1992).

Brinckerhoff, Shaw & McGuire (1993) identified the lack of positive self-concept as being the one consistent counselling issue that presents itself in people with learning difficulties, with Morgan & Klein (2001) suggesting this is the case amongst dyslexics. Battle (1992) claims that once an individual's level of self-esteem is well established, it becomes difficult to alter and remains relatively stable over time.

Dyslexic children with high self-esteem display more confidence and will volunteer answers or try out new subjects/tasks than lower self-esteem children. These high self-esteem children expect to succeed and attribute success to their skill/ability, according to Riddick et al. (1999) and Burden (2005). Coopersmith (1967) also found that dyslexic teenagers with high self-esteem were usually more successful in both academic and social environments compared to teenagers with low self-esteem. Wszeborowska-Lipinska (1997) investigated successful dyslexics who reached university education in Poland. To reach this level, she found that successful dyslexics had higher self-esteem than their peers.

Peer Group

According to Morgan and Klein (2001) responses from the peer group can be a powerful influence on the individual's perception of self. Dyslexics at special schools can overcome feelings of isolation and the sense of being different, those in mainstream school are never allowed to forget they are 'different' and 'abnormal'. Integration in mainstream schools is possible with the right level of support, but this is not commonly possible due to financial constraints (Audit Commission, 2002a, b, c). Morgan and Klein reported feelings of isolation

and loneliness, also they note it shows an awareness that reflects the ability of dyslexic people to make comparisons with peers and to recognize intuitively their undefined and unacknowledged learning differences (p. 53). Loneliness and isolation are typical of many dyslexic people, according to Tur-Kaspa, Weisel and Segev (1988).

Dyslexics Coping with School

In the study of coping, Endler & Parker (1999) suggests that three areas (Task, Emotion & Avoidance) should be investigated, as each play a part in coping methodology:

Task-Based Coping

Coopersmith (1967) found that successful dyslexic teenagers were active, expressive individuals. Wszeborowska-Lipinska (1997) investigated successful dyslexics and found that successful dyslexics were pro-active to overcoming hurdles, which required high levels of self-confidence. Scott et al.'s (1992) study found key factors to success amongst dyslexics to be: encouragement of talents; hobbies (from peers etc); and a search for self-worth. Reiff, Gerber and Ginsberg (1997) study of successful individuals with learning disability (an American term for dyslexia) also found that persistence and stubbornness were assets. McLoughlin et a. (2002) found hard work and determination to be underlying factors in success at school. All these traits: expressive; pro-active; search for self-worth; persistence; stubbornness; and determination are descriptions of task based coping strategies.

Emotional-Based Coping

Trying hard or asking for help and not receiving any, can cause children enormous frustration (Edwards, 1994). Parents and teachers see bright and enthusiastic children who are not successfully learning to read and write. Ryan (1994) comments that no one knows how hard the dyslexic is really trying, and each year that their peers surpass them in reading skills, their frustration increases.

It is important for teachers to recognise the frustration that dyslexics feel at school in the classroom: an inability to express their ideas in written form; an inability to read books of interest (rather than for their reading age) and having to work considerably harder than their peers to attain the same achievement level (Thomson, 1996). The negative experiences of school, as found by dyslexic teenagers in Edwards (1994) had associated reactions of lack of confidence, self-doubt/denigration, and sensitivity to criticism, behavioural problems, truancy/school refusal and competitiveness disorders.

In Butkowsky and Willows' study (1980), average to good readers attributed their success to their ability, whilst poor readers attributed their lack of success to luck. Poor readers however tended to blame themselves by attributing failure to their own incompetence, and success to environmental factors e.g. luck. Correlations to 'learnt helplessness' (Burden, 2005; Diener and Dweck, 1978; Miller and Norman, 1978) can also be made.

Dyslexics often react to their difficulties by withdrawing emotionally, or conversely becoming aggressive, compensating.... by obtaining negative attention from others (Thomson & Hartley, 1980, p. 19). Supporting Butkowsky and Willows, Hales (1995) suggest there is strong evidence to imply that dyslexics are more disturbed by criticism. Hales found dyslexics

experienced considerable amounts of criticism at school, especially before their condition was diagnosed.

All these traits: frustration; lack of confidence; self-doubt; sensitivity to criticism; behavioural problems; competitiveness disorders; self-blame; and aggressiveness are all descriptions of emotion based coping strategies.

Avoidance-Based Coping

In large schools, avoidance of competing or reaching potential goes unnoticed, compared to smaller schools. This extreme non-participation through lack of confidence is a recurring characteristic in dyslexics (Scott, 2004). Avoidance strategies deflect attention from low academic ability and under-performance and teachers see these avoidance strategies very differently, with perceptions such as laziness and lack of parental support.

Edwards (1994, p. 61) also noticed that some dyslexics suffer from competitiveness disorders, with many withdrawing both academically and socially 'Gareth only tries hard if he thinks he can win. If not he merely gives up…. Nevertheless, he had to be very sure of his good standard before making himself vulnerable again'.

Anxiety causes humans to avoid whatever frightens them, and dyslexia is no exception. However Ryan (1994) notes that teachers misinterpret this avoidance as laziness. In fact he notes that the avoidance is more related to anxiety and confusion than apathy. Reid (1988) found when pupils feel 'unwanted, rejected, uncared for and disillusioned … they start to manifest their disaffection by staying away, disrupting lessons, or underachieving'.

If academic success cannot give dyslexics self-worth, then they begin to withdraw from classroom activities (negative environments), according to Morgan (1997). There is a growing body of evidence to suggest that children with dyslexia avoid tasks which highlight their difficulties. Avoidance techniques can be as simple as constantly breaking the tips of pencils, so as to spend maximum time sharpening them and consequently less time at the desk doing work, although dyslexics (especially females) tend to prefer less obtrusive ways to avoid academic work, by rarely putting up their hands or sitting at the back of classes to be invisible (i.e. not picked by teachers to take part in the class).

Riddick (1996, p. 131) suggests 'by secondary age all dyslexic children claim that they avoid difficult to spell words and over half of them claim that they put off or avoid doing writing'. In a study of dyslexic school children (primary and secondary), Riddick (1996, p. 130) found pupils commenting that they 'daily avoided using difficult words to spell, wrote less (avoiding making mistakes) and put off starting work as coping strategies'.

In fact, out of 45 noted strategies found by Riddick, avoidance was featured in 35 of them. The other 10 were characterised by asking classmates to help. These findings were similar to Mosely's (1989) study concerning adults and children with general spelling difficulties. Pollock and Waller (1994) found that dyslexic children were perceived as immature (in their vocabulary choice and mode of expression) by school teachers and examination board markers, as they preferred using words they knew how to spell. But, if they did use words where the spelling is uncertain, they were accused of being careless and risking lower self-esteem. Thus word avoidance has attractive advantages to young dyslexics – they think it is better to be seen as immature than to risk embarrassment.

Another aspect of school refusal is shown by those individuals who develop psychosomatic disorders or other illnesses to avoid school: 'I used to pretend I was sick, make myself puke, and say I don't wanna go today', one dyslexic teenager commented (Edwards,

1994, p. 110). Edwards gives an powerful example of psychosomatic pain in the following story of a 12-year-old dyslexic, Trevor developed a pain in his right leg requiring crutches. To him it felt like a rare disease. The hospital doctor concluded that he was dyslexic but intelligent, was therefore frustrated, and that the frustration was expressed as pain in the right thigh, which occurred about once every six months and could last 10 days at a time. Strangely enough, she found this same teenager was reluctant to be truant, as he felt there would be 'repercussions and (that it) was pointless anyway' (p. 39).

This suggests a main difference between normal truants and dyslexics avoiding school (social conscience). Another 12-year-old used to get into fights with larger or other (dyslexic) kids to get off school. The injuries were for mutual avoidance reasons, not anger, and usually meant two to three days off school.

DEPRESSION AND THE DYSLEXIC CHILD

Scott (2004) suggests that whilst externalising (aggression) strategies are more common in dyslexic males, internalizing strategies such as depression and withdrawal are most commonly found in dyslexic females. Support comes from Alexander-Passe (2006, in press-1) and Wilcutt and Pennington (2000), with Hales (1994) finding that dyslexic females scored higher than controls on anxiety and depression, with Riddick (1996) speculating at the levels of self-blame, sensitivity to others and over perfections of others amongst dyslexic females and their particular vulnerability to adjustment problems.

Scott (2004) hypothesises that dyslexics either internalizing or externalizing the psychological effect of having dyslexia or suffering the effects of having a learning disability, with the former more likely. Support comes from Griorenko (2001, p. 112) suggesting 'internalizing, effects include stress, depression and anxiety and, on balance, are those most widely associated with learning disabilities'.

Humphrey and Mullins (2002, p. 197) conclude that the experience of dyslexia has clear and demonstrable negative effects, adding 'the parallels between leaned helplessness (Seligman, 1991) and children with reading difficulties is striking'. Butkowsky and Willows (1980) claim that good and bad readers have different attribution style, leading poor readers more likely to blame themselves for failure and attribute success to luck, with poorer expectations of success and responding more negatively to failure than good readers. Such poor attribution style has been correlated with depression (Gilbert, 1992; Seligman, Abramson, Semmel and Baeyer, 1979). Bandura et al., (1999) found a significant negative relationship between children's feelings of social and academic efficacy and levels of depression, academic achievement and problem behaviour.

According to Scott (2004) all dyslexics have some form of stress-related disorder and there is no such thing as a 'stress-free dyslexic' (p. 158). She suggests that a useable way forward for counsellors is to treat dyslexics as having either a 'post-traumatic stress disorder' (PTSD) or with the theory of 'daily hassles'.

Duane (1991) and Fawcett (1995, p. 14) note 'in terms of emotional stability, the literature suggests a threefold increase in psychiatric diagnosis in children with dyslexia, in particular of conduct disorders and depression'. Fawcett suggests that such problems are a natural sequence of year-on-year school failure.

Osmond (1994, p. 28) notes one dyslexic child, 'I would get stuck again and again. I couldn't understand why I couldn't do the work when everyone else could. I just wanted to cry and I thought 'I am thick'. I started to get depressed. I would stand in a queue waiting for my book to be marked, watching all the others getting ticks for their work. It would be my turn and I'd get Xs and be told to redo the work. I would still get Xs and I'd be told that I wasn't trying'….I never knew that I'd done it wrong...I felt angry at times and I wanted to scream or even hit out but I didn't because I'd end up in trouble and there would be a letter sent to my mum and dad and they would be angry with me'.

Depression is a frequent complication in dyslexia, according to Ryan (1994), Burden (2005) and Scott (2004). Although most dyslexics are not clinically depressed, children with this type of learning difficulty are at higher risk of intense emotional feelings of pain and sorrow (as found in Alexander-Passe, 2004b, 2008a). Evidence suggests that dyslexics commonly manifest low self-esteem, explaining why many dyslexics (especially female) internalise such sorrow and pain. Depression in school-aged children may be manifested by their being more active in order to cover up painful feelings (extrovert) or their being loathe to enjoy anything from their day (introvert). Both types will manifest negative thoughts about themselves and see the world in a very negative way.

Ryan (1994) notes that depressed dyslexic children tend to have three similar characteristics: (1) they tend to have negative thoughts about themselves, i.e., a negative self-image; (2) they tend to view the world negatively. They are less likely to enjoy the positive experiences in their lives. This makes it difficult for them to have fun; and (3) most depressed youngsters have great trouble imagining anything positive about the future. The depressed dyslexic not only experiences great pain in his present experiences, but also foresees a life of continuing failure.

However, Grigorenko (2001) has observed that learning disabilities are observed in individuals with depression and has also commented that depressive disorders are elevated in youth with learning disabilities. In addition he notes that between 14-32% of youth with learning disabilities are depressed, but it also means that between 78-86% are not. Scott (2004) asks what variable is missing in understanding such a correlation, she suggests that social isolation is the missing variable. As Kennedy, Spence and Hensley (1989, p.562) notes depressed children often have 'deficits in social skills and interpersonal relationships' due to being rejected or isolated by their peers. Thus it is the dyslexics who are isolated and rejected by their peers who will be the most vulnerable to depression.

There is very little research that actually investigates a correlation with dyslexia; the majority of empirical references are based on observations (Scott, 2004; Ryan, 2004; Duane, 1991; Rutter, 1983) rather than actual studies. Three such studies exist which investigated depression with dyslexic children and teenagers, and adult dyslexics discussing their childhood. Boetsch, Green and Pennington (1996) found that the reports of depression by parents and teachers with primary school children, were not confirmed by children's own self-reports of depressive symptomatology. The second study, by Alexander-Passe (2006, in press-3) suggests gender coping differences amongst N=26 dyslexic teenagers between the ages of 15-17yrs old, using standardized measures of coping (Endler and Parker, 1999) and depression (Beck, Steer and Brown, 1996), see Tables 1 and 2. According to the Beck Depression Inventory-II, a depression rating are as follows, minimal between 0-13, a mild rating is between 14-19, moderate is between 20-28 and severe is between 29-63. This is the first study on dyslexics and teenage dyslexics, using standardised measures of both

depression. Females scored higher in emotional coping and depression than dyslexic males, who preferred Task-orientated coping. It is interesting that the Females scored high in all three forms of Avoidance (General, Distraction and Social Diversion), suggesting that withdrawal is a main defence mechanism at this age.

Table 1. Beck Depression Inventory (Beck et al., 1996) – raw mean scores (SD) in a study of N=19 Dyslexic teenagers in Alexander-Passe (in press-3)

	Raw Score
TEEN DYSLEXICS (ALL) N=26	12 (10.8)
TEEN DYSLEXICS - MALES N=17	9 (8.8)
TEEN DYSLEXICS - FEMALES N=9	17.2 (12.3)

Table 2. Coping Inventory for Stressful Situations (Endler and Parker, 1999) - percentile mean scores (SD) in a study of N=26 Dyslexic teenagers in Alexander-Passe (in press-3)

	Task	**Emotion**	**Avoidance**	**Distraction**	**Social diversion**
TEEN DYSLEXICS (ALL) N=26	60 (25.4)	64.5 (31.9)	53 (30.1)	48.1 (29.8)	52.8 (26.7)
TEEN DYSLEXICS - MALES N=17	61.2 (26.5)	61 (34.9)	47.7 (24.7)	41.1 (27.5)	45.9 (27.9)
TEEN DYSLEXICS - FEMALES N=9	57.7 (24.6)	71.6 (24.7)	63.6 (29.6)	60 (32.1)	66.6 (18.5)

The third is a qualititative investigation by Alexander-Passe (in press-1) researched adult dyslexics (N=7) with a clinical depression diagnosis, the study investigated the emotional damage caused by coping with a learning disability such as dyslexia. Whilst the study investigated adult dyslexics with in-depth interviews, it was clear that the seeds of their depression came from childhood or from their teenage years. The overriding theme of this study was that the stress of both the academic and social curriculum of school can drive some dyslexics to depression and in some cases self-harm and attempted suicide. He notes, 'in every interview one sees that either cries for help (especially in chidhood) have been ignored or that the participants have not had anyone to confide in. Depression only happens after a large number of strategies have failed to produce the help needed and is either a last desperate cry for help or a total rejection of the help offered and they conclude that ridding society of their problems would be the best for everyone concerned (suicide attempt)' (p. tba). All of those taking part were identified as adults and thus to a greater or lesser extent they had experienced childhoods of thinking they were stupid and suffered at the hands of numerous teachers they had passed through. 'It was really surprising that apparently none of the ten or fifteen teachers they would have had and formed close relationships throughout their childhood, never realised the child in their care had problems learning and attempted to ensure that something was done (e.g. diagnosis)' (p. tba). This can serve to illustrate problems in the mainstream educational system for this to have happened. The total breakdown of help experienced by the participants of the study, along with parents lack of faith in their own knowledge that their child had problems, was felt by Alexander-Passe to need to be highlighted. 'Camouflage, denial and perfectionism were found to help participants deal with present day life (as adults) and such rewriting of their childhoods correlate with the defence mechanisms and strategies' (p. tba) this was also found by Vaillant (1977).

Alexander-Passe (in press-1) suggested that the secondary manifestations of dealing with dyslexia may be a significant factor for why some individuals develop depression and others do not. The study identified gender differences, suggesting that depressive female dyslexics felt isolated by their peer group, withdraw and self-blame, whilst depressive male dyslexics used denial and helplessness. In both genders, avoidance of words, tasks and situations were commonplace and any failure could bring back childhood memories of 'I'm stupid and unworthy'. 'Returning to school twenty years later to meet their child's teachers, was found to be fraught with danger as the smell and layout of the building made their own childhood feelings of helplessness resurface resulting in many avoiding such situations' (p. tba), along with such feelings affecting their parent/teacher relationship.

Self-Harm and Suicide

In a world where dyslexics are unable to control many aspects of their lives (more so in young dyslexics), self-harm by diet, alcohol abuse or cutting themselves is a common means to have control over their bodies, as noted in Alexander-Passe (in press-1) and Scott (2004).

When children begin to withdraw or are extremely quiet or highly active and agitated, suicide may be seen as an option to dyslexic children, as a result of excessive bullying and rejection (Winkley, 1996). Scott (2004) suggests that problems related to dyslexia maybe a cause of suicide, whilst real numbers are unknown. However as little research has been conducted in this area, numerous newspaper reports and anecdotes are the only real data to go on. Scott (2004) notes that many suicides don't leave notes and also suggests that many cases of dyslexia led suicide are not recorded as the children are unable to write suicide notes, thus real figures are unknown. Correlations between bullying, school failure, pressure to achieve academically, peer rejection, feelings of frustration, depression, guilt and hostility have been correlated with children's suicide (Thompson and Rudolph, 1996; Harrington, Bredenkamp, Groothues, Rutter, Fudge and Pickles, 1994). Thompson and Rudolph (1996, p. 446) go on to note that children with 'learning disabilities or other learning difficulties that cause constant frustration are more likely to attempt suicide…gifted children may attempt suicide because their advanced intellectual ability makes relating to children their own age difficult'. Evidence suggests that attempts of suicide increase during school term and decrease during school holiday (Winkley, 1996) and that the attempts increase in May and June to correspond with GCSE examinations.

Alexander-Passe (in press-1) found that dyslexics may choose an attempt of suicide to firstly try to end their daily hell of being perceived abnormality, but secondly to rid the world and their parents of the strain of dealing with an abnormal learner and the associated pressures that arise from having a disabled child. Lastly there are feelings of not being worthy to be members of their family or society, such a perception is connected to their low self-worth or self-image.

Peer (2002, p. 32) notes that the six cases presented to him from a dyslexic forum suggests that such children are fragile, vulnerable and feel the ramifications for failure are enormous. Riddick (1996, p. 107) describes how the problems encountered because of dyslexia was enough for dyslexic children to want to kill themselves, noting one mother 'he wanted to be dead, there was nothing for him. He wanted his tie so that he could hang himself'.

Drug Abuse

There is very little empirical evidence to rely on to investigate drug abuse amongst dyslexics. Scott (2004, p. 169) suggests that, in general, 60% of alcoholics, mainly men, start drinking due to anxiety. As a counsellor to dyslexics, Scott found high frequencies of drug and alcohol-related anxieties amongst child and adult clients. She postulates that dyslexics are more likely than non-dyslexics to use drink and drugs to cope with anxiety. Scott found a significant proportion of dyslexic children, as young as 13 years wishing to beat their addiction to tobacco, cocaine, marijuana, ecstasy, drink and anti-depressants. She has also come across drug, drink and food abuse as a means to reduce anxiety amongst children and adolescents with dyslexia. In girls anorexia and bulimia are used which represents a need to exert personal control their bodies, in a world where they are unable to control any other segment of their lives (e.g. school and home life). It may also be used as a cry for help as having such disorders gains the attention of parents and health officials, but in similar ways to truancy and behaviour manifestations, health and educational professionals will commonly treat the manifestation without looking for the initial root cause. Dyslexics who use drugs with as part of an Emotional defence mechanism are looking to escape their daily hell of being abnormal.

Post Traumatic Stress Disorder and Daily Hassles

According to According to The National Institute of Mental Health (2008b) Post-traumatic stress disorder (PTSD) is an anxiety disorder that some people develop after seeing or living through an event that caused or threatened serious harm or death. Symptoms include flashbacks or bad dreams, emotional numbness, intense guilt or worry, angry outbursts, feeling "on edge," or avoiding thoughts and situations that remind them of the trauma. In PTSD, these symptoms last at least one month and can be a contributing condition to depression.

'Post-traumatic stress disorder' (PTSD) in children can come from various factors, these include: the sudden exclusion from their peer group; intense anger from a teacher or parent, physical bullying at school; realisation that something unrecognisable is wrong (maybe realising that they are abnormal or unable to learn normally, or being called stupid, lazy etc). There are two forms of PTSD, which Scott (2004) suggest dyslexics suffer. The first is Type 1 (an acute, single-impact traumatic event) and Type 2 or complex PTSD (a series of traumatic events or prolonged exposure to a stress or stressor), both are listed in the DSM-IV (APA, 1994). PTSD is a widely researched aspect of psychology (see Rose, 2002; Stallard, Karwit and Wasik, 1999 for reviews). PTSD is categorised by being the sudden and irrevocable perceptive change of the world from one that is safe and predictable to one that is dangerous and random. Individuals are as traumatised as if they had been in a major car crash. The behavioural effects of PTSD come from repetitive and intrusive thoughts and can be triggered by vision, sound and smell (as noted by Miles and Varma, 1995; Riddick et al., 1999). Yule, Bolton, Udwin, Boyle, O'Ryan and Nurrish (2000) found only 25% of PTSD sufferers had recovered after five years, 33% after eight years and 59% warranted a lifetime diagnosis.

Whilst Perrin, Smith and Yule (2000) note correlations between a sufferer with PTSD with concentration, memory and reading problems, it is unclear if the PTSD caused such difficulties or whether they were there before, a 'chicken and egg scenario'. Tsui (1990) suggests that PTSD is related to academic performance and that the PTSD was the cause. Scott (2004) suggests this is not clear cut and that the PTSD might have been caused by the secondary effects of having dyslexia, a view this author supports.

The second theory presented by Scott (2004, p. 164) is that of 'daily hassles', an opposite concept of PTSD, in that the stress of daily inconveniences are 'even more perilous in the stress lexicon than major life events'. As noted by Lu (1991), Lazaraus (1984), Chamberlain and Zika (1990) the risk of persistent hassles that are endlessly present in the sufferers life are a powerful predictor of psychological distress and have been likened to 'living permanently in a cloud of small, biting mosquitoes' (Scott, 2004 p. 164). Morgan and Klein (2000) observed that even adults with minor dyslexic symptoms are placed under extra stress from the constant effort needed to perform ordinary daily tasks, such as reading instructions to understanding conversations. To support such a concept, Winkley (1996) asked dyslexics at junior school to rank the most stressful things that they can think of, 12 of the 16 stressors mentioned are related to the experience of being dyslexic (getting lost, being left alone, being ridiculed in class, tests and examinations, breaking or loosing things, being different, performing in public). Harrison (1995, p. 116) herself a dyslexic, suffered high levels of stress in her own life, in work with a group of PTSD sufferers she noticed 'the similarities struck me; although I realise they are not as extreme for me. The social dysfunctionality also is, in them exaggerated, but nevertheless comparable to my own experiences and those I have known with other dyslexics'.

Self-Blame

When unidentified dyslexics find school hard and are at a loss to the reasons for their difficulties, the first place they look is to themselves. Is it me? As noted earlier, the interview study by Alexander-Passe (in press-1) investigated seven dyslexics who also had dyslexia. There were several individuals who used self-blame as a strategy to deal with why they were having difficulties in life. Whilst as adults they knew they were dyslexic and would have such problems, as soon as they failed they beat themselves up about failing, calling themselves 'stupid' and in some cases self-harmed. Self-blame is perceived by some as logical reasoning, as who else could be at fault. However, in these cases it leads to a self-perpeptual conceptualisation that their teachers were right and 'that they would never amount to anything in life'. Self-blame could be positive as it can be used to remonstrate oneself to do better next time.

In Butkowsky and Willows' (1980) study, poor readers (including diagnosed and undiagnosed dyslexics) gave up more easily in the face of difficulties. Average to good readers attributed their success to their ability, while poor readers attributed their success to luck. Poor readers tended to blame themselves by attributing failure to their own incompetence, and success to environmental factors such as luck. There are also correlations to 'learnt helplessness' and 'attribution style' (Diener and Dweck, 1978; Miller and Norman, 1978).

Alexander-Passe (2006) investigated three types of coping among dyslexic teenager, the resulting factors included: Task, Blaming and Avoidance. It was hypothesised that the Blaming factor needed to be divided into self-blame and blame others, to deal with the results

of the parallel interview study (Alexander-Passe, 2004b). The results suggested that beating yourself up about your difficulties is natural to some and when one is unaware of dyslexia or have no dyslexic friends and peers, internalization is a nature way to find meaning in a world without meaning (Edwards, 1994; Scott, 2004). Dyslexic peers are important to a young dyslexic, as they learn they are not alone. This is even more important when there is non-dyslexic sibling, as parental comparison is likely.

Perfectionism

Perfectionism is a means to which dyslexics feel they can protect themselves from the outside word by correcting and recorrecting drafts of essays or forms until such a point they feel willing to let others see them and make judgement. In the case of dyslexics who have problems with reading, writing and spelling this can mean: making sure they have read the question correctly and read it many times; to making sure the writing is neat enough and with the correct punctuation and thus rewriting the essay or form several times; to spending an inordinately large amount of time checking every word in a dictionary to make sure they are all spelt correctly (as also found by Alexander-Passe, in press-3 and Scott, 2004). Such strategies may be possible when the academic workload is low but as children move from primary to secondary school and university to the workplace, such a time consuming task is not possible. The stress dyslexics put themselves under to avoid stress via perfectionism is high and can turn out to be a double edge sword. Because the work produced is too a high standard, teachers are unaware of the huge workload needed for such productions and disregard any notion of unidentified learning difficulties. They might be better served to fail and get low marks with the resulting low self-esteem and thus be visually disabled than to continue putting themselves under such stress with resulting anxiety and possibly depression, by hiding their difficulties.

Bed-Wetting

Nocturnal enuresis is a condition in which a person who has bladder control while awake urinates whilst asleep. The condition is commonly called bed-wetting and it often has a psychological impact on children and their families. Children with the condition often have low self-esteem and weak interpersonal relationships, poor quality of life, and poor performance at school (Von Gontard, 2004; Van Hoecke, Hoebeke, Braet and Walle, 2004). Von Gontard (1998) linked enuresis with delayed speech, motor and speech milestones (each correlate to dyslexia). Miles (1994, p. 144) noted a case of an 8 year old dyslexic child who was ‘still bed-wetting and full of nervous twitches’. Scott (2004, p. 171) postulates that ‘30% of dyslexic children suffer from enuresis, some of it very severe including total soaking of the bed, every night, for months on end’.

If parents are aware that a bed-wetter has no medical problems but is having difficulty with: paying attention in school; concentrating on academic material; impulsive behaviour; fidgetiness; intermittent explosive tantrums; or conduct disorder, then dyslexia may be the underlying problem. Therapeutic Resources (2004) notes that in a study of 1822 children with attention-deficit hyperactivity disorder, (a condition with co-morbidity to dyslexia, see Fawcett and Peer, 2004; Gilger, Pennington and DeFries, 1992; Ramus, Pidgeon and Frith, 2003), 48% had been bed-wetters.

Watkins (2004) believes ‘…sometimes enuresis (bed-wetting) may be due to anxiety, a change in the home situation (such as the birth of a sibling) or an emotional trauma. We

particularly look for emotional factors in children who were previously dry and start to wet again. A child with shaky bladder control may be more likely to revert to wetting when under stress'

Scott (2004, p. 171) suggests that bed-wetting (enuresis) in dyslexic children is common (approx 30% of all dyslexic children) and usually starts when they start to have problems at school and can go on for months at a time. Miles (1993) notes two cases along with Thomson (1995) and Von Gothard (1998) suggests an association between delayed motor and speech milestones, which can be found in dyslexics. There is double stress from the bedwetting, firstly from the act itself and secondly from the reactions of others, including peer and sibling teasing. Rutter (1983) and Rutter, Tizard and Whitmore (1970) suggest a third of such children also have emotional or behavioural problems and a higher rate of psychiatric disorders (Von Gontard).

Riddick (1996, p. 136) notes a teachers giving one of her pupils 20 hard spellings a week as a form of bullying, the child's mother commented that 'he was worrying himself sick before the spelling test. In fact, he started bed-wetting because of the pressure of the spelling test, she destroyed his confidence'.

Povey and Todd (1993) suggests that at 7 year old, dyslexic children first begin to be a cause of concern to both parents and teachers experience difficulties at school, this corresponds with the peak of requests for dyslexia assessments (8-11 years) according to Scott (2004). This age responds with the Freudian change from 'latency' to 'age of industry' stages of child development (Freud, 1961; Erickson, 1968). Trauma at this age can affect the progression from one stage to the next. Jacob (1986) suggests problems at this stage can cause: negative self concept; feelings of inadequacy; feelings of inferiority; confused values; dependency among others; and can cause a regression to the 'genital' stages of development as seen as a toddler, recognised as clinging to family and mother for safety. Thomson (1995, p. 41) notes the stress from a school that doesn't assist the dyslexic child can spill into home life and can result in manifestations such as temper tantrums, aggression and bed wetting.

McLoughlin et al., (2002) and Patton and Holloway (1992) and Scott (2004) suggest that dyslexics are generally immature and slow developers, even as adults they are still passive to their parents and allow them an unhealthy control over their lives. They suggest that they resist the notion of growing up as an empowerment decision. Scott (2004) suggests that this happens in several ways: firstly all major organisations helping dyslexics are run by non-dyslexics (as seen in the British Dyslexia Association) and thus they are perceived to be incapable of standing up for themselves; secondly due to their difficulties they are unable to protest in printed media and thus only use verbal media (how a toddler protests in a world of adults); dyslexia is the only disability defined by school learnt abilities of children and thus until they have mastered the abilities of children they can't be adults; as mothers are the main saviour of the dyslexic child, they remain in adulthood 'mummy's little child'; the dyslexic is often seen as creative and this is seen as the one consolation prize for dyslexia, however trapped by this concept they are unable to live in the real adult world of newspapers with political status and education; lastly work published by dyslexics is predominately done unedited and without changing incorrect spelling and grammatical mistakes, thus they are perceived to be childish.

Congdon (1995, p. 93) notes other manifestations in dyslexics of 'emotional disturbance arising from the problem are: enuresis (bed-wetting); stammer; sleep-waking; asthma; and

various physical symptoms, such as vomiting and recurrent abdominal pains for which no physical cause can be found'.

Riddick (1996, p. 48) notes a 7yr old boy who had a new teacher unsympathetic to his dyslexic difficulties, he became distressed and his behaviour at school deteriorated, his mother noted he cried himself to sleep each night, he started to wet the bed and came home shaking (with fear) if he had a spelling test to revise for the next day. His mother was frequently contacted about his bad behaviour at school but by then his behavioural problems were seen as the main source of concern. When asking his teacher whether his behaviour could be down to a learning difficulty, the response received was 'rubbish he's just very immature, when he learns to behave properly and knuckles down to the work he'll be ok'. The next year his teacher was more sympathetic and identified him as dyslexic, helped him and his behaviour improved again.

Stammering

According to Glauber (1958) stammering is a defence mechanism where personality is disturbed and speech reverts to its infant pattern. The causes of stammering are still unclear, with many researchers looking at the condition as a neurosis, caused by anxiety, as a learned behaviour, or as an organic disorder (genetic, central nervous system, sequencing and timing, temporal programming).

Children turning to regression as a defensive mechanism would have experienced numerous stressful situations where they had tried to cope, but failed. When the situation gets just too much for the young preschooler, regression starts and they turn inwards to protect themselves, just as a turtle will when sensing danger. Blood, Blood, Frederick, Ertz and Simpson (1997) found that stammerers scored high on communication apprehension and use of emotion-based coping strategies. Alexander-Passe (2006) found high use of emotion-based coping among dyslexics, compared to the general population.

Stammering is a psychological and physical cry for help, and sadly these secondary symptoms are often treated without investigation of the primary cause. Thus continuous regression is likely in many cases. Unfortunately, such symptoms become a life long 'hard-to-break' habit (Van Riper, 1982, Conture and Caruso, 1987, Starkweather, 1987), and many perceive anxiety as the cause of stammering (Messenger, Onslow, Packman and Menzies, 2004; Ezrati-Vanacour and Levin, 2004; Bloodstein, 1987). Individuals react to situations and academic pressures differently; thus, it should be noted that what may be stressful to one may not be to another. One could ask, is the person disfluent because of the anxiety, or is the anxiety the result of the act of being disfluent?

Johnson (1961) found that disfluent children often came from families where the parents placed strong demands on them to achieve – speech is a major milestone and thus could be interpreted to be a predictor of later achievement. However, not all children from demanding homes become disfluent and visa versa, so there must be other factors (such as possible dyslexia).

Both dyslexics and stammerers are affected by their conditions (avoidance of difficult tasks such as reading, writing and speaking situations). Their avoidance strategies could be explained as defences against unexplained and inappropriate anxiety. Importantly, Van Riper (1982) noted that, 'The neurosis symptoms (stammering) may at first alleviate the unpleasantness (by reducing academic pressures) but soon contribute towards it (further

unhappiness)', in a form of 'conflict and trap' scenario, which has also been noted by Starkweather (1987).

Orton (1937) one of the pioneers of dyslexia study suggests that speech delay and stuttering were more common than usual in a sample of dyslexic individuals examined by him, as supported by the famous 'Isle of Wright study by Rutter, Tizard, Graham and Whitmore (1976) with later support from Snowling (1987) and Stackhouse (1990). In contrast, Scott (2004, p. 195) notes 'stuttering is not a commonly observed problem in dyslexics'; what is more common, in her experience, is that some dyslexics can't get their words out quickly enough.

Riddick (1996, p. 104) describes one mother's comments that her son in French lessons…'He's never going to be academic, that's not him anyway. But he's got disheartened and upset because he wanted to get good results like the other children and he wasn't. Then he stammers, and it's an emotional stammer. It comes and goes. But really it's more there than the times it's not'. This suggests that a stammer is an emotional reaction to failure and can become habit forming.

Fawcett (1995, p. 6) notes that when her dyslexic son entered school 'he became withdrawn and introverted, to the extent that his first teacher questioned whether or not he could speak…it soon became clear that something was wrong…with many broken nights...crying with pains in his legs, coupled with poor reports of his progress at school….after diagnosis he regained his talkativeness'. His reaction to his first teacher at school was to develop a stammer (p. 8) as a defensive mechanism to stress at school and feelings of anxiety and fear of failure, as the teacher thought he 'simply refused to complete his work' (p. 8). Fawcett also notes that at the end of the year after external diagnosis his teacher admitted that she misunderstood his problem and had assumed that he was being difficult when he failed to follow instructions. It is interesting to note that as a mother, she allowed her son to stay away from school at times of class testing to 'avoid humiliation which we felt would be counterproductive' (p. 9).

Fawcett (1995, p. 19) also notes a second nervous dyslexic boy that had developed a stammer as a defensive mechanism, however 'his attendant stress and the amount of effort that he constantly needed to input into his work to try and achieve his potential took its toll….by the time he reached GCSE level, he suffered a nervous breakdown, and was simply no longer able to maintain the level of effort needed to keep up'. As a result, his parents removed all stress from his life and are unsure if he will ever obtain work.

Gilroy (1995, p. 65) also noted that two of her adult dyslexic students developed a stammer as a reaction to stress along with another one that lived on bananas and milk 'as it did not make any demands on her' to deal with supermarket shopping.

The cognitive learning theory by Conture and Caruso (1987) suggests that stammering is a result of the relationship a child has between their abilities and their environment; thus, if a child's abilities fall short of what their environment requires (parents, peers or teacher), then they will learn behaviours that will either lower the requirements (e.g. a parent expects less from someone with a known disability such as a stammer) to cope (a threat/response scenario). So disfluency could be a learnt behaviour, especially in situations children perceive as anxious. Thus disfluencies could lower these parental predictions (stress), thus reinforcing the stammer as a useful coping strategy.

Studies (British Stammering Association, 2003) suggest that most children who develop a stammer in early childhood (possibly as a coping strategy for attention) have spontaneous

recovery in 2 out of 3 cases. A third recovers within 18 months of the onset of the stammer and another third before adulthood (both cases possibly as a reaction to the readjustment of academic expectations).

Stuttering could be explained as a learnt behaviour (operant conditioning) as a consequence of punishment and reinforcement. According to Shames and Sherrick (1963), repetitions in stammering which produce desired consequences (e.g. gaining parental attention) will increase in frequency until they become a major feature of communication. However, if the child gets punished, he will take steps to avoid speaking.

DYSLEXIC COPING MECHANANISMS

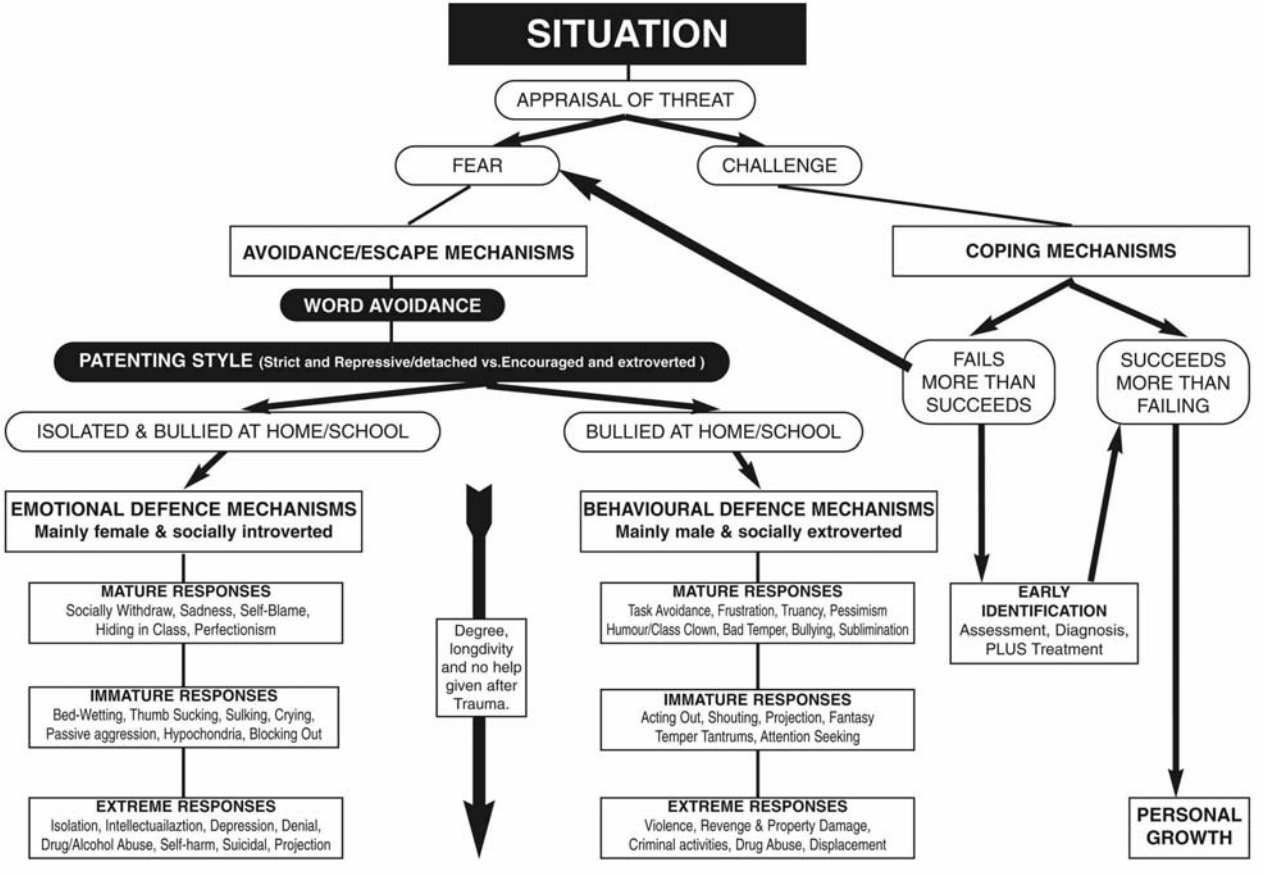
Defence Mechanisms

In a study by the author (Alexander-Passe, in press-2) a hypothetical model (see Table 3) was proposed based on personal experience, recent research (Alexander-Passe, 2004a, 2004b, 2005, 2006, 2008a, 2008b, in press-1) and supporting empirical evidence, as discussed earlier.

Whilst all children when entering education, be it at nursery or primary school, would have been encouraged by their parents on a one to one basis and feel the world is their oyster. As soon as they enter school or nursery they begin to see differences between themselves and others, but most importantly they will either experience success or failure in any new tasks presented to them. Where encouragement results in success in more cases than failure, they will learn that tackling new tasks is exciting and enjoyable. Where they experience failure more times than success, they will feel that learning is an activity they are unable to control, with failure being perceived in the majority of learning environments as something negative and to be feared. However, progressive educational techniques (Montessori, 1965) encourage failure and perceive any failure as an expected aspect and encouraged part of the learning process; in mainstream UK education such views are not valued. Failure and especially public failure is seen as socially unacceptable.

Early screening of children as soon as they enter school (the first 6 weeks of their first term of their first year at school) is seen as a powerful tool in screening for learning difficulties such as dyslexia, ADHD etc Such screening is a means to monitor, diagnose and treat educational difficulties before a child learns that education establishments can be threatening environments. Research suggests that early assessment and correct specialist dyslexia treatment where required, can dramatically reduce adverse reactions to mainstream schools.

Table 3. Hypothetical Model of 'Dyslexia Defence Mechanisms'



Fear of failure is such a powerful psychological concept and can be extremely potent in changing an individual's whole basis of pre-judging new learning situations, or any situation that may risk public ridicule. Once the appraisal is of fear, the body aims to avoid or escape from such threat in various ways; however parental example and the reaction of parents to their own and child's difficulties have a strong impact on how they act themselves. If the parenting style is understanding and supportive, then the child may be encouraged to try and risk again, if the parenting style is strict, repressive and unsupportive, then the child will develop mechanisms to reduce their exposure to any feared stimulus.

First they will try basic word avoidances (e.g. writing less and using simple words); however this commonly is not sufficient to deal with school demands. Mature defensive mechanisms are first likely to be developed and are classed as either Emotional or Behaviourial, with the former being social withdrawal, self-blame, perfectionism, hiding in class and feeling sad. The later are likely to include task avoidance, truancy, frustration, bad temper, bullying, pessimism and sublimination. These are classed as mature responses according to empirical studies (Vaillant, 1977 and Meissner, 1980). Scott (2004, p. 257) notes from clinical experience that dyslexics are outstanding at defence mechanisms and 'the subtext is that you [counsellors] get lost in the smoke screen of the dyslexic's behaviours [Defence mechanisms] and do not notice the failure and vulnerability beneath'. With counsellors noting that you never forget a dyslexic client as they are very cunning, 'refined and slippery' and that commonly their defences can isolate them from others, noting 'in short, we have to notice when the iron defences turns into iron cages'. Alexander-Passe (2006, in press-3) suggests that clear Emotional or Behaviourial differences exist among dyslexics, with gender splits being evident.

The key is understanding why some children choose Emotional or Behaviourial defensive strategies. Experience suggests that children with strict and repressive parents choose emotional strategies, whilst those with parents displaying aggression encourage their children to replicate such aggression at school (aggression begets aggression). Such children learn that its okay to display anger and a bullied child is more likely to bully others or even younger siblings.

However in the primary school, immature Emotional strategies may be evident, these include bed wetting, delayed or regressive language skills, sulking, thumb sucking and crying, hypochondria, passive aggression and blocking out. These regressive strategies are direct responses to difficulties at school and a wish to return to their mother's safety as an infant. Regressive strategies are less common in late primary school however regressive language skills such as stammering are habit forming and can be life-long complaints. Other immature Behavioural strategies may also be evident during primary school years with shouting, acting out, temper tantrums, biting, and attention seeking, as a means to ward away feared stimulus.

Whilst it is unclear how much is too much fear, children and adolescents with dyslexia experience high levels of stress as their dyslexic difficulties also evolves from the classroom into the playground and to socialising outside of school (the whole school experience). Any defence strategies used are cries for help; however these secondary manifestations are commonly treated without looking at the primary causes.

When the fear gets too much and mature and immature mechanisms are ineffective e.g. unable to protect the individual from threats, the individual will look for more complex escape mechanisms, either extreme Emotional or Behaviourial defensive mechanisms, with

isolation, intellectualization, depression, projection, escapism, drug abuse to numb the pain and self-harm by those using Emotional mechanisms.

Such responses are perceived by them as the only means left to them to escape or control the risk in their hostile world and can lead to suicidal tendencies. Those who use Behavioural mechanisms are likely to turn to revenge on teachers leading to violence, displacement, criminal activities, not only repaying the hostility they themselves have experienced from a society they feel excluded from, but also as a means to gain self-esteem from material gain. Alcohol and drug abuse is also common for ticks, voyeurism and part of taking enlarged risks in life.

There are three main keys to this hypothesis: (1) how does parenting style and other factors make individuals choose between Emotional and Behavioural defensive strategies? (2) What factors are needed for individuals to move from mature, to immature and extreme Emotional and Behavioural responses in dealing with situations? (3) are Emotional and Behavioural defensive strategies exclusive or can combined responses be found?

How Does Parenting Style and other Factors Make Individuals Choose Between Emotional and Behavioural Defensive Strategies?

Children learn explanatory styles from those around them, if parents or peers are anxious, depressive or aggressive towards them, then they have been shown successful mechanisms which they then will replicate in similar stressful situations. The same is true for parents and peers who are relaxed and show concern for their child's learning problems, the child will learn such lessons well and will replicate in similar situations. One question that is always posed is how much influence does a parent have when their child goes to school? Whilst their child's peers have a strong influence (especially from late primary school onwards), a parent can support or make it difficult for their child to mix socially with any undesirable peers, however the parenting style seen before a child goes to school can instil a basis for their coping mechanisms. Factors such as bullying in the classroom by teachers and in the playground by peers can teach behavioural coping to vulnerable children. Isolation in the playground and after school can made a vulnerable child turn to emotional defences as a means to cope with their feelings; it can also confirm a child's concept that they are unloved and unwanted.

How parents deal with homework and allowing their child to relax after school is important and can provide a safe haven from a hostile and threatening day at school. However homework interactions can be fraught and difficult situations as parents find helping their dyslexic child frustrating and in many cases feel helpless and unqualified in helping them. Many parents use techniques they themselves used at school just confirms that traditional teaching methods are unsuitable for dyslexics and again unwittingly their child relives the anxiety and frustrations they experience at school.

A main factor can be gender, in that more girls turn to emotional defences than boys, with boys choosing more macho behavioural mechanisms. Not only at home but at school, gender is a strong factor and is reinforced in the classroom, playground and role-play. Brody (1985) reports that parents encourage sons to be more aggressive but unemotional, but encourage daughters to be emotional but non-aggressive. Feschbach (1970) therefore suggests that girls believe it is inappropriate to express anger openly, as compared to boys and that boys will tend to think it is inappropriate to express fear so will use internal attempts to control or hide that fear. Gender differences are apparent in both dyslexic and norm population and suggest

that females will choose more emotional and males will choose more behavioral defence mechanisms (Alexander-Passe, 2006, in press-3).

What Factors are Needed for Individuals to Move from Mature, to Immature and Extreme Emotional and Behavioural Responses in Dealing with Situations?

When a child starts to use defence mechanisms they are saying to themselves and others 'I can't cope, help me', whilst some defences are shouting, others are whispering and thus if they are not heard and responded to then the feelings of failure are reinforced. Factors such as the degree and longevity of the trauma (e.g. a bad supply teacher may only be there a week) are important factors. However, if the trauma is continuous (e.g. bullying or unfair year teacher/tutor), then extreme responses may be used to escape the threat.

Scott (2004) suggests a child can be bullied twice, first by the actual bully and secondly by parents/teachers ignoring the cries for help. If help is not given their feelings of poor-concept (e.g. 'I'm stupid, I'm lazy') are confirmed and they then go further down a spiral of lower self-concept to a point of no return. Such points either turn to depression, suicide if they blame themselves or criminal activities if they blame others and society for the injustice in their lives.

Are Emotional and Behavioural Defensive Strategies Exclusive or can Combined Responses be Found?

It is hypothesised that initial mature responses may be interchangeable between Emotional and Behavioural defence mechanisms, however as each is reinforced they begin to separate into strongly Emotional or strongly Behavioural. Some mature responses may be found by both defensive mechanism models, as it is the basis of avoidance for dyslexics. This may take the form of writing less, choosing to use shorter easier to remember words in essays, losing books and forgetting homework assignments. Whilst some may be attributed to core dyslexic deficits e.g. short term memory problems, on the whole they are suppressive defence mechanisms

Could the Decision to Choose Emotional or Behavioural Defence Mechanisms Happen at Childhood?

Empirical studies (Donaldson et al, 2000) suggest that defence mechanisms are stable over age and gender. However, Vaillant (1992) suggests differences in age appropriate defense mechanism, with: 'psychotic' (or extreme) defenses being in healthy individuals till 5 years old and common in adult dreams and fantasies; 'immature' defenses in healthy individuals between 3-15 years old; 'neurotic' (or extreme) defenses in healthy individuals between 3-90 years old; and 'mature' defenses mechanism in healthy individuals between 12-90 years old. Whilst there is very little difference in Valliant's scales, it cannot be taken for granted that all defenses are stable over time.

A recent study mentioned earlier (Alexander-Passe, 2008a, b) compared the sources and manifestations of stress with dyslexic and non-dyslexic siblings, using a standardized measure by Helms and Gable (1989). Results investigated the data by age and gender. Results indicate that the manifestations of stress vary according to age. High emotional and physiological manifestations were found in the primary school group suggesting immature emotional defence mechanisms are used e.g. fear, shyness, loneliness, nausea, tremors

because of the school experience. In the early secondary school group there was no clear manifestation differential, thus, it is difficult to assess their primary defence mechanism and these were similar to the sibling control data. However the late secondary school group suggested that emotional manifestations or defence mechanisms were again used as the primary strategy e.g. fear, shyness, loneliness. Interestingly in the control (late secondary school) group, behaviour e.g. acting out and being disrespectful was the primary manifestation and defence mechanism. With gender and age variables. Whilst these sub-groups are small, they suggest no gender differences at an early age (primary school group) because of the school experience. The results were due to high teacher, academic stress with a poor academic concept. In the early primary school group, dyslexic females showed high behavioural manifestations e.g. being hurtful, disrespectful or striking out as a defence mechanism, compared to dyslexic males. The data suggests that this defence mechanism was used up to the point of leaving school. Interestingly the data suggests that dyslexic males use less behavioural and more emotional and physiological manifestations at the point of leaving school e.g. fear, shyness, loneliness, nausea, tremors as a result of the school experience.

One explanation may come from Hales (2001) who has studied post-private educational psychologist diagnosis of dyslexia. He found that the mood in nearly half of all those diagnosed changed to denial, depression and resentment and that this might be caused by a large percentage of schools ignoring the diagnosis. Severe and immature defences may be used in the primary school as dyslexics are getting to know and deal with a world they have difficulty interacting with. Bu early secondary school, dyslexics they are hopeful of the changes in help following assessment, however when this does not happen, they become emotionally defensive again (as found in the late secondary school data).

In Hales (1994) a study was conducted with various aged dyslexics (6-8, 8-12, 12-18 and 18+years) using the Cattell Sixteen Personality Factor Questionnaire (Cattell, Elber and Tatsuoka, 1970) on N=300 dyslexics (75% Male and 25% Female), ranging from 6 to 58 years old. A selection of the results shown in Table 4 have been drawn from his published diagrams and are approximate, but indicate gender and age differences in how dyslexics cope from childhood to adulthood. Taking a global view, dyslexics in all age groups score in the average range, except for 8-12year old dyslexic females who score very high in the apprehension scale suggesting they worry about things, feel anxiety and insecurity; this may indicate difficulties changing to secondary school, difficulties making new friends and dealing with the increased academic pressures. High self-confidence scores for 6-8year old dyslexic males suggest they enter school with high confidence and optimism, however this dramatically drops to average levels as they come to terms with having learning difficulties, however to norm levels. Hale's analysis of the full data suggests that children between 6-8 years old feel tense, frustrated, inferior to peers and lack an obligation to conform to society's value schemes which may be due to their inability to conceive why they find school difficult and others do not. At between 8-12 years old they have already resigned themselves to having learning difficulties, and are unmotivated and anxious due to demands made on them. He also notes they are unmotivated to control their own emotions and may have fits of anger. Children with this score are normally have problems socially with peers and problems interacting in the classroom as these are repeated chances for them to appear inadequate. In the 12-18 year age group, the scores still indicate they prefer to work by themselves and show high independence. With the 18+ year group, high dominance scores suggest such a group are more assertive and independent-minded, however also solemn, unconventional and rebellious.

Hales remarks these this may be part of their coping strategy to reduce hostile environments that would affect their self-esteem.

Table 4. Data taken from Hales (1994) Dyslexics of various ages in measures from the Cattell 16 Personality Factor Questionnaire. N=300. Range 1=low and 10-high, 3.5-7.5=average.

	Dominance	Tension	Independence	Emotional Stability	Apprehension	Self-confidence	Super-ego strength
Females							
6-8yrs	5.25	6.25	5.6	5.5	5.25	6	5.6
8-12yrs	4.5	6.1	6.7	4.25	9.4	4.25	5.25
12-18yrs	6.5	5	5	4.5	6	5.75	5.5
18+yrs	7.5	6.5	4.5	5	6.5	6.5	3.75
Males							
6-8yrs	5.5	6.75	4	4.8	6.75	7	3.75
8-12yrs	5.5	5.5	5	4.6	4.5	5.5	4.1
12-18yrs	5.6	5.2	6.25	5.4	5	5.75	5
18+yrs	6.7	5.4	4.4	5.4	4.75	6	5.4

The data from Hales (1994) indicates defence strategies are already being used from an early age (6-8 yrs) and that school is perceived as a threatening environment that needs to be controlled, results also suggest that withdrawal in both school and social environments and signs of perfectionism confirm several of the aspects found in the hypothetical model. Overall, there is evidence that DDMs develop early on in childhood and these vary according to age and gender, with many males choosing behavioural and females emotional defence mechanisms to deal with the hostilities they face.

How Dyslexic Defence Mechanisms (Ddms) Compare to those of other Researchers?

The DDMs as conceptualised have been developed from various research studies (Alexander-Passe, 2004a, 2004b, 2005, 2006, 2008a, b, in press-1, in press-3). They suggest that dyslexics will either predominantly use emotional or behavioural coping strategies/defence mechanisms. The closest defence mechanism models to that of the findings of empirical studies and the DDM are specifically those of Vaillant (1977) and Meissner (1980); however they suggest no difference between emotional and behavioural mechanisms in their immature, mature and neurotic models. Even though Vaillant's model is now incorporated in to the DSM-IV (APA, 1994), it was created from investigating a small sample (N=100 male Harvard graduates), and thus are still questionable if such models can be generalized to not only male and female as a whole, but specifically dyslexic populations. Both Vaillant's and Meissner's models proved problematic in the case of dyslexics (child, adolescent and adult models) and thus modifications were made. In addition, an initial strategy of using word avoidances has been suggested which is used when first faced with possible challenges to their self-concept. Dyslexics, due to the reactions of educators and peers to their invisible learning difficulties and the resulting emotional reactions to maintain their self-esteem, are a unique group and it is hypothesised they require a different framework in understanding how such individuals deal with constant stressors.

The DDM framework notes the importance of early identification and how important it is for teachers to identify children failing more than they are succeeding in the classroom. Studies support self-efficacy in the classrooms (Bandura, Pastorelli, Barbaraneli and Caprara, 1999; Cramer and Gaul, 1988; Burns, 1982; Frydenberg, 1999) suggests that children whose belief system is positive are more able to apply themselves to the curriculum and access the support they need.

DISCUSSION

The empirical evidence reviewed in this chapter points to various issues and studies, these can be grouped into 'supporting evidence' and 'true evidence' to support the chapter title 'Dyslexic Children and Depression'.

The supporting evidence is both broad and extensive. Broad in that it points to various anxiety led evidence of both first hand accounts and experiences in the field by researchers, teachers and counsellors that a dyslexic child exhibits the various symptoms and expressions of depression, thus in-direct correlations. Whilst not all evidence points to depression per se, and may be of varying quality and integrity, it should be noted that depression is made up of a collection of symptoms exhibited over a period of time. There is a extensive body of evidence which suggests that it has been experienced by many researchers in the field and through publication, been subjected to multiple peer-reviewed processes.

The empirical review also suggests there is a small but growing body of 'true evidence', mainly from this author, that investigates dyslexia and depression directly. Thise author has experienced at first hand the difficulty of sourcing not only teenagers with depression but teenagers with depression and dyslexia, thus it is no wonder that the body of direct research on dyslexia and depression is small.

Based on the evidence presented in this chapter, there seems to be a strong suggestion that the relationship between dyslexics and their peers, and school is a factor causing both stress and anxiety. It is these interactions that inform the young dyslexic that they learn differently to those around them, thus they are learning in an abnormal way. Unfortunately mainstream education has moved away from progressive methodologies (e.g. Montessori) where failure is part of the learning process and is encouraged, to a league table methodology that encourages success at all costs and in the quickest way possible. Thus a strict national curriculum, as in the UK have been prescribed by governments and educationists to get from 'a' to 'c', without going via 'b'. Such a route not only means learning by rote without a good grounding of the reasoning and structures behind such knowledge, but also that experimental learning does not happen, in essence there is a failure to learn 'understanding'. Such a methodology leaves no choice but to quickly label children as successful or failures at picking up knowledge, based on test results. Competition is the name of the game, not only in the classroom between peers, but between schools locally and nationally in the form of published league tables. Those children unable to learn in the prescribed way are labelled as lacking and in many instances removed or excused from local and national examinations which league tables are based on.

Whilst teacher training in the last few decades has changed to allow mixed-ability classrooms, it has been at the cost of not only specialist schools for those unable to learn in

the prescribed way, as set by educationalists, but also specialist 'grammar' schools for those with higher intelligences who would appreciate a faster learning program, much of this has been done without additional training or resources to cater for such children. Such a change to mixed-ability classrooms has been at the cost of both these groups and this results in teachers teaching to the middle sixty percent of the class that learn in 'normal' ranges. These children who are unable to cope (inclusing dyslexics) and those too able to cope (those with high intelligence or aptitude for what is being taught) are bored. Both groups are not served in the modern classroom which can be as large as forty children, all requiring attention at the same time.

Dyslexics who commonly form the bottom twenty percent of the classroom (in varying severities) struggle to keep up and according to the evidence presented in this empirical review, develop anxiety and stress related conditions as means to deal with a teaching methodology which is unsuited to their needs.

The dyslexia indicators noted earlier are common parts to the continuous teacher training given not only at universities but as part of teacher professional development programs. However, only 17% of student teachers feel confident in supporting a dyslexic child in their classrooms suggesting current teacher training is inadequate, however surprisingly only 10% felt more training was needed (Burden and Gwernan-Jones, 2008). The study did however find that such trainee teachers believed that dyslexia was not an excuse for laziness and see that such a label can bring with long-term positives to a child.

So if the dyslexic child is not recognised at school, how do they cope? The evidence noted in this empirical review suggest they cope badly, not only academically but emotionally as well. All children recognise those children around who do them not learn as fast as they do, the dyslexic is no different. They recognise they are different to their peers and not as fast to pick up reading, writing and remembering things. To begin with, they get frustrated and logically conclude the problem is with them and feel alien to their peers and in most cases at home as well with their families. Their parents can't understand what is going on and blame the dyslexic child by telling them they need to work harder or concentrate more. This conflict of working hard but not getting the results they feel they should get is troubling and can cause stress and anxiety. As in the example of a child putting a hand near a hot iron, you only need to get burnt once to know that it should be avoided. So the dyslexic child learns to avoid learning, avoiding putting their hand up in case they say the wrong thing and get laughed at by their peers. In essence they become reluctant learners who will not experiment with new learning and withdraw. It is the teachers job to recognise such reluctance, but sadly in most cases they are quickly labelled 'lazy' or 'careless' by such authority figures rather the source of the problem being investigated. The treachers themselves are fire-fighting in many cases in large and sometime unmanageable classrooms. Commonly, teachers perceive failing children as a threat to their integrity, which means in most cases they perceive the child as the problem, not their teaching methods. Thus dyslexic children can be even more reluctant to display publicly inadequacies, and will learn that avoidance is the key to survive school. Avoidance leads to withdrawal and unfortunately depression in some cases.

Whilst the evidence suggests that many dyslexics could develop depression, it is a hard hypothesis to prove or support with any certainty. The following chapter (Alexander-Passe, in press-4) aims to support such a hypothesis by analysis of semi-structured investigative biographical interviews with N=29 dyslexic adults, ranging in age between twenty-two to sixty-five years old. N=18 are female and N=6 are male. N=14 are degree educated and N=15

are not. N=21 are depressive (as defined by their physician with a diagnosis of clinical depression or have at least two courses of anti-depressants prescribed to them over a course of more than a year). N=15 of the depressives are female and N=6 are male. N=3 of the non-depressives are female whilst N=5 are male. The next chapter aims to investigate if the source of their depression comes from their childhood and also if those interviewed recognise that these same depressive symptoms are rooted in their childhood, thus implying that they were not only unidentified dyslexics but unidentified depressives as well.

CONCLUSIONS

This chapter began by defining a child, depression and lastly dyslexia. It was felt by the author that such a strategy was needed to clearly define the direction of the empirical investigation.

The chapter clearly defines the indicators of dyslexia, not only in pre-school but in primary and secondary school age so that any reader can assist in recognising unidentified dyslexia amongst those they meet. This is necessary as it would seem from the evidence presented that teachers are either unable or unwilling to identify dyslexia in their pupils. Such a situation causes frustrations and stress, with children therefore seeing the classroom as an environment that must be controlled and avoided, that learning is not the investigative process that it should be but an opportunity where embarrassment and failure commonly takes place in front of their peers.

Dyslexics with high intelligence will use their intellect to carefully choose subject options that will limit their time doing feared tasks (e.g. reading out loud in class and essay writing) and choose science or design subjects. They will use their energies to avoid and choose easier to spell words.

Dyslexics with normal or lower intelligence will have a harder time. They will have fewer strategies to avoid feared tasks and will begin to self-doubt and may either lash out or withdraw.

A large number of empirical studies in this area suggests that many dyslexics develop negative learning strategies, as a reaction to the frustrations and stresses they encounter. It is hoped that in most cases dyslexics will just avoid reading and writing to get through school, whilst this is an unhealthy attitude to learning, it is one option which will protect their self-esteem. Unfortunately studies point to secondary manifestations developing which are both emotionally and psychological damaging. In some cases these can turn into physical manifestations with self-harm and suicide as the ultimate conclusion.

As noted earlier by Burden (2005) all children hate to fail, hate to be different and hate to be singled out as having special needs. Their segregation to the remedial or ‘slow’ table or taken out of the classroom for extra help may be a negative strategy as they are marked out as being different in the eyes of their class peers.

Empirical evidence suggests that dyslexics do not feel integral to their family, peers and society as a whole, and they look for ways to protect them from the harm that such a conclusion poses. This idea is most damaging as it suggests that their attitude towards their families, peers and teachers is faulty, along with the perception that they have no place in the society around them. Such a conclusion could if taken to the extremes, mean that young

dyslexics can withdraw from society or they may wish to ease their burden on their parents and society by attempting suicide. Such a conclusion, especially in childhood suggests the support framework which children need to rely on from parents, peers, teachers, school and the medical profession is not working effectively.

The Dyslexic Defence Mechanisms DDMs (Alexander-Passe, in press-2) suggest a means to understanding the processes involved in coping with an unidentified learning difficulty such as dyslexia. In the next chapter (Alexander-Passe, in press-4) an investigative biographical semi-structured interview study will investigate this area further with a range of dyslexic adults.

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Chapter 3

DEPRESSION IN CHILDREN AND ADOLESCENTS

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ABSTRACT

This paper reviews recent literature on various aspects of depressive disorders in children and adolescents, including the epidemiology, clinical presentation, natural history, etiology and treatment. Depression is an important psychiatric disorder in youngsters that increases in frequency with age, often coexists with anxiety disorders and behavior disorders, and is associated with significant psychosocial impairment. Early depressive episodes often recur and persist into adult life along with long-term morbidity and mortality. The etiology of pediatric depression is complex, involving genetic, neurobiological, psychological and environmental processes. There is some evidence for short-term efficacy of both pharmacological and psychosocial interventions for the treatment of depression in children and adolescents. Given the relatively long duration of depressive episodes and high propensity for relapse and impaired psychosocial functioning, further exploration of the etiology, natural history and treatment of pediatric depression is warranted.

INTRODUCTION

The prevalence of depression in children and adolescents is relatively high, and depressive illness during these developmental periods is associated with significant impairment in multiple social domains. Also, there is evidence that early depressive episodes recur and persist into adult life along with ongoing psychosocial difficulties. This paper describes the nosology, correlates, clinical course and consequences of pediatric depression, highlighting the similarities and differences in the characteristics of depression between children and adolescents. In addition, the paper will summarize assessment methods and treatment interventions for these disorders in youngsters.

HISTORICAL CONTEXT OF DEPRESISON IN CHILDREN AND ADOLESCENTS

Until the mid-1970's, it was assumed that sadness in children is a transient emotional response to difficult circumstances that differed fundamentally from the depressive syndrome in adults. It was also believed that many of the problems associated with depression, such as sadness, irritable mood, self-doubt and social withdrawal, were normative expressions of adolescent angst [1, 2]. Research in this area shifted fundamentally with the development of semi-structured interviews and application of operational diagnostic criteria for depression in youngsters [3, 4]. Although nosological questions continue to be raised about depressive disorders in children and adolescents, significant information has accrued about their clinical presentation, course and outcome, as well as neurobiological, psychological, social and familial correlates and selected risk factors. In addition, both pharmacological and psychosocial interventions have been tested to assess their efficacy in the treatment of depression in children and adolescents.

A DEVELOPMENTAL FRAMEWORK FOR UNDERSTANDING DEPRESSION IN CHILDREN AND ADOLESCENTS

In the past three decades, depression research in children and adolescents has progressed from applying simple extensions of clinical descriptions and theories developed in adults to generating an increasingly sophisticated understanding of these disorders informed by the emerging field of developmental psychopathology. This perspective takes into account the intersection between normative developmental processes and the development of psychopathology [5-8]. When applying a developmental perspective on psychopathology, one important issue to consider is the conceptualization of different life stages. For example, the transition from childhood to adolescence involves changes in multiple domains, including physical, sexual, cognitive and social development, with a considerable range of individual differences in the age at which each of these changes occur. At present, there is no consensus on the clear boundaries in defining childhood and adolescent stages of development. Because gathering information on these multiple domains is complex, for pragmatic reasons, majority of the studies used chronological age to define these boundaries; children $\leq$12 years and adolescents between 13-18 years. In some cases, however, studies are reviewed in which these ages overlap (e.g., some studies included 13-year-olds in the child samples, whereas others included 12-year-olds among adolescent samples, and still others reported findings according to grade level or external manifestations of pubertal status).

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PREVALENCE OF DEPRESSION IN CHILDREN AND ADOLESCENTS

Lifetime prevalence of depression increases dramatically from 1-2% in children to 15-25% by the end of adolescence [9]. The greatest surge in newly emergent cases occurs between 15 and 18 years [10, 11]. The prevalence estimates of adolescent depression are comparable to the lifetime rates reported in adults, suggesting that the rates of depression begin to plateau by early adult life [11, 12]. These data also indicate that, for a substantial proportion of adult cases, the onset occurred during adolescence [13].

During childhood, rates of depression are about equal in boys and girls, or even higher among boys [9, 14]. Around puberty, the rate of depressive symptoms and disorders in girls rises by two to three times that of boys, a trend that continues through adult life [11, 12, 15, 16]. The increased risk for depression in adolescent females is attributed to a complex interplay of psychological and social factors against the background of biological changes [8, 17, 18].

NOSOLOGY OF CHILDHOOD AND ADOLESCENT DEPRESSION

Depressive disorders include a heterogeneous group of conditions. Major depressive disorder is defined by a combination of protracted depressed mood, irritability and/or loss of pleasure along with a cluster of other symptoms including loss of energy, changes in eating patterns (e.g., appetite change, weight loss, failure to make expected weight gains), sleep dysregulation (e.g., insomnia, hypersomnia, non-restorative sleep), motor behavior (e.g., psychomotor retardation, agitation, restlessness), thought processes (e.g., distractibility, indecisiveness, hopelessness, suicidal ideation), and self-esteem (e.g., feelings of worthlessness, guilt). The Diagnostic and Statistical Manual of Mental Disorders (4^{th} Edition with text revisions; DSM-IV-TR) criteria require that, in order to be diagnosed with major depressive disorder, a youngster must have at least five such co-occurring symptoms for at least two weeks along with personal distress or functional impairment [19]. Dysthymic disorder is a symptomatically less severe but temporally more protracted form of depression. The DSM-IV criteria require that depressed mood and two or more associated symptoms last for at least one year in youth, along with functional impairment or clinically significant distress [19]. The validity of distinguishing between major depression and dysthymic disorder as two separate entities in youngsters has been called into question by the National Institute of Mental Health Methods for the Epidemiology of Child and Adolescent Mental Disorders (MECA) study [20]. The findings from this study indicated that children and adolescents with these two conditions could not be distinguished on demographic or clinical features, or on specific correlates.

Depression in youngsters also can present as a sub-threshold, subsyndromal or subclinical condition. In other words, the youth might experience dysphoric mood and associated symptoms (e.g., pessimism, poor self-esteem) that are more frequent and severe than normal but not sufficient to meet the criteria for a depressive disorder. Subsyndromal depression is associated with high levels of distress and impaired functioning [21, 22], and prospective studies indicated that it is a strong predictor of major depressive disorder [23, 24]. In one

study, anhedonia and thoughts of death were particularly predictive of subsequent depressive episodes [24].

The diagnosis of dysthymic disorder and major depressive disorder are based on similar criteria for children, adolescents and adults, with two exceptions. The DSM-IV has allowed the substitution of irritability for depressed mood in children and adolescents, and the duration for dysthymic disorder is one year instead of two [19]. Research also has indicated that the clinical syndrome of depression is remarkably similar among children, adolescents and adults [25-27]. Specifically, hypersomnia shows a developmental trend with a higher prevalence in depressed adolescents than in children [28-31]. Suicide attempts, particularly those involving high lethality, also increase with age [29, 31]. Melancholic and psychotic symptoms may occur less frequently in children, whereas somatic complaints and behavior problems are more common during this developmental period [25, 30, 32]. Psychotic depression in children appears to be manifested by auditory hallucinations instead of delusions, as seen in adolescents [25, 29]. Although the reasons for the developmental variations in depressive symptoms are not known, maturational effects on emotional and behavioral regulation and cognitive function might contribute to these differences [8, 25].

Gender differences also have been documented with respect to the severity and symptom profiles of unipolar depression among children, adolescents and adults although no compelling gender effects were found on the salient features [30, 33-35]. Among adolescents and adult, females typically report higher levels of symptoms [33, 36]. With regard to symptom patterns, somatic symptoms, such as changes in appetite and weight, sleep problems and psychomotor retardation are more common in females [37-40]. Increased crying, feelings of failure, guilt, poor self-esteem and other cognitive symptoms also may be more frequent in females [37, 38, 41-43]. In contrast, depressed males more frequently report anhedonia, diurnal variation in mood and energy, social withdrawal and work impairment [37, 38, 42]. The reasons for gender differences in depression are not well-understood. One model suggests that females are more prone to exhibit a cognitive style characterized by negative self-evaluation and rumination [44, 45]. Gender differences in brain structure and function also has been postulated as one potential reason for the symptom variability [46].

COMORBIDITY

A striking and widely replicated characteristic of both clinic-referred and community samples of youngsters with major depression is that up to 40-90% also suffer from another psychiatric disorder, and many youngsters have two or more comorbid diagnoses [47-49]. Approximately 70% of children and adolescents with dysthymic disorder eventually will develop an episode of major depressive disorder, resulting in "double depression" [50, 51]. Anxiety disorder is the most common co-occurring disorder with depression, with estimates ranging from 30-75% in children and between 25-50% in adolescents [47, 52]. There also is increasing evidence that anxiety symptoms precede the emergence of depression and, therefore, might be a risk factor for depression [52-55]. It is suggested that anxiety and depressive disorders share a common genetic basis with a different developmental time course [53, 56]. In one investigation, anxiety disorder preceded the onset of insomnia in youngsters whereas episodes of depression followed bouts of insomnia, suggesting that sleep

disturbance might serve as a mediating link between these two disorders in some instances [57]. Disruptive and substance use disorders also frequently co-occur with depression [49, 58].

Age and gender can influence the patterns of comorbidity [49, 59]. Specifically, separation anxiety disorder and attention-deficit hyperactivity disorder are more common in children whereas conduct disorder, panic disorder and substance abuse are more common in adolescents [47, 49, 59, 60]. Similarly, disruptive and substance use disorders are less likely, and eating disorders are more likely, in girls than boys [10, 49, 59, 61]. Data in adults suggest that, compared with depressed patients whose first depressive episode occurs in adult life, patients with early-onset illness have increased rates of anxiety disorders, substance use disorders as well as personality disorders [62-66].

The presence of comorbidity with depressive disorders has important clinical and functional implications [67, 68]. In particular, youth with co-occurring dysthymia and major depression have more severe and longer depressive episodes, higher frequency of suicidal behavior and social impairment than those who have only dysthymic disorder or major depression [20, 23, 51, 69]. Similarly, comorbid anxiety disorder was associated with increased severity and duration of depressive symptoms, increased suicidality, poor response to psychotherapy, or elevated risk for addictive disorders [52, 70-74]. In contrast, depressed youth with co-occurring disruptive disorders tended to have fewer melancholic symptoms, fewer recurrent episodes of depression, a lower frequency of familial mood disorders, a higher incidence of criminal behavior and a higher response to placebo than patients with pure depressive illness [75-78]. Comorbid substance abuse is associated with earlier onset and more severe substance-related problems, increased frequency of behavioral problems, more prolonged and recurrent depressive episodes, more severe impairment in family, school and legal domains, higher risk for suicidal behavior, and increased utilization of health services and substantially higher treatment costs [58]. Data in adults suggest that early-onset cases have a higher prevalence of comorbidity, more chronic course and disability than adult-onset patients [66].

NATURAL COURSE OF DEPRESSION IN CHILDREN AND ADOLESCENTS

Episode Duration

Considerable variations have been found in the duration of depressive episodes in non-referred and clinical samples of youth. For example, in a large sample of high-school students, the duration of a major depressive episode ranged from 2 weeks to 250 weeks, with a mean duration of 26 weeks [79]. The probability of remission was 3 weeks in 25% of the sample, 8 weeks in 50%, and 24 weeks in 75% of the sample. The mean length of a major depressive episode in clinic-referred youth is approximately 7 to 9 months [80-83]. Up to 30-40% of patients can be expected to recover by 6 months, and 70-80% by 12 months, from the onset [23, 79, 84-86]. Approximately 5-10% of patients have a protracted episode, lasting longer than 2 years [82, 84]. In contrast to this, recovery from a dysthymic episode is slow. In a prospective study of a clinical sample, only 7% of youth with dysthymia showed evidence

of recovery two years after the onset of a first episode [82]. The average duration of a dysthymic episode is 2.5 to 3.5 years [51, 82, 87].

For the most part, children, adolescents and adults have similar recovery patterns [32, 82, 88, 89]. Among the baseline demographic and clinical variables that were examined, none has been shown yet to consistently predict recovery from a depressive episode in youngsters. Age at onset of illness, greater severity, presence of comorbid disorders and parental history of depression potentially influence the time to recovery [32, 88-90].

Recurrence

Longitudinal studies of both epidemiological and clinical samples consistently demonstrated that children and adolescents with depression tend to have recurrent episodes. The probability of recurrence following the recovery of a major depressive episode is approximately 40% by two years and 70% by 5 years [80, 81, 83, 91]. These rates are comparable to the rates of recurrence in adult samples [92-94].

In addition to recurrent episodes during childhood and adolescence, longitudinal studies of depressed youngsters documented recurrent episodes in adult life [81, 83, 95]. There also appears to be some specificity in the continuation of psychopathology in adult life, particularly with respect to adolescent-onset depression. Several studies of depressed adolescents documented increased risk for recurrent depressive episodes, but not other psychiatric disorders, when compared with their counterparts without depression [95-98]. In contrast, there is some evidence that childhood-onset depression is not necessarily predictive of depression in adulthood except for a subsample with symptoms characteristic of the adult disorder; nevertheless, these youngsters have other disorders in adult life, also including minor depression [96, 98, 99].

Few baseline demographic or clinical characteristics predict who will or will not experience a recurrent depressive episode. There is disagreement regarding whether girls are at increased risk for recurrent depressive episodes than boys [34, 80, 81, 84, 100]. In several studies, family history of depression, particularly with childhood-onset illness, was associated with increased risk for recurrence [101-103]. Co-occurring personality problems, specifically borderline personality disorder symptoms, also were associated with recurrence in some studies [93, 100]. Among adults, early-onset illness, number of prior episodes, stressful experiences, cognitive vulnerability, negative family interaction patterns, and comorbid personality disorders were associated with recurrence [66, 104].

Other Psychiatric Outcomes

Although recurrent unipolar depression is the primary outcome for depressed youth, development of other psychiatric disorders is also common. Longitudinal studies reported that 20-40% of youngsters with a major depressive disorder developed bipolar disorder within a period of 5 years [86, 97, 105, 106]. The clinical characteristics associated with increased risk for bipolar disorder include early-onset illness, mood lability, depressive episode accompanied by psychomotor retardation or psychotic features, atypical depression, protracted depressive episodes, family history of bipolar disorder or heavy familial loading

for mood disorders, and pharmacologically-induced hypomania [86, 107-109]. Depressed youngsters also are at higher risk for developing substance use disorders [49, 73, 74, 110-112]. Protracted depressive episodes, comorbid anxiety or conduct disorder and hypothalamic-pituitary-adrenal (HPA) dysregulation may be associated with increased risk for substance use disorders in depressed youth [73, 74, 76].

CONSEQUENCES OF DEPRESSION IN CHILDREN AND ADOLESCENTS

The functional consequences that characterize depressed youngsters suggest that the disorder can interfere with developmental milestones. For example, depression is frequently associated with problems in interpersonal relationships and school performance, as well as delays in social, emotional and cognitive development [113-119]. It is not clear, however, whether these psychosocial disturbances are precursors or consequences of depression. Moreover, other factors frequently associated with depression, such as comorbid psychiatric disorders, low socioeconomic status, poor family functioning and exposure to stressful situations, have an impact on psychosocial functioning [58, 120, 121]. Pediatric depression is also associated with an increased frequency of suicidal behaviors, delinquency, and alcohol and drug use [117, 120]. Prospective studies found that, after recovery, children and adolescents continue to manifest impaired psychosocial functioning in multiple domains [119, 122-124]. Moreover, children and adolescents with depression have persistent psychosocial problems in adult life, including criminal behavior, dysfunctional interpersonal relationships, early pregnancy, low educational attainment, poor occupational functioning, unemployment, and suicidal behavior [13, 96-98, 111, 125-132]. Some studies also reported high rates of psychiatric hospitalization and mental health services compared to their counterparts without depression [96, 98, 132, 133]. Data in adults suggest that depressed patients with early-onset illness have more impaired social and occupational functioning and poorer quality of life compared with patients whose episode(s) first started in adult life [66].

ETIOLOGY OF CHILDHOOD AND ADOLESCENT DEPRESSION

The development of depression in children and adolescents involves a complex, multi-factorial model [7, 134, 135]. No single risk factor accounts for all or even most of the variance. Rather, it is more likely that the accumulation, and/or interaction among multiple risk factors will lead to depression [136-138].

Family-Genetic Factors

There is clear evidence of familial transmission of depression [139-141]. These data, however, cannot distinguish environmental from genetic causes of transmission [139, 142, 143]. Family, twin and adoption studies documented effects of both genetic and environmental factors for unipolar depression [144, 145]. In a large study of adolescent female twins, genetic factors accounted for 40.4% of the variance in risk for major

depression. Non-shared environmental effects accounted for the remaining portion of the variance. Similarly, large-scale studies indicate that exposure to early adverse situations (e.g., parental loss, chaotic family environment, childhood abuse) account for over 50% of the attributable risk for depression [146, 147]. Most importantly, genes and early experiences interact. For example, Caspi et al. [136] found that exposure to severe childhood maltreatment doubled the risk of major depression in individuals with two copies of the short allele in the promoter region of the gene encoding serotonin transporter (5-HTT). In contrast, childhood maltreatment was not associated with increased risk for depression in individuals with two copies of the long allele of the 5-HTT gene.

Genetic and environmental influences have been found to vary with age and sex. Shared environmental influences may be more important in younger children, and these influences may be replaced by new genetic and unique environmental influences as children grow older [140, 148]. In one study, the increased heritability effect in adolescents was found only for girls, and not boys [149]. In summary, many cases of depression might arise from the confluence of genetic predisposition interacting with experiential factors that occur during a specific window of vulnerability. This, in turn, leads to a cascade of events that unfold over the course of maturation.

Neurobiology

Neurobiological studies of childhood and adolescent depression have employed methods used in adult studies and attempted to replicate those observations. Most of the studies focused on sleep architecture and neuroendocrine systems [150, 151]. There also is emerging literature on structural and functional neuroimaging techniques [152, 153]. It is important to note that the sample sizes in many of these neurobiological studies are modest.

Sleep Architecture and Electrophysiological Studies

In adults, the most reliable sleep architecture changes associated with major depression include sleep continuity disturbances, shorter latency to rapid eye movement (REM) sleep, increased phasic REM sleep and diminished slow-wave sleep [154, 155]. Sleep architecture measures have shown considerable variability with regard to group differences between depressed youngsters and matched controls [150, 156]. The findings vary as a function of age, gender, familial risk, severity of illness and clinical course [150, 157, 158]. Depressed adolescents seem to have relatively more frequent disturbances in circadian rest-activity rhythms, sleep architecture and electroencephalographic (EEG) rhythms during sleep compared with depressed children [150, 159, 160]. Changes in sleep architecture and sleep-related EEG rhythms also were documented in healthy adolescents at high-risk for depression based on family history, and these changes were associated with vulnerability for depression during prospective follow-up [157, 161, 162]. Additionally, baseline sleep architecture patterns differed between depressed adolescents who subsequently had a recurrent unipolar course versus those who developed bipolar illness; adolescents with unipolar course had predominantly REM sleep changes while adolescents with bipolar course manifested non-REM sleep changes [163]. In the same study, adolescents who subsequently developed substance use disorders had relatively normal EEG sleep patterns [73]. The observed

variability in sleep architecture changes in depressed youngsters may reflect, at least in part, heterogeneity in the longitudinal clinical course of these disorders.

Electrophysiological studies also documented reduced left frontal electrical activity in infant and adolescent offspring of depressed mothers [164-166], and in adolescents with major depressive disorder [167, 168]. Deceased left frontal EEG activity probably reflects an under-activation of the approach system and reduced positive emotional expression, which also might be a vulnerability marker for depression [169].

Neuroendocrine Studies

Among the neuroendocrine markers of pediatric depression, there has been considerable interest in the HPA system, consistent with the hypothesis that depression is linked to altered responses to stress. Numerous studies documented HPA dysregulation in adult depression including higher basal corticotropin-releasing hormone (CRH) and cortisol secretion, non-suppression of cortisol in response to dexamethasone administration, and blunted corticotropin response to CRH adminsitration [170, 171]. Findings from HPA studies in depressed children and adolescents have been inconsistent [150, 151]. Specifically, depressed children did not display changes in 24-hour cortisol patterns compared to matched controls without a psychiatric disorder. Few differences in basal cortisol secretion have been observed between depressed adolescents and controls, and when group differences were detected, they tend to be subtle alterations in normal diurnal patterns. These subtle changes, however, were relatively robust in predicting the longitudinal clinical course; higher cortisol secretion in the evening or during sleep, a time when the HPA axis is relatively quiescent, was associated with a longer time to recovery from the depressive episode [172], a propensity for recurrence [162, 173], and suicide attempts [174]. Higher cortisol secretion also was detected in at-risk youth who subsequently developed depression [157, 175, 176]. In cross-sectional studies, HPA activity varied as a function of exposure to stressful experiences in depressed children and adolescents, with increased activity specifically in youth with adverse experiences [177, 178].

Another neuroendocrine marker possibly related to depression is growth hormone, which is secreted by the anterior pituitary and follows a circadian pattern with increased secretion during slow-wave sleep. Although the precise role of growth hormone secretion in depression is not known, it appears to be a marker of central noradrenergic and 5-HT systems [179]. Reduced growth hormone secretion during sleep has been observed in adult depression [180], but findings in children and adolescents have been variable [150, 151]. One study found that depressed children with stressful life events had increased growth hormone secretion compared to their counterparts who did not experience recent stress, suggesting that environmental factors have a moderating influence [181]. In another investigation, depressed adolescents who subsequently exhibited suicidal behavior had increased growth hormone secretion during sleep, and when this group was separated, depressed adolescents manifested blunted growth hormone secretion compared with controls [182]. In contrast to the findings in basal secretion, pharmacological challenge studies documented blunted growth hormone response to a variety of pharmacological agents in depressed children, similar to those reported in depressed adults [179]. In contrast, data in adolescents were predominantly negative. Although the sample sizes were modest in these adolescent studies, pubertal changes and gender might account for some variability among child, adolescent and adult samples [150, 151].

Neuroimaging Studies

Studies using various neuroimaging techniques provided converging lines of evidence supporting prefrontal cortical-striatal and medial temporolimbic dysfunction in adult depression [183, 184]. There is preliminary evidence of anatomic and functional differences in the brains of depressed and at-risk youth compared to matched controls without disorder [152, 153]. Reduced left frontal volume was documented, particularly in youngsters with familial depression [185-187]. Alterations in amygdala and hippocamplal volumes also were found [188-190], although the effects may be moderated by anxiety symptoms [191]. Moreover, amygdala responses to emotional tasks have been documented in normal and depressed youngsters [192, 193]. In neurochemical studies, reduced glutamate and creatinine/phosphocreatinine concentrations in the anterior cingulate, and increased choline concentrations in the left dorsolateral prefrontal cortex, were documented in children and adolescents with depression [153].

Summary of Neurobiological Research

Neurobiological research in pediatric depression suggests that neurobiological factors change during the course of development, and developmentally-influenced neurobiological processes may become disrupted during depressive episodes. Longitudinal studies that account for familial and clinical variability allude to this possibility. These data also indicate that pediatric depressive disorders may not necessarily result from the same etiological processes, and the specific subtype with familial loading or depressive disorder with a recurrent unipolar course may be associated with neurobiological changes typically observed in adult unipolar depression. Experiential factors also appear to influence neurobiological findings [150, 178, 181, 194]. Future longitudinal investigations with large sample sizes should examine genetic, developmental and socio-cultural influences on neurobiological factors associated with the onset and clinical course of depression in children and adolescents.

Temperament and Personality

Temperament is broadly defined as individual differences in emotional and behavioral style that appear early in life, are consistent over time and across situations, and presumed to have a genetic/biological basis [195, 196]. It is however, important to note that experience and learning, particularly within the social context, also can influence the development and expression of temperament [195, 196]. The trait that is associated with most emotional disorders has been given various labels by different theorists, including behavioral inhibition [197], harm avoidance [198], negative affectivity [199], neurotism [200], and trait anxiety [201], although there is a significant overlap among these constructs both from conceptual and empirical perspectives.

Negative affectivity is the propensity to experience negative emotions, and it reflects sensitivity to negative stimuli, increased wariness, vigilance, physiological arousal and emotional distress. In contrast, positive affectivity is characterized by sensitivity to reward cues, sociability and adventurousness [199]. Depression is characterized by high levels of negative affectivity and low levels of positive affectivity [202], and these features also have been found in depressed children [203]. A higher frequency of behavioral inhibition also has

been observed in laboratory tasks with young offspring of depressed parents [204]. Moreover, some longitudinal studies have shown that children with inhibited, socially-reticent and easily-upset temperament in early childhood had higher rates of depressive disorders during adolescence and/or early adulthood than those who did not demonstrate these characteristics [205, 206]. Also, difficult temperament, characterized by inflexibility, low positive mood, withdrawal, and poor concentration correlated with depressive symptoms both concurrently and prospectively in adolescents and adults [207, 208]. There is also evidence that the effects of temperament on depression may be mediated by other factors, including cognitive and contextual variables [209]. Some of these factors will be discussed in subsequent sections.

The relation between temperament and depression may vary with age. In one study, neurotic-like symptoms predicted the first episode of depression in 31-41-year-old individuals but this was not the case for 17-30-year-olds [210]. Similarly, adult participants who experienced a first episode of depression had exhibited elevated levels of dependent traits 2-3 years earlier [211]. However, no differences were found with regard to such dependent traits between adolescents who later developed depression and those who did not develop the disorder [124]. Gender also might influence the relationship between temperament and depression. In a prospective study, females with higher levels of chronic depression during young adulthood were described as shy and withdrawn at 3-4 years of age, whereas males with chronic depression exhibited higher levels of under-controlled behavior as young children [212].

Cognitive Vulnerability

Cognitive theories posit that negative belief systems and maladaptive thought processes confer vulnerability to depression [213, 214]. Generally, the cognitive theories are framed in terms of a diathesis-stress perspective, wherein maladaptive cognitive appraisals about the self and the world heighten susceptibility to depression when individuals are confronted with stressful life experiences [138, 215]. Several variants of cognitive models have been proposed [6, 216, 217].

Cognitive Schemas and Information-Processing

Beck [214] defined cognitive vulnerability as the presence of maladaptive self-schema reflecting themes of negative views of the self, world and the future. These negative cognitive schemas guide information-processing in the form of attention to, interpretation of, and memory for, specific life situations [218]. Consistent with this theory, depression in children and adolescents has been linked to self-critical beliefs and negative thoughts about the world, including diminished self-worth, dysfunctional attitudes and irrational beliefs about interpersonal or other experiences, and negative automatic thoughts [6, 215, 217, 219]. However, direct investigations of information-processing using laboratory paradigms have yielded mixed results [217, 220]. The variability in findings might be due to questionable reliability and validity of the current information-processing paradigms [217, 221]. Also, most of the studies on information-processing in children and adolescents are cross-sectional, and they do not explicate whether the cognitive biases contribute to the etiology or maintenance of depression.

Attributional Style and Control-Related Beliefs

Among the various cognitive models of depression, attributional style has garnered the most theoretical and empirical attention. Abramson et al. [213] proposed that depression results from an inferential style that involves a tendency to make internal, global, and stable attributions for negative experiences, and to interpret negative events in ways that compromise one's self-worth. In a similar vein, self-regulation perspectives suggest that depression results from negative expectations about outcomes, and one's ability to control such outcomes [222-225]. These types of cognitive patterns evolve into hopelessness, which is hypothesized to be a proximal cause of depression [226, 227]. In an elaborated transactional cognitive vulnerability-stress model of depression cycle [44], negative events contribute to initial elevations of general negative affect. Cognitive vulnerabilities, such as negative attributional style, then moderate the risk for depression. Depression, in turn, can lead to more negative life events, creating a "vicious cycle". There is empirical support for these theories of depression in children and adolescents [6, 215, 217, 226].

Developmental and Gender Influences on Cognitive Vulnerability

Developmental theorists have suggested that negative cognitions emerge over time and that their relation with depression becomes stronger with development [122, 225, 228]. Indeed, the association between negative cognitions and depression is less robust in younger children than in older children and adolescents [122, 217, 229]. There are also gender differences in cognitive patterns. For example, girls may be more susceptible to negative self-perceptions about global self-worth, physical appearance and achievement, thereby taking responsibility interpersonal problems or failure which then increases the risk for depression [230-232]. The tendency of girls to dwell or ruminate on problems also can increase and prolong negative mood [8, 17, 231].

A Diathesis-Stress Model of Cognitive Vulnerability

Diathesis-stress models propose that depression results from the interaction between personal vulnerability and stressful events or circumstances. Several studies documented interactions between cognitive styles, such as negative attributional style or low perceived self-efficacy, and life stress in the prediction of depression in youngsters [44, 233-235]. Even further refining these theories, it has been speculated that a key determinant of depression may be the match between a particular cognitive vulnerability (e.g., a tendency to base one's self-worth on success in interpersonal relationships) and the nature of the stress (e.g., interpersonal conflict). Supporting this theory, diathesis-stress interactions seem to be most powerful when there is a match between the type of cognitive vulnerability and the type of stressful experience [229]. Consistent with the theory that cognitive styles may not yet be consolidated in younger children, cognitive-stress interactions predicted depression in adolescents but not in children [226, 229].

Environmental Factors

Interpersonal Relationships

Interpersonal theories of depression emphasize the importance of the social environment on emotional and behavioral regulation and social adjustment. Vulnerability to depression presumably arises in the context of early family environments in which the children's needs for security, comfort and acceptance are not met [236]. Research on the relation between family environment and depression indicates that families of depressed youngsters are characterized by problems with attachment, communication, conflict, cohesion and support, as well as poor child-rearing practices [139, 237]. Additionally, perceived rejection by peers, family and teachers predicts increases in depressive symptoms in children and adolescents [238-241]. Interpersonal theories of depression propose that depressed individuals both react and contribute to interpersonal problems [242, 243]. Depressive symptoms and associated behaviors are presumed to elicit negative reactions from others; these aversive interpersonal experiences then foster the persistence or exacerbation of depression [244]. Consistent with interpersonal models, depressed youngsters demonstrate difficulties in many aspects of relationships with peers and family members [79, 118, 119, 121, 245, 246]. Longitudinal studies on the association between interpersonal relationships and depression indicate that social problems temporally precede depression, and that depression also contributes to interpersonal difficulties [243].

Theory and research on interpersonal relationships have yielded a wealth of information, indicating significant variance in the nature, role and consequences of interpersonal relationships across development and gender. For example, adolescence is a transitional period during which the salience of peer relationships for socialization and emotional experience increases as the salience of family diminishes [247, 248]. Furthermore, adolescent friendships are characterized by higher levels of intimacy and loyalty than preadolescent friendships [247, 248]. In particular, adolescent girls are more invested in relationships with friends and romantic partners [249-251]. Although these characteristics of adolescent females may provide a strong sense of social connectedness and support [230], the heightened interpersonal focus and reliance on others may be accompanied by increased vulnerability to depression during interpersonal conflicts or losses [249-251].

Life Stress

Stress plays a prominent role in most theories of depression, and there is a clear link between stress and depression in children and adolescents [135, 252, 253]. The relationship between stress and depression appears to be stronger in adolescents than in children, particularly in girls [250, 254, 255]. The reasons for this are not entirely clear; hormonal effects, consolidation of cognitive styles, cumulative stress burden, and stress reactivity might have a potential role [8, 250]. One theory proposes that childhood adversity alters neurobiological and psychosocial processes whereby individuals may be sensitized to the effects of recent stressful events, leading to depression at lower levels of stress [256], or with greater physiological reactivity to the effects of stress [178, 257]. Another model suggests that childhood stressors contribute to lifetime stress burden and independently predict depression along with recent stress [258, 259].

Developmental models of psychopathology also suggest a transactional perspective in which stress-exposure contributes to depressive symptoms and, in turn, depressed individuals contribute to negative events through their own behavior [260]. Longitudinal studies have shown support for the stress-generation model, particularly with regard to interpersonal relationships [260-262]. Factors that might contribute to the generation of stress include temperament and personality characteristics [263, 264], lack of interpersonal competence [261, 265], and comorbid psychopathology [262, 266]. The reciprocal model highlights the "vicious cycle" that can occur between stress and depression, and support for this reciprocal model has been found in a few studies of youngsters [260, 266-268].

Coping with Stress

Although stress clearly plays a role in depression, individuals vary in their response to stress, and how they respond to stress can affect emotional well-being and their future adjustment [135, 209]. In addition to the cognitive adaptive styles described above, other types of coping mechanisms, such as behavioral styles and problem-solving skills, have been examined in relation to depression in children and adolescents [269, 270]. Earlier theories differentiated between problem-focused and emotion-focused coping. Problem-focused coping involves responses that act directly on the source of stress, whereas emotion-focused coping involves palliative measures to counter the negative emotions that arise from stressful situations [270]. Recently, Compas et al. [269] proposed two sets of processes involved in stress-response, one of which is automatic and a second set that is controlled [269]. Automatic and controlled responses to stress can be further distinguished as involving engagement with, or disengagement from, the sources of stress or one's emotional responses to the stress [209, 271].

Automatic responses to stress are hypothesized to be driven by temperamental differences in arousal and reactivity to threat, or through learning and conditioning [269]. Automatic coping responses include physiological or emotional arousal, intrusive thoughts, automatic biases, impulsive responses, and involuntary escape behavior. In contrast, controlled responses to stress involve volitional efforts to regulate emotion, cognition, behavior, physiology and the environment [269]. Although automatic responses to stress are considered to be a function of temperamental characteristics, temperament also influences voluntary/controlled coping responses, and coping may interact with temperamentally-based automatic stress responses [209]. Engagement coping responses are further distinguished as primary-control (or active) and secondary-control (or accommodative) coping responses. Primary-control responses include problem-solving, emotional expression and emotional modulation, whereas secondary-control responses involve acceptance, cognitive-restructuring, positive reappraisal and distraction [269, 272]. On the other hand, disengagement coping includes avoidance, denial, and self-blame [209, 271].

Studies in children and adolescents indicated that higher levels of engaged and problem-focused coping are associated with lower levels of depressive symptoms, whereas disengagement and emotion-focused coping are related to higher levels of depressive symptoms under stressful circumstances [135, 209, 272-275]. Recent investigations also have begun to examine the role of coping methods in the relationship between temperament and depressive symptoms in youngsters [209]. For example, Beevers and Meyer [276] reported that positive experiences and expectancies mediated the relationship between behavioral activation system and depressive symptoms in a sample of college students. In a separate

investigation, negative emotionality was related to greater appraisals of threat, higher utilization of avoidance coping and higher levels of both depressive and conduct problems [277]. However, most of the studies that simultaneously assessed temperament, coping methods and depressive symptoms are limited by methodological problems and they also lack a framework for understanding the processes through which temperament and depression are linked. Most of the research on coping has been cross-sectional though, thereby limiting our ability to draw conclusions about the direction of associations among temperament, coping and depression. Future investigations should refine the methods for measuring temperament beyond self-report inventories in addition to incorporating systematic measures of stress and neurobiological processes in both cross-sectional and longitudinal designs [209].

AN INTEGRATIVE MODEL OF CHILDHOOD AND ADOLESCENT DEPRESSION

An integrative model of depression in children and adolescents is presented, taking into account the complex interplay among genetic, neurobiological, cognitive and environmental factors in concert with developmental challenges and gender influences in the onset and maintenance of depression (see Figure 1; also see [7]). According to the model, genetic factors and early-life experiences influence temperamental characteristics and the neurobiological substrate (physiological stress reactivity, in particular). Early adverse experiences (e.g., poor-parenting practices and family disruption) also interfere with the development of secure and nurturing parent-child relationships. These adverse experiences alone or in interaction with temperament are internalized in the form of maladaptive conceptions about the self and interpersonal relationships. Family dysfunction and/or temperament also are likely to have an impact on coping styles. Maladaptive conceptions of the self and interpersonal relationships, ineffective coping methods and neurobiological vulnerability, in turn, may create difficulties in interpersonal relationships, resulting in depression, or may augment vulnerability to depression under stressful situations. Sensitivity to stress may be highest during developmental transitions marked by maturational changes (e.g., the onset of puberty) or social-contextual changes (e.g., school transitions), thereby intensifying the association between prior vulnerability and depression. Girls may be particularly at risk during this period due to gender-related distinctive characteristics that make them more vulnerable to the effects of transitions (i.e., a tendency toward negative self-evaluation, high investment in maintaining close relationships, and less adaptive responses to stress) as well as increased exposure to stress (e.g., increased parent-child conflict due to social expectations for gender-related roles).

Depression is likely to further compromise development by interfering with the achievement of key developmental tasks (e.g., academic achievement, negotiating changes in family relationships, and establishing peer networks), resulting in the generation of additional stress, and perhaps even contributing to compromised neurobiological development and sensitization to future stress, depression and other psychopathology. These dynamic processes may account, in part, for why early-onset depression tends to be recurrent throughout the life-span and is also accompanied by other psychiatric problems and significant disability.

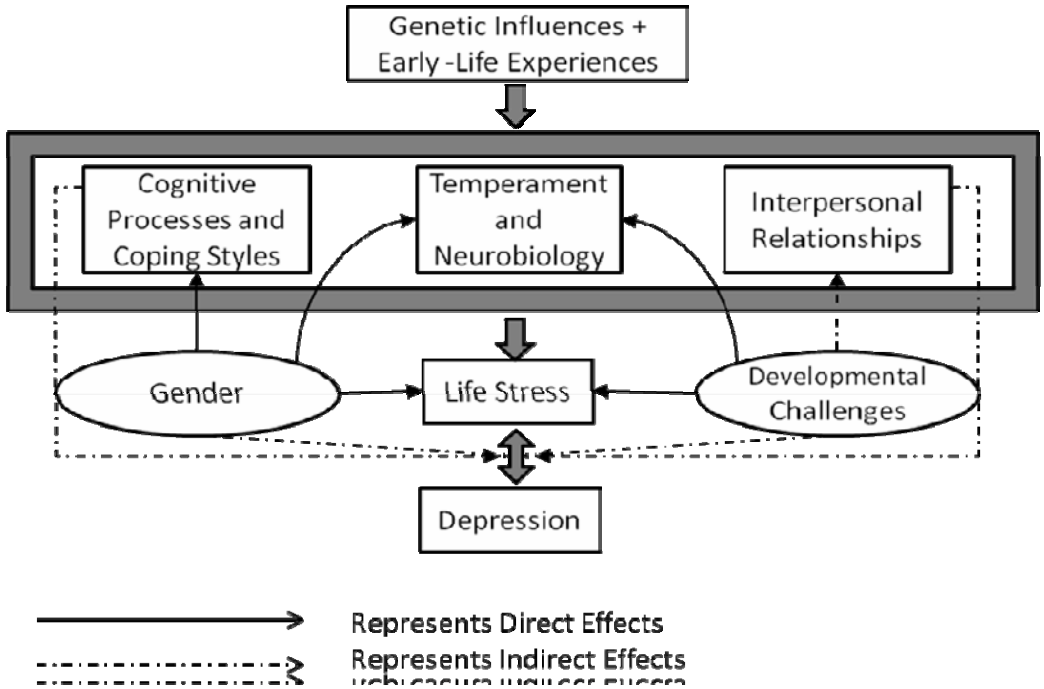

Figure 1.

ASSESSMENT OF DEPRESSION IN CHILDREN AND ADOLESCENTS

The assessment of depression in children and adolescents should include a careful evaluation of depressive symptoms, as well as assessment of other co-occurring psychiatric disorders and associated academic and psychosocial problems. It is also important to identify specific strengths of the child in addition to a medical evaluation. In order to reduce the variability in diagnosis across clinicians, a number of standardized tools have been developed to assess depression and comorbid disorders in youngsters (for reviews, see [278, 279]). Overall, these instruments have demonstrated good inter-rater reliability for depressive symptoms, especially for children over 6 years. Test-retest reliability, however, has been less favorable since depressive symptoms tend to be unstable in younger age groups. In addition to standard diagnostic assessments, several ratings scales are available for assessing depressive symptoms in youngsters [278]. Because of their low specificity, these scales are not helpful for diagnosing clinical depression but they are useful to screen for depressive symptoms, to assess severity and to monitor clinical response to treatment. It is also important to note that many of these instruments were developed for the adult population, and the language of adult measures may be too sophisticated for youngsters [278]. This is particularly relevant for self-report measures that are completed independently. The contexts in which adults and youngsters function also are different. For example, items that reflect impairment in work and sexual domains will be less relevant for children than those that address school performance. Moreover, a more thorough assessment may be warranted to assess symptoms

found to typify depression in youngsters since maturational differences have been documented in the characteristics of depressive symptoms [25, 27, 30, 31].

A key issue in assessing psychopathology in youngsters is the selection of informants and the problems of integrating reports from different sources of information [280, 281]. For example, cognitive and linguistic abilities in reporting internal feelings and perceptions vary markedly across development. Even though older children and adolescents are capable of reporting on their emotional states, they have limited cognitive ability to reflect on their functional states in the context of prior history and in comparison with their peers. Therefore, other sources of information, particularly from parents and teachers, are essential in the determination of psychopathology. The agreement among informants in reporting depressive symptoms, however, is generally low [281]. One potential source of these discrepancies is the characteristics of the rater. For example, mothers who are depressed appear to overestimate the level of their children's depressive symptoms [282]. Discrepancies also may result from true differences in behavior observed in different settings [280]. At present, there are no clear guidelines on how best to integrate data from different informants. It is generally accepted that children offer a better account of internal symptoms, such as depressed mood and suicidal ideation, whereas parents and teachers are more aware of overt behaviors. Further investigation, especially focusing on the predictive validity of data from different informants, is necessary in clarifying these issues.

TREATMENT AND PREVENTION OF DEPRESSION IN CHILDREN AND ADOLESCENTS

Pharmacological Interventions

In adults, a number of antidepressant medications have shown efficacy not only in alleviating depressive symptoms and improving function but also in reducing relapse rates [283, 284]. In contrast, randomized clinical trials of these agents in children and adolescents showed only limited efficacy [285, 286]. Tricyclic antidepressants did not show efficacy in the treatment of pediatric depression [286, 287], and moreover, these agents were considerably more lethal when overdosed in youngsters than in adults [288]. Selective serotonin re-uptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs) showed limited efficacy in pediatric depression [285, 286]. A number of methodological issues need to be considered in interpreting these findings, including modest sample sizes, limited number of trials, heterogeneity of samples and methods across sites, and higher placebo response in younger patients [285, 286]. Fluoxetine is the best-studied antidepressant in pediatric depression, and in multiple trials, fluoxetine showed efficacy in relatively large samples of children and adolescents [289-291]. These three studies were conducted at a single or limited number of sites by investigators with significant clinical and research experience in pharmacologic treatment of pediatric depression, thereby reducing the variance in depression ratings across raters and sites. Furthermore, these investigations either had an extended evaluation or placebo washout phase, potentially reducing the placebo response rate. In addition to short-term efficacy, in a well-designed randomized controlled

trial, continuation treatment with fluoxetine was found to reduce the risk of relapse of depression [292].

The most troublesome issue related to pediatric use of antidepressants has been warnings about increases in suicidal ideation and behavior in children, adolescents and young adults, although no suicides were reported [293, 294]. No compelling data are available to indicate whether the net result of treatment of depression with these compounds in youth is an overall increase or decrease in the hazard of completed suicide [285]. Until further evidence is available, clinicians should carefully weigh the risks and benefits of antidepressant treatment in each individual patient, and to monitor patients closely after treatment is instituted, particularly during periods of initial treatment and dose titration. In addition to expanding the clinical trials of newer antidepressant drugs in children and adolescents, future investigations should attempt identify predictors of clinical response and adverse events so that more specific and safe treatment strategies can be implemented for specific subgroups of patients [295].

Psychosocial Interventions

Psychosocial approaches have shown promise in the treatment of pediatric depression [296-298]. Of the various psychosocial interventions, cognitive-behavioral therapy (CBT) has received the most empirical attention in children and adolescents. Randomized controlled trials of CBT have been conducted in both clinical and community samples, targeting depressive symptoms or clinical depression [296-298]. Although CBT programs for pediatric depression broadly target cognitive distortions and behavioral skill deficits, specific CBT manuals vary substantially in the extent to which they emphasize cognitive vs. behavioral strategies, employ techniques drawn from other therapeutic interventions (e.g., family therapy), utilize a group vs. individual approach, or require meeting with the youth alone or involve parents in some components of the treatment [299]. The programs also differ in the number and overall structure of the sessions.

In addition to CBT, interpersonal psychotherapy has shown efficacy in adolescent depression [296, 299]. Interpersonal psychotherapy for adolescents (IPT-A) focuses more strongly on reducing interpersonal stress and developing more adaptive responses to interpersonal difficulties [300]. IPT-A also appears to target cognitive processes [299]. There is limited evidence for the efficacy of family therapy in adolescent depression [301, 302].

Despite the demonstrated efficacy of psychosocial interventions for pediatric depression, CBT in particular, dissemination of these therapeutic interventions in clinical settings has not been successful [299, 303]. Effect sizes for psychotherapeutic interventions in mental health clinics are significantly lower than those seen in randomized efficacy trials [304]. A number of factors might explain this discrepancy to this gap, including the characteristics of youth and families enrolled research studies versus those seen in community care [121, 304], and the resources available for training and supervision in controlled trials versus practice settings [299, 305]. There is also a lack of understanding of which core components of empirically-based treatments need to be disseminated because very few studies measured the core processes of treatment and evaluated whether the changes in these processes accounted for intervention outcomes [299, 306]. In order to bridge the gap between laboratory and clinical settings, a dramatic shift is necessary both in research and policy.

Combined Pharmacological and Psychosocial Intervention

Reviews of controlled studies in adult depression have historically concluded that the combination of medication and psychotherapy does not yield appreciable benefits above the effects of medication or psychotherapy alone (see [307, 308]). However, combined treatment may be more effective than psychotherapy alone for more severe or recurrent depression [308]. In a recent study, combined treatment was more effective (73% response rate) than medication (48%) or psychotherapy (48%) in the treatment of chronic depression [309]. In a sample of 378 adolescents receiving 12 weeks of treatment for depression, 71% responded to combination treatment, compared to 61% who received fluoxetine alone, 43% who received CBT alone, and 35% of those treated with placebo [291]. In a more recent study of 334 adolescents whose depression did not respond to an SSRI, the subjects were randomized to switch to another SSRI, switch to venlafaxine, or either of these medication strategies in conjunction with CBT. At 12 weeks, response rates were 47% for alternate SSRI and 48% for venlafaxine. Overall, 40.5% of those treated with medication alone responded vs. 54.8% for the combined treatment [310]. Additional trials should be conducted in children and adolescents, also including placebo conditions for psychotherapy, to assess whether certain types of depression (i.e., more severe or chronic or recurrent episodes) require combination therapy, or whether combination therapy is important even for first episodes that may be relatively brief.

SUMMARY AND FUTURE DIRECTIONS

Pediatric depression is of great public health significance, with serious morbidity and mortality, thereby emphasizing the need for early identification and effective treatment strategies. In the past three decades, considerable advances have been made regarding our knowledge on the phenomenology and natural course of depression in children and adolescents. Basic epidemiologic and clinical research also has helped identify a number of risk factors associated with pediatric depression. Moreover, there is emerging literature on the short-term efficacy of both psychosocial and pharmacological interventions.

Additional research is needed in understanding the pathogenesis of early-onset illness. There appears to be a complex interplay among genetic, neurobiological, cognitive and environmental factors in concert with developmental challenges and gender influences in the onset and maintenance of depression in youth. Therefore, studies aimed at elucidating mechanisms and interrelationships among the different domains of risk factors will be helpful in developing effective treatment and preventive strategies. Intervention research also needs to be expanded, testing a variety of interventions both individually and in combination, targeting not only depressive symptoms but also comorbid conditions and functional measures. The identification of potential mediators and moderators of clinical response is critical for implementing more specific treatments for the different subgroups of patients. In all these aspects, it is important to examine developmental continuities and discontinuities among children, adolescents and adults. Understanding the maturational variations will be helpful not only for developing more effective interventions for youngsters but also for adult patients who do not respond adequately to the currently available treatments.

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Chapter 4

CHILDREN, DEPRESSION AND ESSENTIAL FATTY ACIDS

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ABSTRACT

The World Health Organisation (WHO) estimates that major depression is the single greatest cause of years of life lost to disability worldwide (Global Health Forum for Research, 2008). In the United Kingdom, the cost of mental ill-health is currently estimated at £77 billion pounds, greater than heart disease and cancer combined. By 2020, the WHO predicts a 50% rise in child mental health disorders. The rise in mental health disorders may reflect the dramatic change in diet observed during the past century favouring mass food production coupled with a global decrease in fish and seafood consumption both during pregnancy and in children of school age. It is well established that polyunsaturated fatty acids are vital for the optimal development of both the brain and retina. They are abundant in the central nervous system and play a pivotal role in neurotransmission, serotonergic and dopaminergic function. The omega 3, docosahexaenoic acid (DHA; 22: 6n-3) and the omega-6, Arachidonic Acid (AA; 20:4n-6) are key components of cell membranes; they are required for normal brain structure and early visual development during pregnancy and infancy. Low maternal seafood consumption, that is less than 340 grams per week during pregnancy, is linked with higher scores of suboptimal outcome across a range of developmental measures in children. Furthermore, children with lower levels of omega-3 fatty acids are reported to have more temper tantrums, sleep disorders and behavioural problems than controls. This chapter discusses the role of nutrition in child and adolescent depression, with specific reference to essential fatty acids and brain function. An overview of specific comorbid neurodevelopmental disorders (such as attention deficit hyperactivity disorder; ADHD) is provided along with clinical trial evidence of the efficacy and safety of dietary

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supplementation. The review concludes with an evaluation of nutritional trends as reflected in the blood profiles of patients with depression and ADHD.

INTRODUCTION

According to the World Health Organisation (WHO), major depression is likely the single greatest cause of years of life lost to disability worldwide (Global Health Forum for Research, 2008). In the United Kingdom, the cost of mental ill-health is currently estimated at £77 billion. This is greater than heart disease and cancer combined. By 2020, the WHO predicts a 50% rise in child mental health disorders (Global Health Forum for Research, 2008). The investigation of the relationship between diet and behaviour in children is of current scientific and government interest especially due to the rise in anti-social behaviour and reported increase in statements of special educational needs among UK school children and adolescents (Associate Parliamentary Food and Health Forum, 2008). Research efforts into nutrition and mental health are currently being undertaken with specific focus on the potential benefit of essential fatty acids (EFAs) to the mood, skills and cognitive development of young people (Sinn, Bryan & Wilson, 2008; Johnson, Östlund, Fransson, Kadesjo, & Gillberg, 2008; Gow, Matsudaira, Taylor, Crawford, Ghebremeskel, Ibrahimovic, Vallée-Tourangeau, Williams & Sumich., 2009). It is well established that polyunsaturated fatty acids are vital for the optimal development of both the brain and retina (Innis, 1991; 1997). They are abundant in the central nervous system and play a pivotal role in neurotransmission, serotonergic and dopaminergic function (Chalon, 2006). Several studies have reported relationships between DHA intake in infants and increased visual acuteness, motor skills and IQ scores (Willats, Forsyth, DiModugno, Varma, & Colvin, 1998; Dunstan, Simmer, Dixon, & Prescott, 2008). In contrast, low maternal seafood consumption, that is less than 340 grams per week during pregnancy, is linked with higher scores of suboptimal outcome in children across a range of measures including fine motor skills, verbal IQ and social development (Hibbeln, Davis, Steer, Emmett, Rogers, Williams, & Golding, 2007). Furthermore, children with lower levels of omega-3 fatty acids are reported to have more temper tantrums, sleep disorders and behavioural problems than controls (Burgess, Stevens, Zhang, & Peck, 2000). Following the publication of several randomised controlled trials which reported reductions in symptoms of depression following supplementation with fish oil (Peet & Horrobin, 2002; Jazayeri, Tehrani-Doost, Keshavarz, Hosseini, Djazayery, 2008), the American Psychiatric Association (2006) recommended that people with mood disorders such as depression should consume at least 1 gram of EPA and DHA daily (Freeman, Hibbeln, Wisner, Davis, Mischoulon et al., 2006). However, the impact of nutrition in children in relation to depression is currently underfunded and under researched.

This chapter aims to discuss the role of nutrition in mental health in relation to child and adolescent depression with specific reference to essential fatty acids and brain function. Evidence for the efficacy and safety of essential fatty acid supplementation in depression from randomised clinical trials will also be presented and discussed. Depression in children and adolescents is highly comorbid with attention deficit hyperactivity disorder (ADHD). Furthermore, both conditions are associated with abnormal essential fatty acid blood profiles. In light of this, an overview of the research in both these areas will be presented. The final

section will discuss the abnormal EFA blood profiles and discuss whether they may be due to a problem with deficiencies or metabolism before concluding.

DEPRESSION IN CHILDREN AND ADOLESCENTS

Depression is a psychiatric disorder characterised by a shift in mood from pleasant to unpleasant, also known as *dysphoria* or a *dysphoric mood* (DSM-IV-TR, 2002). In teenagers it is linked to a range of negative outcomes and is often presented as irritability rather than displays of sadness or depression (DSM-IV-TR, 2002). A dysphoric mood is described as pervasive, persisting over time and sufficiently severe to disrupt ability and/or interest in school work or spending time with friends. Physical characteristics and symptoms of depression can include a fluctuation in appetite, sleep disturbance, fatigue and loss of energy (DSM-IV-TR, 2002). It is also accompanied by other symptoms such as a diminished ability to think, concentrate or pay attention, indecisiveness, and feelings of worthlessness (DSM-IV-TR, 2002). A negative or distorted self-image can often result in the loss or gain of weight in affected adolescents. It is important to note that for a diagnosis of depression, the presenting symptoms should not be as a direct result of the misuse of substances and/or medication, nor be accounted for by a medical condition, or follow a bereavement/loss of a loved one (DSM-IV-TR, 2002). An accurate assessment of depression can only really be obtained through direct questioning of the individual by significant others such as parents. For a clinical diagnosis at least 5 or more symptoms must be met according to the DSM-IV and be persistent for a minimum duration of 2 weeks (DSM-IV-TR, 2002). Episodes of depression are changeable, ranging from mild, suggesting a slight deviation from normal functioning, to severe. In severe cases, the result is often an inability to look after one self which can result in psychiatric care (Harrington, 2003). Adolescents dealing with depressive symptoms might attempt to cope with affective problems by over-investing time and energy with certain types of peer groups, experimenting sexually, using illicit substances and self harm (Shaffer & Waslick, 2002; Harrington, 2001; Kovacs et al., 1993).

Spitz (1946) was among the earliest researchers to examine depression in children. His work described the condition *anaclitic depression* which involved the separation of nursery infants from their mother. In addition, to withdrawal and weepiness, the children displayed signs of loss of appetite, slow reactions and retardation of movement and development. Spitz and Wolf (1946) hypothesised that the most significant aetiological factor was the "loss of the love object". Family dysfunction (along with emotional problems in parents) is also cited as a reliable predictor of continued depressive symptoms into early adulthood (Velez, Johnson, & Cohen, 1989). It is important to note, that no mention of nutrition is cited in the early literature concerning depression in children and adolescents. The knowledge and understanding that nutrition can mediate and influence brain function and neurotransmission has been largely overlooked from a psychiatric perspective until recently.

The psychiatric approach in depression to date tends to focus on the association between genetic factors and low levels of the brain transmitters serotonin and norepinephrine (Davey, 2008). Stemming from this biological approach, various drugs have been developed with some success in an attempt to address these imbalances and alleviate symptoms of depression (Davey, 2008). However, there are adverse side effects to medication such as fluoxetine

(Prozac) which include sexual dysfunction, headaches and gastric disorders (Rosen, Lane, & Menza, 1999). Moreover, fluoxetine use has recently been associated with an increased risk of suicide in teenagers and young adults (Gunnell, Saperia, & Ashby, 2005). Many patients with depression are also treatment resistant and it has been noted, that complementary and alternative treatments, such as dietary supplements, are often sought as a replacement to conventional medication (NICE Guidelines, Appendix K, p. 199; Jorm, Korten, Rodgers, Pollitt, Jacomb, et al., 1997).

Researchers working with paediatric populations have identified various risk factors for depression arising from a combination of biological, psychological, social/environmental and more recently genetic variables (Davey, 2008). However, little attention is paid to dietary influences, eating habits or nutrition and this is probably attributable to a lack of knowledge on how these variables can mediate mood (Bamber, Stokes, & Stephen, 2007). Diet, along with other aspects of daily living such as exercise, has had a crucial role in the evolution of the brain and its cognitive capacities (Gomez-Pinilla, 2008; Crawford & Marsh, 1989). Recent advances in molecular biology have highlighted the ability that food has releasing signals which in turn can influence energy metabolism and synaptic plasticity. In other words, food has a mediating effect on cognitive function (Gomez-Pinilla, 2008). The action of specific dietary interventions, such as omega-3 fatty acids on the activation of molecular systems that are involved in synaptic plasticity has strong implications for public health and therapeutic interventions (Gomez-Pinilla, 2008). By understanding the molecular basis of the effects of food on cognition one can manipulate the diet in order to strengthen the resistance of neurons to damage while simultaneously promoting mental fitness (Gomez-Pinilla, 2008). There is a pressing need for alternative support in the emotional and behavioural difficulties faced by some young people (Bamber, 2007). It is estimated that 50% of referrals to child and adolescent mental health services in the UK alone could be markedly reduced if schools were equipped with evidence-based knowledge and the tools to carry out focused health programmes (Bamber, 2007) such as improvements in school meals. New research by Haskell, Scholey, Jackson, Elliot and Defeyter et al., (2008) has reported that providing children with multi-vitamin and mineral supplementation improved performance in assessments of brain function; a proposition that warrants further investigation.

The manufacture of food has dramatically altered during the past century in favour of mass production; wheat and grains are now highly refined and stripped of much nutritional content; the addition of artificial additives and preservatives is in practically every supermarket foods; sugar and vegetable oils are refined and the result of the hydrogenation process is the novel *trans* fatty acid (Cordain, Boyd-Eaton, Sebastian, Mann, Lindeberg, et al., 2004). Arguably, these novel means of food production go against the evolution of the genome, have altered nutritional characteristics of ancestral hominid diets and contribute to the increase in diseases among so-called civilised populations (Cordain, 2004). The health implications concerning the changes in agriculture and food production have not gone unnoticed. A letter to the Lancet by the scientist Hugh Sinclair (1956) argued that defects in intake or metabolism of EFAs may play a fundamental role in diseases common to Western society. However, his advice was deemed controversial as it contractided the current paradigm. He was not only ignored but subsequently ostracised by the medical society, unable to obtain research grants to investigate this further. It is now established that an association exists between nutrition (and a lack of omega-3 fatty acids) and the rise in

cardiovascular disease, obesity and mental illness (Horrobin, 1990; GISSI-HF, 2008; Klein-Platat, Drai, Oujaa, Schienger & Simon, 2005; Peet, 2002; Cordain ET AL., 2004).

In addition to EFAs, it is important to note that brain function requires an adequate amount of key vitamins and trace minerals such as zinc, magnesium, folate, iron, selenium, and the C and B family of vitamins (Watts, 2008). These are needed not only for the synthesis of DHA but for the production of serotonin, dopamine, noradrenaline and similar neurotransmitters (Watts, 2008). In spite of this knowledge, research by the U.S. Department of Agriculture in the 1990's suggested that children and adolescents are lacking in folate, magnesium, zinc, and vitamins A and E (The School Nutrition Dietary Assessment Study, 1993). In addition, research by Bruner, Joffe, Duggan, Casella, and Brandt, (1996) report that approximately 25% of adolescent girls in the United States are deficient in iron (Bruner, Joffe, Duggan, Casella, & Brandt, 1996). However, the affect of these deficiencies on brain function remain relatively unknown. A recent clinical trial which supplemented children and young people with zinc reported some promising results with significant improvements in hyperactive and restless behaviours (Bilici, Yildirim, Kandal, Bekaroglu, Yildirim, Deger et al., 2004).

In reference to food additives, a publication by McCann, Barrett, Cooper, Crumpler, Dalen et al., (2008) has confirmed that the preservative, sodium benzoate and artificial colouring can cause hyperactivity in children. High levels of hyperactive behaviours (inattention, over activity and impulsivity) in young children often result in a diagnosis of ADHD and are also associated with educational difficulties especially related to reading (McCann, Barrett, Cooper, Crumpler, Dalen et al., 2008; McGee, Prior, Williams, Smart, & Sanson, 2002). In light of the health importance of vitamins, minerals and essential fatty acids more research is required to address optimum intake and potential deficiencies in children, to further explore and understand the links between mental health and nutrition (Associate Parliamentary Food and Health Forum, 2008).

ESSENTIAL FATTY ACIDS (EFA)

The association between long-chain polyunsaturated fatty acids (LC-PUFA) and mental illness has steadily increased since the role of omega-3 PUFA in brain structure and function became apparent in the 1970's through the work of Crawford and his colleagues. Crawford and Sinclair (1972) were the first to demonstrate that arachidonic and docosahexaenoic acids were essential for mammalian brain development and evolution. Their pioneering work with primates provided experimental evidence for specific omega-3 fatty acid deficiency inducing behavioural pathology (Fiennes, Sinclair, & Crawford, 1973). Since then there have been indications that essential fatty acid supplementation may benefit several psychiatric, neurological and neurodevelopmental illnesses, such as depression, bi-polar disorder, schizophrenia, dementia, attention deficit hyperactivity disorder (ADHD), dyspraxia, dyslexia, and autistic spectrum disorders.

Several mechanisms of action have been proposed to account for the potential benefits of essential fatty acids, predominately related to their structural role in the brain and neurotransmission (Sinn & Howe, 2008). Traditionally, neurobioogical research into the origin and cause of psychiatric illness has centered on the role of neurotransmitters (e.g.

serotonin and dopamine release and malfunction). However, there is also a strong and plausible link between mental health, cerebral blood flow (CBF) and impairments in blood-brain barrier function (Sinn & Howe, 2008). The relationship between psychiatric pathologies and cardiovascular disease further indicate the potential underlying vascular component in psychiatric illness. For example, it has been proposed that treatment with vasoactive nutrients (such as omega-3 fatty acids) which improve cerebral perfusion may contribute towards an improvement in mental disorders (for a review see Sinn & Howe, 2008).

Long-chain polyunsaturated fatty acids (LC-PUFAs) are known as essential fatty acids because they cannot be made or stored by the body for very long periods of time and therefore must be obtained via the diet (Horrobin, 1990). They are implicated in practically every significant biological process in the central nervous system and crucial for its early development. Polyunsaturated fatty acids (PUFA) are carbon atoms linked together with two or more double bonds (Cunnane, 2006). Most fatty acids can be synthesised by the body. However humans lack the enzyme needed to produce two *essential* fatty acids; these are the omega-3 PUFA α-linolenic acid (ALA) and the omega-6 PUFA linolenic acid (LA); the parent compounds or precursors to other omega-6 and omega-3 EFAs (Cunnane, 2006). ALA is metabolised into the long chain omega-3 PUFAs eicosapentaenoic acid (EPA) and DHA. The biochemical pathway is complex and the conversion process is influenced by many factors including oxidative stress, alcohol, smoking, age and genetic factors (Crawford, 1989). Omega-3 and omega-6 EFAs also compete for incorporation into cell membranes (Yehuda & Carrosa, 1993). Thus, diets rich in omega-6 and saturated fats would result in very little omega-3 in phospholipids (Crawford, 1989). The optimum ratio of omega-6 to omega-3 for physical and mental well being is estimated to be 4:1 which is in stark contrast to the approximately 20:1 that has been reported in a typical Western diet (Yehuda & Carrosa, 1993). The omega-6 EFA, LA is typically found in fried and 'fast' foods, seed and vegetable oils and other commercially manufactured foods (McGregor, Allen, Mary, Reece, Wheeler et al., 2001) and is estimated to make up in excess of 85% of the total PUFA intake in the Western diet (Sinclair, Johnson, O'Dea, & Holman, 1994). This percentage considerably favours omega-6 over omega-3 fatty acids with potentially unfavourable consequences for physical and mental health (Mostofsky, Yehuda, & Salem, 2001; Hallahan & Garland, 2005). ALA is found in green leafy vegetables, some seeds and oils such as flax seeds and canola oil, seaweed and algae. However, seafood and oily fish are the richest source of natural foods that contain sufficient quantities of EPA and DHA, as well as the trace elements needed to synthesise them (Crawford & Marsh, 1989). Recommended fish consumption in adults is at least twice a week, but optimal requirements for brain function are still not yet known. There is a definite lack of omega 3 fatty acids in most Western societies. Indeed, the omega 6/omega 3 balance has increased to the detriment of the omega 3 status (Ghosh, Butsanis, Ghebremeskel, Crawford, & Poston, 2001).

DHA, and the omega-6 fatty acid, AA are abundant in the brain, comprising approximately 6% to 8% of its non-aqueous weight (Horrobin, 2001). DHA and AA are found in high concentrations in the grey matter of the cerebral cortex, specifically in the membranes of neuronal synapses (Horrobin, 2001). In infants, lower concentrations of cortical DHA are reported in formula fed babies compared to those that were breast fed (Farquharson, Cockburn, Ainslie-Patrick, Cherry Jamieson & Logan, 1992). The precursor of DHA, EPA, is also considered of importance in the brain, specifically in its role in eicosanoid production; eicosanoids have anti-inflammatory, anti-thrombotic and vasodilatory properties

(Sinn & Howe, 2008). Both EPA and DHA are associated with many important functions related to neural activity such as membrane fluidity, neurotransmission, ion channel, enzyme regulation, gene expression and myelination (Sinn & Howe, 2008).

The brain is critically dependent on a steady supply of oxygen and glucose for cell maintenance and information processing. Glucose is the key energy source for the brain and due to the brains limited energy storage capability it depends on a constant supply from the blood (Sinn & Howe, 2008). Chemical transmission and metabolic activities in the brain also rely on a large variety of nutrients which need to be transported by the blood across the blood brain barrier. The transportation of substrates including EFAs across the blood brain barrier are carried out by a family of six proteins (Albumins, Apolipoprotein, Lipoprotein, Fatty Acid transport protein; FATPs, Fatty Acid binding proteins; FABPs and Acylation stimulating proteins; ASPs) and therefore the relationship between protein and plasma along with cerebral blood flow are crucial components of brain function (Horrobin, 2001; Sinn & Howe, 2008).

COGNITIVE DEVELOPMENT AND EFAS

The role of DHA in visual and cognitive development during infancy and childhood is of significant scientific interest. DHA is extremely concentrated in the photoreceptor membranes of the retina and is implicated in both learning and vision (Innis, 2007). DHA and AA are vital for normal brain growth and a precursor of the series 2 eicosanoids that induce labor (Innis, 1991). In the third trimester of pregnancy, a time of prolific synaptogenesis and photoreceptor cell development, DHA accumulates preferentially in the brain of the fetus. The ability to synthesise DHA and AA is restricted until 16 weeks postpartum. There is active transport across the placenta which is evidenced by the significantly larger amounts of DHA concentrations in the fetus relative to the mother. Following delivery substantial amounts of DHA and AA are transferred via the mother's milk (Martinez, 1992; Dutta-Roy, 2000; Birch, Garfield, Hoffman, Uauy, & Birch, 2000). The levels of DHA will vary in humans according to dietary intake. DHA can be synthesised by its precursor ALA, but the conversion is limited therefore a direct supply is more likely to ensure optimal benefit. For infants, the mother's milk is the most appropriate source of DHA but the amount transferred to the child will depend on the mother's intake. Mothers who consume fish and other food sources containing omega-3 LC-PUFA produce breast milk with higher DHA levels compared to those who do not (Yuhas, Pramuk, & Lien, 2006). Infants who are not breastfed can receive a supply of DHA and AA via commercially available infant formula but this has only been the case since 2002 following the pioneering work of Carlson (2001). Despite this, a typical American diet for children today contains very little DHA and LC-PUFAs compared to children's diets in other industrialised nations (Ervin, Wright, Wang, & Kennedy-Stephenson, 2004).

A recent review concluded that mothers receiving omega-3 LC-PUFA supplementation during pregnancy and lactation produce off-spring with enhanced cognitive development (Eilander, Hundscheid, & Osendarp, 2007). A quantitative analysis of eight trials estimated that an intake of 1 gram per day of DHA during pregnancy increases a child's IQ by 0.8 to 1.8 points; the children were followed up at different ages ranging from 4 months to 4 years of age (Cohen, Bellinger, Connor, & Shaywitz, 2005). Electrophysiological tests have shown

that supplementation with DHA and AA in infants' improved visual development (Eilander, Hundscheid, Osendarp et al., 2007).

Jensen, Voigt, Prager, Zou, Fraley et al., (2005) examined the effects of maternal supplementation with DHA on the neurodevelopment and visual function in breastfed term infants. Cognition was measured at 30 months of age. The findings revealed significantly higher scores on the Bayley Psychomotor Development Index following supplementation with DHA. No improvement was found in scores for the Mental Development Index (Jensen et al., 2005). Birch, Garfield, Castaneda, Hughbanks-Wheaton, Uauyand & Hoffman (2007) observed infants over 4 years who were supplemented with DHA and AA during their first 17 weeks of life. They found that supplementation of infant formula with DHA and AA resulted in visual and cognitive development akin to that of breastfed babies (Birch et al., 2007). Zhang, Herbert and Muldoon (2005) examined cross-sectional dietary intake data from the Third National Health and Nutritional Examination Survey (1988-1994) to assess the relationship between LC-PUFA intake and psychosocial and cognitive performance of children aged between 6 and 16 years old. They concluded that LC-PUFA intake was associated with improved working memory performance. However, the exact intakes of omega-3 and 6 fatty acids were not reported. Several other clinical trials have reported an increase in the length of gestation following DHA supplementation during pregnancy (Olsen et al., 1999; 2000; Smuts et al., 2003).

A recent study by Jacobson, Jacobson, Muckle, Kaplan-Estrin, and Ayotte (2008) found that DHA was significantly higher in cord plasma than maternal plasma, adding weight to evidence which suggests that there is a preferential placenta uptake of DHA during the last trimester spurt of synaptogenesis in brain and photoreceptor development (Dutta-Roy, 2000). The strong correlation between cord and maternal DHA concentration is also consistent with data from 5 other countries (Otto, Houwelingen, Antaal, Manninen, Godfrey et al., 1997) and supports the dependence of the fetus on the mother for a sufficient quantity of DHA (Jacobson, Jacobson, Muckle, Kaplan-Estrin, Ayotte, & Dewailly, 2008).

Inadequate supplies of DHA are associated with impaired learning and attention in addition to emotional irregularities such as increased depression, anxiety and aggression (Fedorova & Salem, 2006). Mathieu, Denis, Lavialle and Vancassel (2008) have shown that these effects are in part based on changes in neurotransmission function. They showed that dietary induced deficiencies in DHA result in a deregulation of the meso-cortico-limbic dopaminergic pathway which is implicated in emotion and reward processes (Zimmer, Hembert, Durand, Breton, Guilloteau et al., 1998; Zimmer, Delion-Vancassel, Durand, Guilloteau & Bodard et al., 2000; Zimmer, Vancassel, Cantagrel, Breton, Delamanche et al., 2002).

Animal Studies

In rodents receiving a chronically deficient omega-3 diet a reduction of the release of serotonin and acetylcholine in the hippocampus is observed. Furthermore, the omega-3 PUFA contents of the diet influenced the serotonergic receptor and the muscarinic receptor binding. These results were thought to negatively contribute to poor performance across a series of cognitive tasks in the rats that were given an omega-3 deficient diet (Mathieu, Denis, Lavialle & Vancassel, 2008).

Brain Composition of DHA and Risk Factors

Research now suggests that deficiencies in the brain composition of DHA during perinatal development may represent a preventable neurodevelopmental risk factor for the later emergence of psychopathology (McNamara, 2006). Epidemiology studies have revealed an association between reduced DHA levels and disorders such as ADHD and depression (Young, Maharaj & Conquer et al., 2004; 2005; Maes et al., 1999; Tanskanen et al., 2001). Comparably, prophylactic effects of omega-3 PUFA intake for mood disorders have recently been reported in a meta-analysis (Freeman et al., 2006). Evidence also shows how postnatal child rearing conditions can wield a continued modulation over neural systems thus impacting a predisposition to psychopathology in adults (Mathieu, Denis, Lavialle, & Vancassel, 2008). Early adverse life experiences such as neglect, loss of a parent, and abuse are associated with altered neurobiological and physiological function alongside long-term vulnerability to depressive disorders (Mathieu et al., 2008). The early bond between mother and child is considered key for the optimal development of the infant (Hofer, 1994). The disruption of normal maternal-infant interaction by early maternal separation (MS) is considered one of the most powerful stressors in animal studies. Adults that have been separated from their mother as children are thought to possibly have a higher risk for drug abuse (indicated by changes in locomotor sensitization to morphine in animal studies). As a consequence they exhibit increased levels of stress hormone responsiveness and alterations in emotion and behaviour regulation when challenged in particular experimental conditions (Plotsky & Meaney, 1993). Alterations in levels of neurotransmitters in the brain play a large role in the behavioural effects induced by continual disruption of the mother-child bond with particular changes in limbic structures in relation to adjustments of the dopamine systems involved in reward and emotion. These effects tend to be reversible with anti-depressant treatments providing support for the validity of the MS model (Mathieu, Denis, Lavialle & Vancassel, 2008).

DEPRESSION, AGGRESSION AND EFAS

Several lines of research have established an association between major depressive disorders and omega-3 fatty acids in adults (Logan, 2003). Fish and seafood are the main dietary source of omega-3 PUFAs, and irregular fish intake cross culturally is linked with depression in epidemiological studies (Sontrop & Campbell, 2006). Research in this area has focused on links between specific nutrients and depression. The most studied nutrients, and for which the evidence is strongest, is omega-3 fatty acids and folic acid (Peet et al, 1998; Papakostas, Petersen, Mischoulon, Green, Nierenberg et al, 2004). High levels of fatty acids lead to an increase in the fluidity of membranes which in turn increases the transport of serotonin into the endothelial cells (Block & Edwards, 1987). People suffering from depression are reported to have reduced serotonin uptake (Block & Edwards, 1987) and therefore the role of fatty acids in depression has important implications.

The only placebo-controlled double-blind, pilot study to investigate EFAs and childhood depression was carried out by Nemets, Nemets, Apter, Bracha and Belmaker (2006). They recruited 28 children and randomised them into groups to receive one 1000 g of fish oil containing 190 mg EPA and 90 mg of DHA (active supplement) or placebo containing olive

oil. Participants were recruited via the depression clinic or child psychiatry clinic in Israel. Supplementation lasted 16 weeks and assessments of depression using the Childhood Depression Rating Scale (CDRS), Childhood Depression Inventory (CDI) and Clinical Global Impression (CGI) were taken at baseline, 2, 4, 8, 12 and 16 weeks. From the 28 children recruited, 20 completed at least 1 month's ratings and were included in the data analysis (n=10 active and n=10 placebo). The results found highly significant effects of omega-3 fatty acids on self-rated symptoms of depression using the CDRS, CDI and CGI concluding that they may have therapeutic benefits in childhood depression. Further research is needed with larger sample sizes to replicate these findings.

Affective impairment seen in attention deficit hyperactivity disorder (ADHD) may underlie co-morbid depression, anxiety and/or conduct disorder. Low levels of LC-PUFA, specifically omega-3 fatty acids in blood measures have been linked to a range of behavioural and mood disorders including ADHD. However, little is known about the relationship between omega-3 and brain function in humans. A recent study by Gow and colleagues found that total red blood cell concentrations of omega-3 fatty acids are associated with emotion elicited neural activity in twenty adolescent boys with ADHD (Gow, Matsudaira, Taylor, Crawford, Ghebremeskel, Ibrahimovic, Vallée-Tourangeau, Williams & Sumich, 2009). They assessed the total lipid fractions in the red blood cells of the young boys (aged 12-16 years old) and their event-related potential (ERP) response to the presentation of facial expressions of happiness, sadness and fearfulness. The results supported the hypothesis of a positive association between EPA and a cognitive bias in orientation to overt expressions of happiness over both sad and fearful faces as indexed by midline frontal P300 amplitude. Additional exploratory analyses revealed a positive association between levels of DHA as well as the AA/DHA ratio and the right temporal N170 amplitude in response to covert expressions of fear. These findings indicate that EPA and DHA may be involved in distinct aspects of affect processing in ADHD and have implications for understanding currently inconsistent findings in the literature on EFA supplementation in ADHD and depression (Gow et al., 2009).

Clinical Trials with EFAs in Adults

Although, the research in EFAs and depression in children is limited it is pertinent to mention the clinical investigations in this area with adults with depression due to the implications they hold for future research. The potential benefit of omega-3's (specifically high EPA supplementation) has been explored as an adjunct to treatment with medication in adults. For example, Peet and Horrobin (2002) recruited patients with depression whose initial experience with antidepressant medication had been unsuccessful and gave them 1, 2, or 4g of ethyl-EPA. Those patients taking ethyl-EPA showed a significant improvement compared to those on placebo (see also Sontrop & Campbell, 2006 for a review; DeMar, Bell, Igarashi, Greenstein & Rapoport, 2006, for evidence supporting the link between EFA and depression in animals)

A very strong negative correlation between fish consumption and global prevalence of major depression has been reported recently in The Lancet. Germany, Canada and New Zealand had the highest prevalence of depression and Japan the lowest (Hibbeln, 2007; NIAAA; Lancet, 1998). Iceland is also reported to have a very low incident of depression

which could be due to the country's overall very high consumption of fish (approximately 225 lbs per person per year) as per Japan (147lbs per person per year; Cott & Hibbeln, 2001).

A recent placebo controlled double blind study by Jazayeri and colleagues recruited patients with depression (aged 20-59). The patients met the DSM-IV criteria for major depressive disorder without psychotic features and were randomised into 3 groups to receive 2 capsules of (i) (1000 mg) of EPA plus fluoxetine placebo, (ii) 1 fluoxetine capsule (20 mg fluoxetine) plus ethyl-EPA placebo, or (iii) 2 ethyl-EPA soft gels (1000 mg EPA) plus 1 fluoxetine capsule (20 mg fluoxetine) for 8 weeks. Psychiatric assessments were carried out at weeks 2, 4, 6, and 8. Compliance was set at 90% of consumption of the medication. Results showed that the fluoxetine and EPA combination was significantly better than fluoxetine or EPA alone. Fluoxetine and EPA appeared to be equally effective in controlling depressive symptoms. Treatment was shown to have an effect at both weeks 4 and 6. Response rates ($\geq$ 50% decrease in baseline Hamilton Depression Rating Scale) were 50%, 56% and 81% in the fluoxetine, EPA and combination groups respectively. These findings suggest that EPA had equal therapeutic benefits to fluoxetine but was superior as an adjunctive treatment with fluoxetine. The authors suggested that the consumption of dietary supplements may be more acceptable to patients than antidepressants. Furthermore, because major depression is also a risk factor for cardiovascular disease, supplementation with EPA may be of mutual benefit, reducing inflammatory cytokines and controlling depressive symptoms. Larger trials are needed to replicate these findings (Jazayeri, Tehrani-Doost, Keshavarz, Hosseini, & Djazayery et al., 2008).

Other studies have been conducted investigating the efficacy of omega-3 to aggression (which is comorbid with depression and anxiety) and psychopathic disorders. For example, Gesch, Hammond, Hampson, Eves, and Crowder (2002) examined the effect of supplementary essential fatty acids (in conjunction with a vitamin supplement) in a young adult UK prison (aged 18 – 21 years) population using a randomised, double blind design. The aim of the study was specifically to investigate whether alterations in diet by way of supplementation could reduce anti-social behaviours among inmates. Two hundred and thirty one young offenders took active (1260 mg linoleic acid, 160 mg gamma linolenic acid, 80 mg eicosapentaenoic acid and 44 mg docosahexaenoic acid) or placebo supplementation for an average period of 142 days. The results revealed a marked reduction in anti social behaviour and violent offences for active versus placebo. It was concluded that the supplementation of vitamins, minerals and essential fatty acids reduces anti-social behaviour in a prison population (Gesch et al., 2002).

The work of Gesch and colleagues (2002) investigating the link between diet and aggression is also supported by others. Itomura and colleagues, (2005) carried out a randomised, placebo-controlled, double blind trial in Japan. They reported a reduction in physical aggression following supplementation of fish oil (3600 mg of DHA, 840 mg of EPA in fortified foods) in school children aged 9 -12 years of age (Itomura, Hamazaki, Sawazaki, Kobayashi, Terasawa, Watanabe & Hamazaki, 2005). In the USA, Schoenthaler (1983) carried out an experimental study substantially reducing the sugar content in the diets of 3000 imprisoned juveniles. Instead, the young offenders were given healthier snack options containing reduced sugar and refined foods. The results of the study over a 12 month period showed a 21% reduction in antisocial behaviour, 25% reduction in assaults, 75% reduction in physical restraints by staff and a 100% reduction in suicides (Schoenthaler, 1983). In a similar study by the same author with 402 Californian prisoners those given 100% of the U.S.

recommended daily allowance of vitamins committed fewer offences than those given 300% implying that the *correct* dose is crucial for optimum brain function (Schoenthaler, 1983).

Suicide Risk and Trends

Suicide trends among youths in the United States were reported to have increased by 18 percent during the year 2003 to 2004 (Bridge, Greenhouse, Weldon, Campo, Kelleher et al., 2008). Furthermore, questions have been raised concerning the safety and efficacy of both anti-depressant medication, and selective serotonin reuptake inhibitors (SSRIs), and the potential increased risk of suicidal thoughts and behaviour in young people (Moller, Baldwin, Goodwin, Kasper, Okasha et al., 2008). The link between mental health and nutrition in children is not well documented and investigations need to be undertaken to establish whether there is an association between nutrition and suicide risk in children. An elevated ratio of omega- 6/omega- 3 EFAs has however been found to predict the risk of suicide behaviour in depressed adult patients (Sublette, Hibbeln, Galfalvy, Oquendo & Mann, 2006). Sublette et al., (2006) measured the plasma levels of polyunsaturated fatty acid in the phospholipids in 33 medication-free depressed patients and monitored them for suicide attempts over a 2-year timeframe. On follow up, 7 patients in this group attempted suicide. A low DHA percentage, low omega-3 status and higher omega-6/omega-3 ratio of lipid profile predicted risk of suicidal behaviour. The authors concluded that low omega-3 fatty acids may adversely affect serotonergic and corticotrophic function, resulting in a greater susceptibility to suicide (Sublette et al., 2006).

Mild Depression in Young People

Crowe, Murray, Skeaff, Green, and Gray, (2007) explored the relationship between the levels of omega-3 LC-PUFA in serum phospholipids and scores of self-reported mental and physical well-being among adults and adolescents living in New Zealand. The study employed the short-form (SF-36) which is not a diagnostic tool for depression but rather assesses the mental and physical well being of a population. This study used data collected from the 1997 National Nutrition Survey which was a population-based survey that assessed the health status, health risk behaviours and health services in young adults aged 15 years and over. The 36 items were grouped into 8 scales that evaluate physical functioning (e.g., bodily pain and general health) and mental health (e.g., social functioning, limitations due to emotional health). The scales were standardised and aggregated by factor analysis to form a mental component and physical component score with a mean of 50 (SD = 10). The general health questionnaire also included 10 questions which constitute the Alcohol Use Disorders Identification Test. This test evaluates the consumption of alcohol and drinking behaviour over the past year. Values are ranked between, the lowest ‘abstainers’ and the highest ranking classification of ‘potential hazardous drinkers’. Multiple linear regression analyses were employed to examine the relation between key fatty acid indices and the mental and physical components.

The results showed significant inverse relationships for the proportion of women, those who had visited a mental health professional in the past 4 weeks across the quintiles of EPA.

Lower levels of DHA were related to the proportion of hazardous drinkers. There were no significant relationships across the quintiles of DHA for the physical or mental component scores. Over all, the results of this survey suggested that the amount of EPA and the ratio of EPA to AA in serum phospholipids were positively associated with self-reported physical well-being and there was a positive relationship between the ratio of EPA to AA and self-reported mental well-being. The authors concluded that higher levels of EPA may be a proxy for a diet with higher fish consumption which in turn was linked to increased rating of physical health. The results of this study are consistent with experimental evidence which shows that EPA is a better inhibitor than DHA of the synthesis of series-2 and series-4 leukotrienes from AA. The association has a solid biological plausibility and therefore deserves further investigation (Crowe et al., 2007).

The American Psychiatric Association recently reviewed the evidence in this area and formulated several recommendations for the use of omega-3 fatty acids. Namely, 1) that all adults should eat 2 portions of fish per week; 2) patients with mood, psychotic disorders or impulse control should consume 1 to 9 grams of EPA and DHA per day; and 3) supplementation should be considered in patients with mood disorders (between 1 to 9 grams) with doses over 3 grams per day monitored by a physician (Freedman et al., 2006). Recommendations by the Food Standards Agency regarding fish consumption for children less than 16 years of age are currently set at 2 portions of fish per week, one of which should be oily. However, children under 16 are advised not to eat shark, marlin and swordfish due to potential risk of mercury contamination.

COMORBID DISORDERS/ ATTENTION DEFICIT HYPERACTIVITY DISORDER

Children and adolescents with depression frequently have psychiatric comorbidities such as anxiety, behavioural conditions such as conduct disorder, oppositional defiant disorder and ADHD (Julien, 2001). This section will focus on ADHD as it is estimated to be highly comorbid with symptoms of depression in young people. ADHD is a neurodevelopmental disorder found worldwide and thought to affect 5% to 10% of all school children (Faraone, Sergeant, Gillberg, & Biederman, 2003). It has a complex etiology with multifaceted symptoms and a strong genetic underpinning (Biederman & Faraone, 2005) but the precise cause of ADHD to date remains unknown (Barkley, 1990; Taylor, 1999). There are three recognised subtypes of the disorder *hyperactive-impulsive*, *inattentive* and *combined*. The two distinct behavioural dimensions, inattention and hyperactive-impulsive have been recognised to exist across cultures in a variety of ethnic groups (Barkley, 2003). The *hyperactive-impulsive* subtype is multidimensional and characterised by deficits in inhibition. The *inattentive* component impairs an individual's ability to remember (affecting predominantly working memory) and therefore ADHD children struggle to follow instructions or abide by rules. Children with the disorder are more likely to generally misbehave, frequently interrupt and/or intrude on others' conversations and activities, not listen to adult instructions and impulsively repeat the same behaviours (de Boo & Prins, 2006). They often engage in high-risk taking, rule-violating, noisy and disruptive behaviour (Landau & Moore, 1991) which

frequently results in negative response from peers, teachers and parents (Guevremont & Dumas, 1994).

According to the most recent Diagnostic and Statistical Manual for Mental Illness (DSM-IV, American Psychiatric Association, 1994) the symptoms of ADHD must be present before the age of 7 years old, be persistent across at least two different situational contexts (e.g., at home and at school or work) and be severe enough to considerably impair everyday functioning (i.e., be chronic). The symptoms must have been present before the age of 7, have lasted for at least 6 months and are not accounted for by another disorder such as schizophrenia or a psychotic disorder (DSM-IV).

Several lines of enquiry have suggested a link between Attention Deficit Hyperactivity Disorder (ADHD) and deficiencies in omega3/6 fatty acids (Richardson & Puri, 2002; Richardson & Montgomery, 2005; Stevens et al., 1995). One of the first studies to suggest an association was published by Colquhoun and Bundy (1981). They surveyed the diets of 214 children (161 boys) that attended The Hyperactive Children's Support Group (HACSG) in the U.K. and suggested that boys with ADHD-type symptoms displayed signs of fatty acid deficiency. For example, they reported that two thirds of the children suffered from polydipsia (excessive thirst), had zinc values below the normal range and suffered from allergies including eczema, asthma with intolerances to milk and wheat; all of which are symptoms directly or indirectly related to EFA deficiency.

Since then, several randomised, placebo controlled, double blind trials have explored the plausibility of omega-3 supplementation to ADHD symptoms but with varying design methods and consequently results. For example, Voigt, Llorente, Jensen, Fraley, Berretta and Heird (2005) recruited 63 children with ADHD aged 6-12 years old, that were already successfully receiving stimulant medication, and randomly assigned them to receive either supplementation of an algae-derived triglyceride capsule (containing 345 mg of DHA only) or placebo for 4 months. There were no significant improvements in the outcome variables as measured by the Child Behaviour Checklist and Conners' Rating Scales (CRS). The active group was 2.6 fold higher in plasma phospholipid concentrations of DHA at completion of the study compared to the placebo group but the results concluded that a 4-month period of supplementation of DHA did not decrease ADHD symptoms (Voigt et al, 2005; see also Hirayama, Hamazaki, & Terasawa (2004).

The Oxford-Durham study however provided important evidence linking omega-3/6 supplementation and improvements in behaviour and concentration in underachieving mainstream schoolchildren (who had symptoms of ADHD and Developmental Coordination Disorder (DCD is also known as dyspraxia; Richardson & Montgomery, 2005). DCD is a motor coordination disorder which is comorbid with dyslexia and ADHD. It is characterised by poor impulse control/motor skills and writing difficulties. This randomised clinical trial (RCT) was placebo controlled and double blind. It recruited 117 school children aged 5-12 years old and supplemented them either an omega-3/6 fatty acid treatment (dose of active: x 6 daily; each containing 400 mg fish oil and 100 mg evening primrose oil with active ingredients EPA: 93 mg, DHA: 29 mg, gamma- linolenic acid; GLA: 10 mg, and vitamin E: 1.8 mg) or placebo (non-active; palm oil) for over a three month period. At 15 weeks the placebo group was crossed over onto the active supplement while the active group continued with the active supplement for a further 15 weeks.

The results showed no effects for motor skills, however significant improvements were observed for spelling, reading and behaviour over three months of treatment in the active

group compared to the placebo. Specifically, the active group over the first three-month period of treatment made a 6-month reading gain and a 3-month gain in spelling. The cross over from placebo to active groups showed similar changes while the active group continued to make improvements up to the end of the trial at six months. The authors concluded that fatty acid supplementation may be an effective treatment consideration for children with behavioural and educational difficulties associated with DCD (Richardson & Montgomery, 2005).

A similar study by Sinn and Bryan (2007) in Adelaide, Australia examined the effects of PUFAs supplementation on children with learning and behaviour problems associated with ADHD not DCD. One hundred and thirty two South Australian children aged 7-12 were recruited for this RCT who suffered from symptoms but were not necessarily diagnosed with ADHD. Children were randomised to receive either active (same supplementation, formula and dose as the Oxford-Durham trial: 400 mg fish oil and 100 mg evening primrose oil with active ingredients EPA: 93 mg, DHA: 29 mg, gamma- linolenic acid; GLA: 10 mg, and vitamin E: 1.8 mg) or placebo (palm oil) for a period of 3 months, followed by a one-way cross over for the placebo group to active for a further 3 months. The Parent and Teacher Conners Rating Scales were used to evaluate the change in symptoms from baseline and across the two study time-points (3 and 6 months). Overall, the findings of the Adelaide study revealed significant, medium to strong effects on Conners Parent Rating Scale (CPRS). Specifically, in the first 15 weeks of the trial significant improvements were observed for the active versus placebo group in *Cognitive Problems/Inattention*, on the Conners ADHD Index and *Restless/Impulsive* and *Oppositional Behaviour* on the ADHD Conners Global Index. Both are symptoms of the DSM-IV definition for ADHD. After the crossover at 15 weeks, the results were replicated; both treatment groups showed significant improvements on CPRS key symptoms. However, no significant effects were found for Conners Teachers Rating Scale (CTRS) at either 15 or 30 weeks or any significant difference with or without micronutrients. The dose of micronutrients (20 mg) was quite low and therefore may account for the lack of findings.

A recent trial placebo-controlled, RCT carried out by researchers at the University of Gothenburg in Sweden recruited 75 children and adolescents with ADHD aged between 8 and 18 years of age (Johnson, Östlund, Fransson, Kadesjö & Gillberg, 2008). The active group received omega-3/6 supplements (same formula and dose: x 6 daily as the two previously mentioned trials: 186 mg EPA, 58 mg DHA, 20 mg of GLA, Natural Vitamin E 3.6 mg per 2 capsules; dose a total of 6 daily) for 3 months, followed by a cross over where all groups continued with active supplementation for a further 3 months.

Overall, this study was essentially negative as there was no statistically significant difference between the active and placebo groups in the full sample of ADHD children and adolescents. However, there was a subgroup within the full sample who responded with a clinically significant improvement in ADHD key symptoms (a reduction of a least 25% on the ADHD-RS and a reduction of CGI scores to within near to the normal range). Among these were a small proportion of responders who demonstrated a 50% reduction in symptoms. Comparison of treatment effect size to the Oxford-Durham and Adelaide trials was not possible due to the differences in rating scales employed by this study. Patient samples were also rather different, with this sample containing more severe symptoms or at least all had a clinical diagnosis of ADHD, the majority of which also had complex comorbidities. Statistical power was also limited by the small sample size especially in the subgroups.

There is only one study to date by Harding and colleagues (2003) which has compared the effects of stimulant medication with dietary supplements and EFAs in children with ADHD. The results reported that both groups, that is (i) stimulant medication versus (ii) food supplements, yielded significant improvements in measures of attention and self-control. Furthermore, the results showed no significant difference in the level of improvement between each group; suggesting that food supplementation may be of equal efficacy as stimulant medication in reducing symptoms of ADHD (Harding, Judah, & Grant, 2003). However, it should be noted that this study had a small sample size (n = 20) and absence of a control group.

Design and Methodology Issues

To date, the data on the benefits of LC-PUFAs supplementation for ADHD have been criticised as both limited and fragmentary (Busch, 2007). This is largely due to differences in the method and design across studies. For example, important methodological variations include differences in dose, duration of the dose and the type of supplementation provided (i.e., the specific ratio of omega-3 to omega-6, and levels of EPA and/or DHA). This is important because some studies which have provided supplementation with omega-6 and/or DHA only to ADHD children have proved to be inconclusive supporting the different role and function of EPA and DHA (see Voigt, 2001; Hirayama et al., 2004). In addition, these methodological variations create substantial problems for cross-study comparisons and replicability. It is pertinent to note that the PUFA supplementation trials to date which have yielded statistically significant results, whether in a subgroup sample (as ADHD is not a heterogeneous condition) or the full cohort, have used a combination of EPA, DHA (omega-3) and GLA (omega-6) with vitamin E (which is a natural preservative and aids the synthesis of EFAs). There is much debate as to whether EPA or DHA is more effective but it seems indeed that it is a combination of omega-3 and 6 is more effective in alleviating symptoms and improving attention and behaviour problems. This observation also coincides with blood data which has found a low DHA/AA profile in both children and young adults with ADHD.

BLOOD ANALYSES OF EFAS IN DEPRESSION AND COMORBID DISORDERS/ADHD

Patients suffering from depression have shown deficiencies and/or lower levels of omega-3 fatty acids when blood samples have been taken and analysed. For example, Peet et al. (1998) found significantly decreased concentrations of omega-3 polyunsaturated fatty acids (PUFA) and docosahexaenoic acid (DHA) in the red blood cells (RBC) of 15 depressed patients in comparison to 15 healthy adults. RBC's are an ideal way to measure fatty acid composition in the membranes and provide potential indicators of EFAs status in the brain. High levels of fatty acids lead to an increase in the fluidity of membranes which in turn increases the transport of serotonin into the endothelial cells (Block & Edwards, 1987).

Table 1. Randomised controlled trials with ADHD and EFAs

Name of trial	Author & Journal	Type of Formula	Total fish oil	EPA	DHA	ω-6	Vitamins	Daily dose	Durati
Oxford-Durham (*N* = 117)	Richardson & Montgomery *Pediatrics* (2005)	natural triglyceride	400 mg	186 mg /2 capsules	58 mg	20 mg	3.6 mg	x 6	6 mo.
Adelaide (*N* = 132)	Sinn & Bryan *J Dev Behav Pediatr* (2007)	natural triglyceride	400 mg	186 mg /2 capsules	58 mg	20 mg	3.6 mg	x 6	6 mo.
BNK *(N* = 75)	Johnson, Östlund, Fransson, Kadesjö & Gillberg *J Attention Disorders* (2008)	natural triglyceride	400 mg	186 mg / 2 capsules	58 mg	20 mg	3.6 mg	x 6	6 mo.
DHA only (*N* = 63)	Voigt et al. *J Pediatr* (2001)	algae-derived triglyceride capsule	n/a	n/a	345 mg	n/a	n/a	x 3	4 mo.
DHA containing food (*N* = 40)	Hirayama et al. *Eur J Clin Nutr* (2004)	fortified foods (soybean milk/ bread rolls)	n/a	700 mg /week	3.6 g /week	n/a	n/a	n/a	2 mo.

Research in comorbid disorders such as ADHD have found depleted amounts of LC-PUFAs, specifically omega-3 fatty acids in the blood profiles of children and adults with ADHD and other behavioural disorders (Antalis, Stevens, Campbell, Pazdro, Ericson, & Burgess, 2006; Chen, Hsu, Hsu, Hwang, & Yang, 2004). However, the exact nature of this association is not yet clear and further research is necessary to establish whether this is due to a deficiency in EFAs, or an abnormality in metabolism due to genetic influences such as the delta 5 and delta 6 desaturase genes which enable the synthesis of DHA in the erythrocytes. The publications in this area are limited but the trends persistently demonstrate abnormal EFA profiles. Stevens, Zentall, Abate, Kuczek and Burgess (1996) reported that approximately 40% of children suffering from symptoms of ADHD showed thirst and skin problems and the proportion of arachidonic acid (AA) and DHA were significantly lower than matched controls. However, it was not evident that blood levels of EFAs observed in this sample were either indicative of a deficiency or confirmation that they consumed less dietary PUFAs than controls (Burgess et al., 2000). Researchers in Taiwan also examined the dietary patterns of children (aged 4-12) with ADHD and found lower LA, AA and DHA fatty acid compositions in red blood cells compared to controls (Chen, Hsu, Hwang & Yang, 2004).

Antalis and colleagues in the U.S. (2006) found that children with ADHD displayed classic signs of fatty acid deficiency such as skin problems and excessive thirst, supporting the earlier work of Colquhoun and Bundy (1981). This led them to concentrate on a potential metabolic insufficiency as a cause for the changed fatty acid phenotype. They did this by selecting a population of rigorously diagnosed young college-age population with ADHD, and compared them to a broad cross-section of young adults without a behaviour disorder. In addition comparisons were drawn between the blood fatty acid proportions, as well as blood markers, for a range of nutrients and conditions that might influence long-chain polyunsaturated fatty acid status and abnormal behaviour (Antalis, Stevens, Campbell, Pazdro, Ericson & Burgess, 2006).

In phase one of this study the regularity of thirst and the severity of skin problems were assessed with students with a diagnosis of ADHD. They were then compared to consenting controls from a general student population. Students filled out Conners Adult ADHD Rating Scales along with extensive dietary, health, demographic and thirst/skin symptoms. In phase two, biological evidence of essential fatty acid deficiency in ADHD was evaluated. Participants provided a blood and urine samples along with a 3 day diet record. A fatty acid analysis was carried out of plasma and red blood cells.

The statistical analysis showed no significant differences between the two groups in terms of age, gender, ethnicity, body mass index, blood pressure or self rated health scales. However, differences were observed in smoking, skin and thirst symptom scores between the ADHD participants and the control group. It is important to note, that oxidative stress which can be caused by smoking (and/or excessive alcohol consumption) rapidly depletes stores of highly unsaturated fatty acids (HUFA; Watts, 2008). In plasma, the ADHD group had a higher proportion of total saturated fatty acids and total monounsaturates compared to controls. In terms of PUFAs, no significant differences were found for most of the individual members of the omega-6 essential fatty acids in plasma, but total omega-6 proportions were about 6% higher in controls. Conversely, in the red blood cells (RBC), AA was the only omega-6 that differed between the 2 groups; it was approximately 10% higher in ADHD versus controls. For the omega-3 essential fatty acids, DHA was found to be lower in the ADHD group versus controls (53% less for plasma and 36% less for RBC). The same pattern

was observed for total omega-3 fatty acids. In plasma, the parent omega-3 fatty acid, alpha-linolenic acid, was found to be greater in the ADHD group whereas all of its metabolites were lower. The ratio of total omega-6 to omega-3 was higher in ADHD than controls for both RBC and plasma. Additionally in the ADHD group, the RBC ratio of AA to EPA was 36% higher compared to the controls. This association was also similar in plasma although the difference was not significant.

Associations between behaviour and RBC and plasma proportions of omega-3 fatty acids were tested by correlation. Significant inverse correlations were observed for the Conners Rating Scales and plasma proportions of the omega-3 fatty acid DHA. Similar patterns were found for total omega-3 fatty acids in both blood fractions, suggesting that lower omega-3 status was linked with greater severity of behavioural symptoms (Antalis et al., 2006).

Colter, Cutler and Meckling (2008) recently assessed fatty acid status and behavioural symptoms of attention deficit hyperactivity disorder in adolescents. The purpose of their study was to investigate differences in dietary intake of EFAs to determine whether this could provide insight into the abnormalities in red cell fatty acids that have been observed in the blood samples of young children found in previous studies. A secondary hypothesis sought to establish whether there were relationships between concentrations of EFAs and Conners Parent Rating Scales (CPRS-L). Twenty three participants were recruited for the study. Eleven of which were had a formal diagnosis of ADHD and 12 were healthy controls (i.e., those without ADHD). Six of the 11 ADHD group were taking medication; 5 of the 11 (55%) had a comorbid learning disorder (45%) and 8 of the 11 reported ADHD as existing in other family members (73%). Fatty acids were measured in red blood cell samples from both visits and presented as a percentage of molecular weight. Participants in the ADHD group were breastfed for an average period of 0-7 months compared to an average of 3-39 months. The fatty acid deficiency symptom check reported an average of 0-9 symptoms for the ADHD group versus 0-4 symptoms in the control group. The ADHD group also reported more allergies (27% versus 8%), none of these differences were statistically significant. However, analysis of the Conners' Rating Scales and EFAs showed significant differences between groups. The ADHD group had significantly higher mean scores on 10 of the 14 subscales for restlessness and impulsivity, cognitive problems and inattention, oppositional behaviours, hyperactivity, emotional and overall problematic behaviour. Specific components of diet such as total energy were positively correlated with oppositional and hyperactive behaviours. Saturated fat and total fat intakes revealed a significant positive correlation to scales measuring oppositional, problematic and hyperactive behaviours. Phospholipid analysis of red blood cells revealed that compared to controls, DHA (M = 3.12 $\pm$ 0.75 vs. M = 4.39 $\pm$ 1.34) and total omega-3 fatty acids (M = 5.79 $\pm$ 1.39 vs. M = 7.42 $\pm$ 1.64) were significantly lower in the ADHD group. Red blood cell DHA content was negatively correlated with subscales of the Parent Conners' such as oppositional behaviour, hyperactivity, cognitive problems, restlessness, problematic behaviour, DSM-IV inattention and DSM-IV total . Total red blood cell omega-6 content was positively correlated with problematic behaviour, oppositional, restlessness, DSM-IV inattentive, DSM-IV total and ADHD index scales.

The authors concluded that the ADHD adolescents in this sample had lower DHA levels, lower total omega-3 fatty acids and higher linoleic acid (omega-6: LA) levels compared to age-matched controls. They suggested that the lower levels of DHA may be due to a higher frequency of oxidation. The diet records did not reveal differences in total omega-3 fatty acid consumption or consumption of long-chain polyunsaturated fatty acids (LC-PUFAs).

Therefore, the differences found in the blood samples can not be attributed to diet. However, an important question remains whether supplementation of EFAs can normalize the effects found by this study. This study was limited by the small sample size and imbalance of genders within groups. Clearly, further research is required to establish whether high levels of omega-3 fatty acids can positively influence behavioural symptoms of ADHD in patients across age ranges (Colter, Cutler, & Meckling, 2008).

CONCLUSION

In light of the World Health Organisation predictions regarding the 50% rise in child mental disorders by 2020 (Global Health Forum for Research, 2008) it is important that risk factors are identified and the necessary modifications implemented as a matter of priority. The evidence presented in this chapter regarding the direct and indirect impact of omega-3 fatty acids to cognitive function, depression and behaviour warrants further investigation. The research to date that supports a link between EFAs and alleviation of symptoms of depression and ADHD originates from three main sources: (1) animal and human studies which have shown the behavioural effects of omega-3 deficiency including reduced cognitive ability; (2) studies with children and young adults which have reported reduced omega-3 fatty acids in the blood profiles of patients with ADHD and depression; and (3) randomised controlled trials which have indicated supplementation with omega-3 fatty acids in children with depression[1] and ADHD alleviated symptoms. The American Psychiatric Association supports the beneficial effects of PUFAs in treating adults with depression. However, further research is needed to establish whether EFAs could also be of benefit to children and adolescents with depression. This is important to establish as specific supplementation such as fatty acids and general dietary improvement may prove to be an inexpensive, acceptable and safe intervention.

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Chapter 5

DEPRESSION COMORBIDITY AMONG CHILDREN: POTENTIAL EXPLANATIONS, SHARED RISK FACTORS, AND DIRECTIONS FOR FUTURE RESEARCH

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ABSTRACT

Although childhood depression often co-occurs with other psychological conditions, there is little systematically organized knowledge as to how these co-occurring or comorbid conditions develop. This chapter presents several potential explanations for the co-occurrence of depression with other conditions, and illustrates these explanations using co-occurring depression and conduct problems. In the first section of the chapter, I present three potential explanations for co-occurring depression and conduct problems: (1) depression confers risk for conduct problems, (2) conduct problems confer risk for depression, and (3) shared risk factors account for their co-occurrence. In the second section, I describe a model that extends the shared risk factors explanation to include autonomic, prefrontal, and limbic system processes that may underlie co-occurring depression and conduct problems, and discuss how parent-child processes may interact with these child-specific factors to confer risk for this comorbid condition. In the final section, I identify several gaps in the literature and potential directions for future investigations involving depression comorbidity among children.

INTRODUCTION

Comorbidity, or the co-occurrence of psychological disorders, is more the rule than the exception among children affected by psychopathology (Angold, Costello, & Erkanli, 1999; Lilienfeld, 2003). Given that children with comorbid conditions are likely to experience

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greater symptom severity and persistence, more negative correlates and sequelae, and a more intractable course than children without comorbid conditions (e.g., Capaldi, 1992; Connor et al., 2003; Drabick, Gadow, & Sprafkin, 2006; Jensen, Martin, & Cantwell, 1997; Karlsson et al., 2006), this dearth of research evaluating co-occurring conditions is a crucial limitation and important direction for future research (Jensen, 2003; Lilienfeld, 2003).

Depression has been shown to co-occur with numerous psychological conditions in childhood and adolescence, including attention-deficit/hyperactivity disorder (ADHD; e.g., Biederman, Mick, & Faraone, 1998; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Cuffe et al., 2001; Gadow et al., 2004; Jensen et al., 1997); oppositional defiant disorder (ODD; e.g., Costello et al., 2003; Drabick, Gadow, & Loney, 2007; Gadow & Nolan, 2002; Maughan, Rowe, Messer, Goodman, & Meltzer, 2004); conduct disorder (CD; e.g., Fombonne, Wostear, Cooper, Harrington, & Rutter, 2001; Marmorstein & Iacono, 2004; Maughan et al., 2004; Sack, Beiser, Phillips, & Baker-Brown, 1993); several anxiety disorders (e.g., Beesdo et al., 2007; Franko et al., 2005; Karlsson et al., 2006; Lewinsohn, Holm-Denoma, Small, Seeley, & Joiner, 2008; Moffitt et al., 2007); and substance use and abuse (e.g., Franko et al., 2005; Karlsson et al., 2006; Latimer, Stone, Voight, Winters, & August, 2002; Zeitlin, 1999). Despite the frequent comorbidity of childhood and adolescent depression with other psychological conditions, there is little systematically organized knowledge as to how these co-occurring conditions develop. Understanding how the comorbidity of depression and other conditions develops could facilitate identification of children at risk for comorbid conditions, as well as inform prevention and intervention efforts.

Depression has been defined in varying ways in the literature based on the nature of the sample, assessment strategies, and developmental period considered. Three separate constructs typically are considered in investigations of depression among children and adolescents, including depressed mood, depressive syndromes (i.e., empirically derived syndromes that include depressive symptoms), and depressive disorders (i.e., syndromes consistent with *DSM-IV* diagnostic criteria; American Psychiatric Association, 1994), which could be construed in a hierarchical and sequential framework (Compas, Ey, & Grant, 1993). These constructs are similar in that they each include negative affectivity, but differ in their inclusion of other diagnostic symptoms (e.g., difficulty concentrating and sleeping, anhedonia), symptom severity, symptom frequency, and level of impairment (Compas et al., 1993). The constructs also differ in their prevalence, with depressed mood being the most frequent and depressive disorders being the least frequent. Last, these constructs differ in their developmental course; for example, depressed and irritable mood may be more salient in childhood, whereas symptoms consistent with the syndrome of major depressive disorder (MDD) are more likely to emerge in later childhood and adolescence. In terms of *DSM-IV* depressive disorders, epidemiological studies report prevalence rates ranging from 1.6%-4.3% (Angold et al., 1999; Costello et al., 2003), with rates increasing from childhood to adolescence and overall higher prevalence rates among girls than boys (e.g., 3-month prevalence rates: 2.8% vs. 1.6% among girls and boys, respectively; Costello et al., 2003). Given these shifts in prevalence rates across childhood and this potential sequential framework, along with a dearth of research evaluating depression comorbidity during childhood and early adolescence, I consider both dimensional and categorical characterizations of depression (e.g., depressed mood, MDD) in this chapter to provide a more thorough examination of depression comorbidity during these developmental periods.

Various authors (e.g., Angold et al., 1999; Caron & Rutter, 1991; Klein & Riso, 1993; Simonoff, 2000) have offered useful frameworks for conceptualizing the co-occurrence of psychological syndromes among children that provide potential explanations for comorbidity. In this chapter, I will focus on three of these possible explanations: (1) depression confers risk for additional psychological conditions, (2) other psychological conditions confer risk for depression, and (3) shared risk factors account for their co-occurrence. To illustrate the application of these explanations to depression comorbidity, I will provide evidence involving co-occurring depression and conduct problems (i.e., aggression, ODD, and CD). Examination of co-occurring depression and conduct problems has important clinical implications, given that children who exhibit comorbid depression and conduct problems are at increased risk for delinquency, substance abuse, violence, and suicide, compared to children with depression or conduct problems alone (Capaldi, 1992; Fombonne et al., 2001; Lansford et al., 2008; Miller-Johnson, Lochman, Coie, Terry, & Hyman, 1998; Rohde, Lewinsohn, & Seeley, 1991). Despite apparent dissimilarity at the symptom level, significant concurrent and prospective relations between depression and conduct problems have been demonstrated repeatedly. For example, in their meta-analytic review of general population studies, Angold et al. (1999) reported that the strength of association between depressive disorders and conduct problems (odds ratio) was 6.6 (95% CI = 4.4 to 11.0), and prospective relations between depression and conduct problems have been reported in both clinic-based (Geller, Chestnut, Miller, Price, & Yates, 1985; Kovacs, Paulauskas, Gatsonis, & Richards, 1988; Lahey, Loeber, Burke, Rathouz, & McBurnett, 2002) and community-based (Beyers & Loeber, 2003; Capaldi, 1992; Sack et al., 1993) samples. Given these elevated rates of co-occurrence and negative sequelae, further investigation of the developmental relations between depression and conduct problems is warranted.

In the first part of this chapter, I review and present evidence relevant to each of three potential explanations for co-occurring depression and conduct problems. This evidence generally is drawn from studies that concurrently considered depression and conduct problems. In the second part, I present an alternative, testable model for potential shared processes that may underlie co-occurring depression and conduct problems. I provide evidence for each component of the model, including autonomic, prefrontal, and parent-child processes, and in so doing, draw from a variety of sources, such as cognitive and behavioral neuroscience, child clinical and developmental psychology, and biological psychiatry. I then describe some implications of applying this model. In the third part of this chapter, I describe several remaining gaps in the literature and directions for future research.

POSSIBLE EXPLANATIONS FOR CO-OCCURRING DEPRESSION AND CONDUCT PROBLEMS

Although there are a variety of reasons that conduct problems and depression may co-occur, I will focus on three potential explanations for the co-occurrence of conduct problems and depression that have garnered support based on research to date: (1) depression symptoms confer risk for conduct problems, (2) conduct problems confer risk for depression, and (3) shared risk factors account for their co-occurrence (Angold et al., 1999; Caron &

Rutter, 1991; Klein & Riso, 1993; Simonoff, 2000). Evidence and potential models for each of these explanations are presented next.

Explanation 1: Depression Symptoms Confer Risk for Conduct Problems

The first explanation for co-occurring depression and conduct problems is that depression is a risk factor for conduct problems. One possible model for this relation is that depressed children are irritable and withdrawn at home, which leads to parent-child conflict (Sheeber, Hyman, & Davis, 2001). Such conflict and subsequent increased levels of depression may lead to problematic peer relationships (e.g., rejection, victimization) and withdrawn behavior at school (Rudolph & Clark, 2001). As part of their depression, children may have difficulty concentrating, which leads to poor academic performance. Over time, parents' hostile behaviors may be increasingly replaced with withdrawal from the child, as parents recognize that their behaviors do not serve to modify the child's behaviors. As these children age, peer difficulties and parental detachment persist, increasing the likelihood of associating with deviant peer groups (McGrath & Repetti, 2002).

Some research involving clinic-referred depressed children suggests that the onset of depression precedes the onset of conduct problems (Geller et al., 1985; Kovacs et al., 1988; Puig-Antich, 1982). Collectively, however, these studies are descriptive and do not suggest mechanisms through which depression may lead to conduct problems. Furthermore, these studies do not consider ODD symptoms, which are a common precursor to and may represent a prodromal form of CD (Lahey, Loeber, Quay, Frick, & Grimm, 1992; Moffitt, 1993a). Thus, the inability to determine whether early depressive symptoms (e.g., difficulty concentrating, social withdrawal), unmeasured ODD, or other mechanisms influenced the development of conduct problems in these studies limits the conclusions that can be drawn regarding how conduct problems may have developed in addition to depression over time (Loeber & Keenan, 1994; Rohde et al., 1991). Moreover, because support for this explanation has been limited primarily to clinical samples, it is not clear how useful this explanation will be for understanding conduct problems and depression in community-based samples in which comorbidity rates and symptom severity are likely to be lower.

Explanation 2: Conduct Problems Confer Risk for Depressive Symptoms

A second explanation is that conduct problems are a risk factor for depression. This ordering is consistent with the stage model proposed by Patterson and colleagues (1989), which indicates that children with conduct problems are more likely to fail in interpersonal relationships and academic settings (Capaldi, 1991, 1992; Patterson, DeBaryshe, & Ramsey, 1989). In this type of "failure" model, conduct problems interfere with an acquisition of social skills. These social skills deficits are exacerbated by conflictual interpersonal relationships, which lead to associating with deviant peers and depressed mood. Consistent with this explanation, several prospective studies using community-based samples of adolescents have suggested that conduct problems precede depression significantly more often than depression precedes conduct problems when they co-occur (Capaldi, 1992; Fleming, Boyle, & Offord, 1993; Sack et al., 1993). Furthermore, among clinic-referred boys, CD measured in childhood

predicted later depression even when initial levels of depression were controlled, but depression did not predict later CD (Lahey et al., 2002). Thus, the findings of these studies lend support to the explanation that conduct problems precede depression, though this pattern may be evidenced earlier in clinic-based (Lahey et al., 2002) than community-based (Capaldi, 1992; Fleming et al., 1993) samples. Nevertheless, a general reliance on adolescent samples limits our understanding of how facets of this comorbid condition unfold during childhood, when the mechanisms and symptoms may be more amenable to intervention. In addition, the ability of these studies to account for the comorbidity of conduct problems and depression is limited by a failure to test the shared risk factors explanation for comorbidity (Explanation 3, presented next) concurrently with the explanations that one disorder is a risk factor for the other (Explanations 1 and 2).

Explanation 3: Shared Risk Factors Account for Co-Occurring Conduct Problems and Depression

A third possibility involves the shared risk factors explanation for comorbidity, which suggests that depression and conduct problems are associated with overlapping and unique factors and that comorbidity stems from shared risk factors (Angold et al., 1999; Caron & Rutter, 1991; Klein & Riso, 1993). At the psychological level of analysis, shared risk factors for conduct problems and depression involve at least seven domains: (1) child emotion dysregulation and emotional lability (Beauchaine, 2001; Snyder, Schrepferman, & St. Peter, 1997); (2) child attention problems (Bird, Gould, & Staghezza, 1993; Drabick, Beauchaine, Gadow, Carlson, & Bromet, 2006; Treuting & Hinshaw, 2001); (3) low levels of parent-child communication and parental detachment (Drabick, Gadow, et al., 2006; Fergusson, Lynskey, & Horwood, 1996; Ge, Best, Conger, & Simons, 1996); (4) ineffective and coercive discipline (Compton, Snyder, Schrepferman, Bank, & Shortt, 2003; Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998; Patterson et al., 1989); (5) parental psychological problems, including maternal depression and marital discord (Fendrich, Warner, & Weissman, 1990; Loeber et al., 1998); (6) peer relationship problems (Capaldi, 1992; Fergusson et al., 1996; Keiley, Lofthouse, Bates, Dodge, & Pettit, 2003; Rudolph & Clark, 2001); and (7) poor academic performance (Capaldi, 1992; Loeber et al., 1998; Velez, Johnson, & Cohen, 1989).

Unfortunately, the usefulness of these purported shared risk factors for understanding co-occurring depression and conduct problems is limited. First, these risk factors are not specific to co-occurring depression and conduct problems, which limits their utility for etiological and intervention models for this comorbid condition (Steinberg & Avenevoli, 2000). Second, presuming these are "shared" risk factors is arbitrary as these factors have not been tested effectively in other viable roles, such as mediators or moderators, for co-occurring depression and conduct problems. Indeed, many of these same risk factors are included in Explanations 1 and 2 above as the mechanisms that facilitate development of the comorbid condition (e.g., conduct problems lead to academic and interpersonal difficulties, which may lead to depression). Thus, it is plausible that these factors each play a role in the development of the comorbid condition, but their effects differ depending on when they are experienced. Third, these risk factors are likely inter-related, which restricts the ability to predict risk from these

factors individually (Deater-Deckard, Dodge, Bates, & Pettit, 1998; Greenberg, Speltz, DeKlyen, & Jones, 2001).

It is furthermore likely that there are reciprocal and transactional influences among these risk factors. Using a developmental psychopathology perspective, a model for the development of depression and conduct problems that ties these various domains together could be posited. For instance, the ability to regulate emotions, attend to demands, and inhibit impulsive responding affects how well children tolerate frustration and negotiate interpersonal interactions (Bagwell, Molina, Pelham, & Hoza, 2001; Shaw, Owens, Giovannelli, & Winslow, 2001). Thus, a child who is emotionally dysregulated (Domain 1) and/or exhibits attention problems (Domain 2) may be less responsive to or compliant with parental demands, which may lead parents to spend increased amounts of time addressing discipline issues. Over time, these interactions may lead to parent-child relationships that are characterized by parental detachment and poor parent-child communication (Domain 3). Concurrently, because of the potentially demanding and time-intensive nature of parenting an emotionally dysregulated and inattentive/impulsive child, parents may be more likely to engage in inconsistent and coercive discipline (Domain 4). In response to such discipline, children may use aggressive and depressive behaviors to deflect family members' behaviors and facilitate their removal from aversive interactions (Patterson, 1982). Coercive parent-child interchanges are particularly likely if parents are experiencing their own psychological problems, parenting disagreements, or marital difficulties (Domain 5).

In school, an emotionally dysregulated and/or inattentive child may have problematic peer relationships and experience peer rejection and/or victimization (Domain 7), which ultimately may lead to associating with deviant peers. Last, with increasing academic demands as children age, problems with inattention and emotion dysregulation are likely to lead to poor academic performance (Domain 8). Thus, earlier difficulties may be precursors for later risk factors (e.g., childhood attention problems may be related to preadolescent academic problems; preadolescent peer rejection may be related to adolescent deviant peer involvement). Furthermore, the severity and accumulation of risk over time will impact when children exhibit conduct problems and depression, and suggest that concurrent consideration of multiple domains may better capture the processes that place children at risk for the development of comorbid depression and conduct problems (Deater-Deckard et al., 1998).

AN ALTERNATIVE MODEL FOR SHARED PROCESSES

Sharing common processes suggests a common underlying disease entity, and would be indicated when the term comorbidity is applied in a medical disease model (Lilienfeld, Waldman, & Israel, 1994). However, because the etiological mechanisms responsible for psychological disorders have not been described completely, this definition of comorbidity is difficult to apply to psychological syndromes. As long as these syndromes are defined by behavioral symptoms without reference to biological underpinnings, it is possible that comorbidity may be a function of shared underlying processes that are typically unmeasured. Similarly, the risk factors reviewed above generally rely on observable symptoms, which may not effectively capture the difficulties with behavioral inhibition and regulating negative emotions that often are associated with co-occurring depression and conduct problems.

Multiple levels of analysis, including assessments of autonomic and prefrontal processes, may be more useful in this regard. One possible model, which can be viewed as an extension of the shared risk factors explanation, links autonomic, prefrontal, limbic, and parent-child processes and provides an alternative conceptualization of the processes that may underlie co-occurring depression and conduct problems. Evidence for each component of this model is presented next.

Autonomic Processes

Given that both depression and conduct problems are associated with dysregulation of the autonomic nervous system (ANS), examination of autonomic physiology could inform models of risk for depression and conduct problems. Individuals with depression exhibit elevated resting heart rate, low heart rate variability, and exaggerated heart rate responses to stressors, which also may account for the well-established relation between depression and cardiac events (Carney, Freedland, & Veith, 2005). Although patterns of cardiac activity differ in some ways among individuals with depression or conduct problems only (Lorber, 2004), a more nuanced consideration of ANS influences on cardiac functioning suggests important similarities among ANS processes, depression, and conduct problems.

The myocardium of the heart is innervated by both the sympathetic (SNS) and parasympathetic (PNS) nervous systems. The effects of SNS and PNS influences on cardiac activity are antagonistic; thus, heart rate is determined largely by the dynamic interaction of acceleratory SNS activation and deceleratory PNS activation (Berntson et al., 1994). The deceleratory parasympathetic component is provided by the vagus, or tenth cranial nerve. Although heart rate is controlled by both the SNS and PNS, the PNS typically exerts a stronger influence on heart rate than the SNS given its association with the vagus nerve (Porges, 2003). Parasympathetic influence on cardiac activity, often referred to as vagal tone, is indexed by assessing respiratory sinus arrhythmia (RSA) or the degree of ebbing and flowing of heart rate during the respiratory cycle (Berntson et al., 1997). Reduced parasympathetic activity has been reported among aggressive, depressed, and anxious individuals (Beauchaine, 2001; Beauchaine, Gatzke-Kopp, & Mead, 2007; Boyce et al., 2001; Crowell et al., 2006; Forbes, Fox, Cohn, Galles, & Kovacs, 2006). Although these disorders appear quite different at the symptom level, all are characterized by poor attentional control and dysregulated negative affect, which suggests that reduced parasympathetic activity may be a marker for problems with attention and regulation of negative emotions. Furthermore, both internalizing and externalizing symptoms are associated with reduced PNS-linked cardiac activity, and boys with co-occurring internalizing and externalizing symptoms exhibit even lower heart period variability than boys with only internalizing or externalizing symptoms (Pine et al., 1998). Thus, because cardiac vagal tone is related to the ability to self-regulate via modulation of attention and emotion (Forbes et al., 2006; Porges, 2003; Thayer & Lane, 2000), it is not surprising that psychological problems that involve difficulties with attention and regulation of negative emotions are associated with decreased parasympathetic tone. Difficulties with attention and regulation of negative emotions also are associated with dysregulated prefrontal cortical and limbic system processes, considered next.

Prefrontal Cortical and Limbic System Processes

Several lines of evidence indicate that dysfunction in the prefrontal cortex (PFC) and amygdala is related to depression and conduct problems. In reviewing evidence related to the PFC, I use two relatively superordinate terms: the orbitofrontal cortex (OFC), which subsumes the ventromedial prefrontal cortex; and the dorsolateral prefrontal cortex (DLPFC), which has been implicated in executive functioning abilities (Adolphs, 2001; Drevets, 2001; Ochsner, 2004; Rolls, 2004; Tranel, Bechara, & Damasio, 2000). I also consider the amygdala and limbic system, which have extensive reciprocal connections with the OFC and PFC more generally (Drevets, 2001; Pears, Parkinson, Hopewell, Everitt, & Roberts, 2003). Each of these areas may be particularly important to consider during childhood when connections between the amygdala and PFC, and myelination of the PFC, are still developing (Derryberry & Rothbart, 1997; Raine, 2002).

In terms of depression, electrical stimulation of the amygdala produces several symptoms evidenced with MDD, such as dysphoria and recollection of emotionally provocative events (Drevets, 1999), and therapy effects are associated with changes in the limbic system among individuals with MDD (Ebert & Ebmeier, 1996). With regard to the PFC, the volume of the OFC is smaller among individuals with a history of MDD than controls (Bremner et al., 2002), and there are abnormal elevations in resting blood flow and glucose metabolism in the amygdala, OFC, and DLPFC among depressed individuals (Dolan, Bench, Brown, Scott, & Frackowiak, 1994; Drevets, 1999, 2001; Ebert & Ebmeier, 1996; Kaufman & Charney, 2001; Must et al., 2006). This over-activation of the PFC impacts the ability of the PFC to modulate amygdala activity (Drevets, 1999, 2001; Must et al., 2006). When given negative feedback, however, individuals with depression exhibit a significant attenuation of OFC activity (Rose, Simonotto, & Ebmeier, 2006), suggesting that OFC activity varies depending on the valence of the stimulus. In addition, individuals with depression exhibit deficits in executive functioning, including attention, concentration, planning, and inhibition of prepotent responses (Beblo, Baumann, Bogerts, Wallesch, & Herrmann, 1999; Halperin & Schulz, 2006; Veiel, 1997, but cf. Rose et al., 2006), which have been linked to the DLPFC (Adolphs, 2001; Ochsner, 2004). Last, individuals with MDD exhibit impaired performance on gambling tasks (i.e., choose cards with greater immediate rewards but greater losses over time), which also suggests OFC dysfunction (Must et al., 2006).

In terms of conduct problems, individuals with a history of violence, aggression, and antisocial behavior exhibit structural and functional abnormalities in the PFC (Raine, 1997, 2002). In addition, as with depression, individuals with conduct problems exhibit executive functioning deficits (Halperin & Schulz, 2006; Lynam & Henry, 2001; Moffitt, 1993b). Another line of evidence supporting amygdala and PFC dysfunction among individuals with conduct problems stems from research involving psychopathic traits. Individuals with psychopathic traits have difficulty processing sad and fearful expressions, which is consistent with amygdala dysfunction (Blair, Colledge, & Mitchell, 2001; Blair, Morris, Frith, Perrett, & Dolan, 1999). They also make more disadvantageous choices on gambling tasks and perform poorly on response reversal tasks, which involve learning to respond to the opposite, previously irrelevant stimulus in a stimuli pair (Blair et al., 2001; Budhani & Blair, 2005; Cools, Clark, Owen, & Robbins, 2002). Performance on both gambling and response reversal tasks is associated with the OFC (Cools et al., 2002; Must et al., 2006).

Given that OFC dysfunction is associated with both depression and conduct problems, an alternative way to consider these relations is to examine models based on research involving individuals with known OFC deficits. A likely candidate is the somatic marker hypothesis (Damasio, Everitt, & Bishop, 1996; Tranel et al., 2000), which was developed to account for the finding that individuals with ventromedial prefrontal (OFC) deficits have difficulty linking emotions to behavioral consequences. This model posits that there are patterns of complex situations and associated somatic states in the ventromedial prefrontal cortex that are developed through associations between stimuli and somatic states. These patterns can be reactivated in new situations to inform reasoning and decision making and can mark potential outcomes as good or bad. Evaluation of the somatic marker hypothesis indicates that in affective decision-making (e.g., gambling) tasks, individuals with ventromedial lesions make disadvantageous responses over time, exhibit significantly less electrodermal (somatic) activity prior to making a disadvantageous choice, and appear to be insensitive to future outcomes. Thus, individuals with ventromedial deficits do not experience a "somatic marker" that would facilitate reassessing a situation before acting. Absence of this physiological response may increase the likelihood of engaging in risky behavior. Findings that humans with OFC lesions perseverate in behaviors that are no longer reinforced and have difficulty shifting cognitive strategies are consistent with the somatic marker hypothesis (Drevets, 1999, 2001).

The autonomic, prefrontal, and limbic system processes presented thus far are hypothesized to influence one another to confer risk for depression and conduct problems. For example, autonomic deficiencies likely limit the individual's ability to perform complex cognitive operations. This possibility is consistent with Polyvagal Theory, which proposes that an individual's physiological state restricts his or her range of behaviors and psychological experiences (Porges, 2003). Moreover, it is supported by evidence that men with low heart rate variability perform less quickly and obtain fewer correct responses on tasks of working memory and executive functioning than men with high heart rate variability (Hansen, Johnsen, & Thayer, 2003). There is anatomical support for this possibility as well. For instance, the vagus includes both efferent and afferent fibers, so neural traffic through it is bidirectional. Consistent with the somatic marker hypothesis, we would expect afferent pathways from viscera to specific brain structures and the PFC, which would serve to interpret afferent feedback and mark potential outcomes as good or bad (Porges, 2003; Thayer & Lane, 2000; Tranel et al., 2000). Given its association with the perception and production of emotions, the amygdala may play a role in linking autonomic and prefrontal processes. Specifically, the central amygdala nucleus innervates regions involved in autonomic response to stressors, including the dorsal motor nucleus of the vagus, and also has close anatomical connections with the prefrontal cortex, which modulates the response of the central amygdala to stressors (Grace, 2007). Prefrontal deficits attenuate the ability of the prefrontal cortex to down-regulate the effects of the amygdala, which may result in dysregulated or inappropriate behavioral responses. Thus, afferent and efferent pathways involving the autonomic nervous system, amygdala and other limbic structures, and prefrontal cortex could provide the anatomical framework for the dysregulation of negative emotions and difficulty in planning and inhibiting responses associated with co-occurring depression and conduct problems. It is furthermore likely that various contextual factors moderate these autonomic and prefrontal processes to confer risk. However, given that prefrontal and limbic system processes are developing continually throughout childhood, parent-child interactions represent critical and

proximal factors to consider in this regard. In the next section, I consider relations among parent-child interactions, depression, and conduct problems, and tie parent-child processes to the child-specific processes presented in this section.

Parent-Child Interactional Processes

Each of the three explanations for comorbidity presented suggests a role for interpersonal relationships in the development of co-occurring depression and conduct problems. Given the extensive literature examining relations among parent-child processes, conduct problems, and depression (e.g., Capaldi, 1991, 1992; Compton et al., 2003; Drabick, Beauchaine, et al., 2006; Drabick, Gadow, et al., 2006; Fergusson et al., 1996; Ge et al., 1996; Loeber et al., 1998; Patterson et al., 1989), the following section focuses on several processes that have been shown to exert a proximal influence on conduct problems, depression, and child emotion regulation. Difficulties with child emotion regulation are included because these difficulties are associated with the autonomic, prefrontal, and limbic processes presented above, and thus have direct relevance to the alternative model of shared processes for depression and conduct problems.

One model that articulates processes by which parent-child interchanges may be associated with conduct problems and depression is Coercion Theory (Patterson, 1982). Coercion Theory indicates that many children react to aversive family interactions with either (a) aggressive, hostile behaviors, or (b) depressive, withdrawn behaviors, both of which gain attention and deflect aversive behavior from family members (Compton et al., 2003; Patterson, 1982). Thus, repeated coercive parent-child interactions may increase the likelihood that children will exhibit conduct problems and depression. In addition to their impact on conduct problems and depression, parent-child processes also affect children's emotion regulation strategies. Moreover, alternative mechanisms (e.g., modeling) may account for associations among parent-child interactions, depression, and conduct problems.

From an early age, children learn emotion regulation through modeling, parental responsivity, and parental socialization (Davidov & Grusec, 2006; Frick & Morris, 2004; Scaramella & Leve, 2004). Children model other parental behaviors as well; for example, in terms of depressive symptoms, children may model some of their parents' withdrawn and irritable behavior (Compton et al., 2003; O'Leary & Vidair, 2005). However, depressive symptoms also may be a reaction to familial conflict, which may lead children to feel sad, hopeless, or guilty, all of which would be consistent with depression. Children's development of externalizing problems also likely involves modeling of parents' displays of negative affect and verbal and physical aggression, whether these behaviors are directed toward them or other family members (O'Leary & Vidair, 2005). Moreover, child negative reactivity or irritability may make it more difficult for children to learn emotion regulation, which in turn could elicit harsher parenting (Scaramella & Leve, 2004). These transactional processes have been supported by findings from observed family interactions. Sequential analyses indicate that children are more likely to exhibit anger as their parents' insensitive and negative responses toward them accumulate, and are less likely to become angry if their parents are able to modulate their own negative affect (Snyder, Stoolmiller, Wilson, & Yamamoto, 2003). In sum, simply viewing "emotion dysregulation" as a shared risk factor for depression and conduct problems does not capture the dyadic processes underlying this dysregulation.

Indeed, reciprocal relations among parent and child behaviors and emotions ultimately influence the child's ability to regulate negative affect.

Taken together with the autonomic and prefrontal processes presented above, we can posit a path to depression and conduct problems that begins with autonomic and/or prefrontal deficits, which may be manifested as difficulties with attention and/or regulation of negative emotions. With repeated coercive parent-child interactions, the child may perceive his or her environment as unsafe. Furthermore, if the prefrontal cortex cannot effectively modulate the amygdala in response to these contextual stressors, the child may be more likely to engage in aggressive or disinhibited behavior (Porges, 2003). The parent may become angry and view the child's behavior as volitional rather than constitutional, and in turn become critical and insensitive. Thus, underlying autonomic and prefrontal deficiencies may set the stage for conduct problems and depression, but it is social relational processes that may determine whether these symptoms are amplified. Although this model may provide a useful alternative framework for considering shared processes for conduct problems and depression, it remains to be tested.

Implications of Applying this Framework

Concurrent examination of autonomic and prefrontal processes may aid in identification of subtypes of individuals with depression and conduct problems that have distinct etiologies or patterns of functioning, and also could provide an index of treatment response. For instance, autonomic markers that were presumed to be stable indices of a predisposition for conduct problems have been found to be malleable among young children (e.g., Raine et al., 2001). However, over time, these paths may be more resistant to change. When coupled with impoverished circumstances or other contextual risk factors, one possible direction is to attempt to identify these patterns early and intervene prior to the expression of psychological problems. Because biological risk factors can be identified at a very young age, distinct subgroups of children, defined using biological markers, could be followed prospectively to evaluate what environmental factors serve to increase risk or protection of children from later psychological problems and whether there are sensitive periods in the development of pathological traits (Beauchaine, 2003). For instance, exposure to marital hostility at age 5 among children with low RSA, but not high RSA, predicts externalizing behavior 3 years later (Gottman & Katz, 1995), suggesting that there are indeed meaningful biological-contextual interactions that predict risk. Moreover, given that childhood is a critical time for the development of connections between the amygdala and PFC, and myelination of the PFC (Derryberry & Rothbart, 1997; Raine, 2002), examination of these processes during childhood could inform prevention and early intervention efforts aimed at decreasing the development of co-occurring depression and conduct problems.

GAPS IN THE LITERATURE AND DIRECTIONS FOR FUTURE RESEARCH

There are a number of critical gaps in the current literature that should be addressed in future research. Here I will consider three that are particularly relevant to the aims of the

present chapter: (a) concurrent evaluation of multiple comorbidity explanations, (b) consideration of sex differences, and (c) examination of child × context interactions. Each of these gaps is presented with suggestions for future research that could address these limitations in our understanding of co-occurring depression and conduct problems.

Concurrent Consideration of Multiple Explanations

Given that support for each of the comorbidity explanations is clearly mixed, future research would do well to consider multiple explanations of comorbidity concurrently to determine under which circumstances each explanation applies. Alternatively stated, studies cannot definitively identify which explanation best accounts for the co-occurrence of depression and conduct problems, because doing so requires examination of multiple explanations of comorbidity over time within the same sample. To my knowledge, only two studies have sought to evaluate both longitudinal relations and shared risk factors for conduct problems and depression simultaneously (Beyers & Loeber, 2003; Fergusson et al., 1996), and these studies differed in their conclusions regarding symptom co-occurrence. Beyers and Loeber (2003) found that the relation between conduct problems and depressed mood in a community-based sample of adolescent boys was *not* attributable to social-contextual risk factors, whereas Fergusson et al. (1996) reported that shared risk factors accounted for the relation between conduct problems and depressive symptoms in a community-based sample of adolescent boys and girls.

One possible reason for the mixed support within and across studies is that studies of comorbid depression and conduct problems generally have used multivariate statistical techniques that collapse information in such a way as to obscure unique developmental trajectories. A critical shift in testing how depression and conduct problems arise is from asking which explanation best accounts for comorbid conduct problems and depressive symptoms to asking which explanation best applies to each individual (a person-centered approach). Although it is recognized that numerous risk factors can result in the same outcomes (equifinality; Cicchetti & Rogosch, 1996), the multivariate modeling strategies that are typically employed may not be useful for identifying multiple pathways and homogeneous subgroups (Greenberg et al., 2001). Using both person- and variable-centered analyses would permit an evaluation of the circumstances in which each explanation might apply. Specifically, variable-based approaches allow examination of how risk factors influence the development of depression and conduct problems over time (Bates, 2000), whereas person-centered approaches examine risk factors at the level of the individual and evaluate individual trajectories across time (Magnusson, 1998). By integrating these methods, one can consider the formation of classes of people who are relatively homogeneous with regard to depression and conduct problem trajectories (person-centered), and relate variables to these classes to determine what risk factors and outcomes are associated with each class (variable-centered). A combination of these strategies has been used and provided unique and complementary information in examinations of early-onset conduct problems (Greenberg et al., 2001) and adolescent substance use (Muthén & Muthén, 2000).

Sex Differences

A major limitation for understanding co-occurring depression and conduct problems is that many studies to date have included only boys or have neglected comparisons of boys and girls (e.g., Capaldi, 1991; Drabick, Gadow, et al., 2006; Lahey et al., 2002; Loeber et al., 1998). Research that has examined these symptoms among boys and girls reveals several notable findings. First, the long-term sequelae for depression and conduct problems, such as substance abuse and suicide, occur among both girls and boys (Franko et al., 2005; Marmorstein & Iacono, 2001; Wannan & Fombonne, 1998). Moreover, girls with co-occurring depression and conduct problems are also at increased risk for anxiety, low school attainment, and early child-bearing and pregnancy (Bardone, Moffitt, Caspi, & Dickson, 1996). Second, in terms of prevalence rates, depression is more common among girls, whereas conduct problems are more common among boys (Costello et al., 2003; Velez et al., 1989). However, the difference in rates of depression does not typically emerge until late childhood (Merikangas & Angst, 1995). Third, with regard to symptom co-occurrence, comorbid depression and conduct problems are more likely to occur in preadolescence among boys and adolescence among girls (Zoccolillo, 1992), though the strength of the association may be stronger among girls than boys (Costello et al., 2003). Fourth, the course of this comorbid condition likely differs among boys and girls in that conduct problems tend to precede depression among boys, whereas conduct problems and depression exhibit bidirectional effects among girls (Wiesner, 2003).

Little is known about whether risk factors for comorbid depression and conduct problems differ by sex. Distinct risk factors for each sex or different levels of or sensitivities to the same risk factors may explain varying rates of depression and conduct problems across sex (Rutter, Caspi, & Moffitt, 2003). Coercive family interactions are related to conduct problems for both boys and girls; however, coercive family environments are related to girls', but not boys', depression, suggesting that family factors may differentially confer risk for depression among girls (Compton et al., 2003; Drabick, Beauchaine, et al., 2006). This possibility is consistent with evidence that girls are more susceptible to psychological problems in the presence of problematic interpersonal relationships than boys, as girls focus more on relationships and demonstrate higher levels of empathy (Rosenfield, Vertefuille, & McAlpine, 2000). Second, boys are more likely than girls to exhibit attention problems (Gaub & Carlson, 1997) and to experience peer victimization (Camodeca, Goossens, Terwogt, & Schuengel, 2002), both of which are associated with conduct problems and depression (Snyder, Brooker, et al., 2003; Treuting & Hinshaw, 2001). Thus, different levels of these factors may confer differential risk among boys and girls for this comorbid condition. Future research will need to attend to sex differences and explore whether different explanations are relevant among boys and girls, as well as whether risk factors operate differently by sex.

Child × Context Interactions

Simply identifying risk factors that may be related to depression and conduct problems is likely to have little prognostic value given the reciprocal and transactional relations among children and their contexts. In addition, contextual factors confer non-specific risk for various types of psychopathology; thus, consideration of contextual factors independent of other risk

processes is not likely to improve our understanding of the development of co-occurring depression and conduct problems. To understand why contextual factors lead to negative outcomes among some, but not all, children, examining child-specific factors concurrently with contextual factors can be useful (Steinberg & Avenevoli, 2000). The alternative model for shared processes that I have presented represents one such effort to link child and contextual risk processes, though other important candidate processes could be considered in research involving depression comorbidity.

Several studies have considered child (e.g., temperament) and contextual (e.g., parenting) factors concurrently in predicting child psychological adjustment. For example, in terms of externalizing problems, Bates, Pettit, Dodge, and Ridge (1998) reported that the interaction of child impulsive/unmanageable temperament and parental restrictive control predicted externalizing symptoms. Similarly, Colder, Lochman, and Wells (1997) demonstrated that poorly monitored active boys and fearful boys who experienced harsh parental discipline exhibited high levels of aggression. In a sample of preadolescents, Oldehinkel, Veenstra, Ormel, De Winter, and Verhulst (2006) demonstrated that high frustration interacted with parental overprotection and lack of parental warmth to predict depressive symptoms. Furthermore, a noteworthy sex difference emerged: fearfulness increased the effect of parental rejection on depressive symptoms among girls, but not boys (Oldehinkel et al., 2006). In another example involving temperament and internalizing symptoms, Colder et al. (1997) reported that boys with fearful temperaments whose parents used harsh discipline or were overinvolved exhibited elevated levels of depressive symptoms. Taken together, these findings suggest meaningful child × context interactions that can improve our understanding of risk for depression and conduct problems, as well as other psychological conditions. However, there is still a dearth of literature addressing the additive and interactive effects of child and contextual factors, a limitation that can be addressed in future research. Furthermore, given that additional processes likely confer risk for, exacerbate, or maintain depression and comorbid conditions, future research would do well to consider additional potential shared processes, such as maternal depression (Fendrich et al., 1990; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997), family discord (Drabick, Gadow, et al., 2006; Loeber et al., 1998), peer processes (Deater-Deckard, 2001; Rudolph & Clark, 2001), and neighborhood influences (Evans & English, 2002; Leventhal & Brooks-Gunn, 2000). Last, future research involving depression comorbidity should consider the applicability of these various comorbidity explanations and potential risk processes to other co-occurring conditions (e.g., depression and co-occurring anxiety disorders).

CONCLUSION

Numerous authors have offered useful frameworks for conceptualizing the co-occurrence of psychological syndromes among children that provide potential explanations for comorbidity (e.g., Angold et al., 1999; Caron & Rutter, 1991; Klein & Riso, 1993; Simonoff, 2000). Three explanations that can be specified from these frameworks have received some support when applied to co-occurring depression and conduct problems: (1) depression is a risk factor for conduct problems, (2) conduct problems are a risk factor for depression, and (3) shared risk factors account for their co-occurrence. Identifying the correct explanation

requires concurrent examination of each alternative across time within the same sample, an important but potentially daunting goal for future research. However, because co-occurring conduct problems and depression confer significant risk for suicide, violence, delinquency, and substance use, it is important to understand how this comorbid condition develops.

In addition to concurrent consideration of multiple explanations, future research must consider sex differences and the transactional relations between child and contextual processes. In this chapter, I presented a model that links several child-specific and parent-child processes and thus attempts to address this gap in the literature. Specifically, I extended the shared risk factors explanation to include additional child (e.g., autonomic, prefrontal) and contextual (e.g., parent-child) processes as shared risk factors for depression and conduct problems. This alternative model also considers multiple levels of analysis and describes alternative factors that may confer more proximal risk for depression and conduct problems. Future research will be necessary to evaluate whether these processes are associated with additional conditions that co-occur with depression, and consequently address whether these processes are specific to co-occurring depression and conduct problems or associated with depression and co-occurring conditions more generally. Ultimately, tests of these hypotheses can lead research closer to recognizing etiological mechanisms, shared processes, differential distal outcomes, and homogeneous subgroups within depression and co-occurring conditions that may have different courses and responses to intervention. Furthermore, this work can inform efforts to identify and intervene with children at risk for developing depression comorbidity. Such efforts could consequently limit the negative sequelae associated with these conditions and promote positive development.

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Chapter 6

CONTROVERSIES IN CHILDHOOD AND ADOLESCENT DEPRESSION

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ABSTRACT

Major depressive disorder affects up to 10% of adolescents and is associated with substantial short-and long-term morbidity and mortality.

Depressive disorders are difficult to diagnose in children, because the symptoms are non-specific and due to the process of change at this stage of life. The picture varies enormously depending on the child's developmental stage.

This disorder affects the normal development of a child, school performance, social behavior and family relationships. Major depression in childhood or adolescence increases the risk of affective disorder in adulthood. The precise nature and course of the subsequent disorder remain unclear.

There is very little evidence for treatment in children. Most treatments are based on clinical experience or extrapolation from results of studies in adults. The increase over the last few years in the amount of available clinical research on the use of antidepressants to treat major depression in children and adolescents has substantially improved our knowledge of the safety and efficacy of these medications in the pediatric population. Many questions remain, however, that highlight the need to continue research in this patient population rather than relying on the extrapolation of data from trials involving adults.

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INTRODUCTION

At some time in their life 15-20 % of children and adolescents have some symptoms of depression. The incidence increases with age, especially after puberty when it is significantly more predominant in females [1, 2]. Up to 10% of adolescents between the age of 12 and 16 years may have depressive symptoms [3].

Depression in children and adolescents creates a significant clinical and social burden, both to the individuals as well as families and society. It involves an increase in the use of healthcare services, school services and other social services to which should also be added the cost of interventions for prevention and treatment of the disease [4]. It often becomes chronic and tends to persist into adulthood. It negatively impacts growth and development, school performance, and social or family relationships.

Frequently depression goes unnoticed, therefore it is not diagnosed and remains untreated. Reasons for this could be the stigma attached to these disorders, atypical presentation, reluctance to seek medical attention at this age and the lack of professionals capable of appropriately managing these patients [1].

EPIDEMIOLOGY

Prevalence studies have important limitations due to the heterogeneity of the samples studied and the different diagnostic tools used. Prevalences of 2% in children and 4-10% in adolescents have been reported. Incidence increases with age and after puberty it is twice as frequent in girls. This may be due to changes in life style or hormonal changes that occur at this age. However, boys have earlier, more severe depressive symptoms, which are difficult to diagnose, and are often associated with behavior disorders [5], whereas girls' symptoms tend to be associated with anxiety and eating disorders.

Adolescents with depressive symptoms are more likely to come from dysfunctional families, whereas having a greater number of siblings appears to be a protective factor [3].

Several risk factors have been associated with depression in childhood and adolescence:

- Hereditary factors: depression in parents has been linked with an increased risk of their offspring developing depression and with an earlier onset and poorer prognosis [6].

However, in a meta-analysis performed by McLeod et al. [7] aimed at determining the association between parental and childhood depression, it was demonstrated that parental depression accounted for less than 8% of the variance in childhood depression. The authors admit that even if parental depression plays a small part in childhood depression on average, it could have a potentially catalytic effect on the subgroup of children with vulnerability to developing depression for other reasons. Amongst the different subdimensions of parenting, parental hostility toward the child was that most strongly related to childhood depression.

- Endocrine factors: such as an increase in cortisol and a decrease in growth hormone.
- Neurological factors: neurotransmitter deficit.

- Other biomedical factors: chronic illness, female gender, hormonal changes during puberty, use of certain medicines (such as isotretinoin), sleep disorders …

Before puberty, major depression occurs with approximately the same frequency in both sexes. However, as already pointed out, after reaching adolescence depression is twice as frequent in females. Furthermore, as girls enter adulthood they have a greater risk of early relapse after the remission of an episode of clinical depression, have more severe depressive episodes, experience greater functional deterioration and develop a more chronic course of depression than men do. This increase in depressive symptoms in puberty is not surprising, because at this time of life adolescents are under pressure to form an identity, their sexuality emerges, they begin to distance themselves from their parents and they have to make important personal, academic and vocational decisions. At the same time, they have to adapt themselves to the physical, intellectual and hormonal changes that occur during puberty. All these stress factors can potentially trigger depression. Girls experience additional stress during puberty, such as biological changes (their first menstrual period, breast development) and psychosocial stress (increased concern for their body image).

Although it has been suggested that certain hormones, such as progesterone, estrogens and cortisol could be partly responsible for the gender differences in puberty, there is only limited evidence to suggest that these hormones affect the development of depression in women. Nevertheless, since this increased prevalence of depression occurs during a relatively short period of time, the neuroendocrine changes that take place during puberty must play a part. These biological changes and a certain genetic predisposition, combined with sociocultural factors, could place girls at an increased risk of depression. As regards emotional stress factors, adolescent girls tend to have doubts about themselves, doubts on their problem solving skills seeing their problems as unsolvable. Girls also have negative thoughts, such as self-blame and negative self-evaluation and they need a higher degree of approval and success than their male counterparts. These factors are related to the internalization of problems and depressive symptoms. These negative feelings could be related to being more sensitive to social expectations, difficulty in maintaining "suitable behavior", which is often the result of conflict stemming from contradictory messages sent by family, teachers, peers and society, with both cultural and socioeconomic components. Often girls have a strong desire to meet the skinny woman stereotype, are dissatisfied with their body image and feel pressured to loose weight.

Particular reference should be made to adolescent girls who are depressed during pregnancy or the postpartum period. Up to 68% of pregnant adolescent girls and 47% of those in the postpartum period could become depressed. This could be a reaction to the demands of pregnancy and motherhood complicated by the lack of social support, emotional support and feelings of guilt [8].

- Psychological factors: such as experience of failure, lack of control over negative events and defenselessness, low self-esteem, few social and communication skills, and little support from others.
- Sociocultural factors: premature death of a parent or loss of another loved one, conflict or divorce, family problems, socioeconomic needs, parents losing their job, childhood abuse or neglect and the fact that children spend a lot of time alone [1, 5].

Interaction with his or her surroundings affects the development of the child in an accumulative manner. This includes the influence of the home, school and the community [6].

We have already mentioned that parental depression may play a causal role in mood disorders in children, not only due to genetic mechanisms, but also because parents act as role models. Family risk factors for depression such as criminality, substance abuse by parents, lack of family cohesion, parent-child disputes, could all have an impact within the framework of a family history of depression. Childhood neglect and abuse could not only increase the risk of depression but also of substance abuse, disruptive disorder, posttraumatic stress disorder and suicide [2]. Furthermore, the lack of parental emotional support when the child is undergoing stress could induce the development of depressive disorders, especially in children with low self-esteem or who have a negative view of the world.

The school environment plays an important role in the life of children and adolescents. In addition to academic demands there are growing social demands, especially for children entering adolescence. Peer rejection can also increase the risk of developing depression. The impact of bullying should also be emphasized here.

In the community setting, exposure to violence increases the risk of depression in young people, especially if they are the victims of violence or witness violence towards someone they know. This is especially true in early adolescence [6].

- Cognitive factors: depressed individuals have a negative view of themselves, of the future and of the world. Non-depressed persons who have this tendency are more likely to become depressed when faced with a stressful situation than those who do not [2].
- According to Jacobs et al. [9] certain cognitive factors could be implicated in vulnerability to depression:

- attributional style: depressed individuals attribute negative events to internal, global, and stable causes. Viewing the causes of negative events as external, specific, and unstable represents a positive or adaptive style, which would protect from depression;
- dysfunctional attitudes: either alone or in interaction with life stress, predict depression;
- self-perception: negative self-perceptions regarding competence may constitute a cognitive vulnerability factor for depression. These negative self-perceptions are believed to result from the negative competency evaluations of significant others, such as parents and teachers.

In spite of the presence of several risk factors many individuals are very resilient to developing psychopathological disorders. This resistance may be due to the presence of a number of other factors in the child's life that counteract such risk factors, such an easy-going nature, high level of intelligence or strong social support [10]. Certain factors that could play a protective role have been pointed out:

- Social support: having friends reduces the likelihood of a child developing depression. During adolescence when the support of friends is combined with parental support, the likelihood of becoming depression is decreased.
- Personal competence: the ability to set reasonable goals and achieve them, provides a feeling of self-efficiency and a low risk of developing depression. Feeling good is very important and protects adolescents from depression. Becoming involved in a sport reduces the risk of depressive symptoms, probably due to social acceptance and satisfaction with body image.
- Religion and spirituality: it is not clear whether this protects from depression due to the beliefs themselves (especially regarding life project) or because of the feeling of belonging to a group [6].

It has been suggested that recent adult cohorts would have a greater proportion of psychiatric diseases, particularly depression, than previous cohorts of the same age. Proposing that depression is a growing public health problem. However, as Costello et al [11] have reported in a meta-analysis of 26 studies with around 60000 observations, there is no evidence to support an epidemic of child and adolescent depression (both major depressive disorder and other types of depression diagnoses), if variables such as age of the subjects at the time they are interviewed, gender, diagnostic classification, psychiatric interview used, or the temporal framework for performing the interview, are controlled.

PATHOGENESIS

According to Miller [12], early-onset depression may be related with impairment of neurobiological processes during development, which is modulated by environmental influences. Neuroendocrine, electrophysiological, and neuroimaging studies suggest that depression in children and adolescents emerges from dysfunction in neural systems involved in emotion. Neurobiological factors change during the course of development, which appears to contribute to the capacity for adaptive emotional responses. The neurobiological processes that change during development may become impaired during depressive disorders. Depressed children show abnormal cortisol and growth hormone release pattern, with a blunted response to serotonin-related drug challenges or insulin-induced hypoglycemia. These neuroendocrine findings underline the importance of basic biological rhythms involved in sleep, eating and stress sensitivity. Differences between depressed and non-depressed youngsters have been identified in the structure and function of cortical, limbico and striatal areas of the brain. Basic research with laboratory animals suggests that the prefrontal cortex and related cortical-limbic-striatal circuits regulate emotional functions such as neuroendocrine stress response, autonomic reactivity and reward sensitivity.

Based on these considerations, a dynamic adaptive systems framework has been proposed. It is based on the hypothesis that mood disorders reflect a deficit in emotional adaptation, and facilitating emotional adaptation provides a pathway to resilience to these disorders. Emotional adaptation is a fundamental process that enables the brain to receive and integrate environmental and somatic information and to guide appropriate responses over time. This adaptive systems framework explains how the depressive symptoms can be

analyzed into empirically-derived terms of hedonic capacity, stress sensitivity, ruminative self-focus and attentional impairments. Such components are clinically meaningful and quantitatively assessable and can be related to basic adaptive neurobiological, psychological and development processes that are relevant to etiology and intervention. The adaptive systems framework suggests continuity between typical and atypical developmental processes and it can be applied to the study processes involved in depressive disorders as well as pathways to emotional resilience.

DIAGNOSIS

Diagnosis should be based on an appropriate medical history that takes into account evolutional development and cultural factors. The opinion of parents and teachers, not just that of the child, is important. The child is sometimes unwilling to collaborate, is irritable and finds it difficult to express his/her feelings. The younger the child the more difficult it is to conduct a diagnostic evaluation. This is because children are less able to recognize and understand their symptoms and to express their emotions [5]. Children under 7 years of age may not be able to describe their mood but will express their discomfort by describing somatic symptoms and pain. Their irritable mood may be the reason why they are angry and display hostile behavior. Difficulty in paying attention, poor concentration and anxiety could be the symptoms of attention deficit/hyperactivity disorder and substance abuse may be a form of self-medication for depression. For the diagnosis of a primary depressive disorder the doctor must first rule out medical causes such as endocrine disease, cancer, chronic illnesses, infectious mononucleosis, anemia and vitamin deficiency (especially folic acid) and medicinal products such as isotretinoin. If any of these causes exist then the depression is secondary to medical illness. If there is no improvement after treatment or on discontinuing the medicinal product then a further assessment and additional treatment would be necessary [1].

Depressive disorders in childhood and adolescence are characterized by persistent sadness, anhedonia, boredom and irritability, which do not respond to experiences that would normally provide relief, such as pleasurable activities and contact with and attention of other people. The most important distinction between depression as an illness and the normal ups and downs in children and adolescents is that depression is associated with functional disorder. It is measured by intensity, duration and lack of response of the depressed mood and associated symptoms and is described as a change in the prior behavior in a specific child [2].

The diagnosis of major depressive disorder (MDD) or other depressive disorders in young patients should be based on the Diagnostic and Statistical Manual of Mental Disorders, 4th edition, text revision (DSM-IV-TR) [13]. The criteria for diagnosis of major depression are as follows:

1. depressed mood most of the day, nearly every day
2. markedly diminished interest or pleasure
3. significant weight loss when not dieting or weight gain
4. insomnia or hypersomnia nearly every day
5. psychomotor agitation or retardation

6. fatigue or loss of energy nearly every day
7. feelings of worthlessness or excessive or inappropriate guilt
8. diminished ability to think or concentrate or indecisiveness
9. recurrent thoughts of death, recurrent suicidal ideation or a suicide attempt or a specific plan for committing suicide.

At least 5 of the 9 criteria must be met and persist for at least 2 weeks. Criteria 1 and 2 must always be met. In children under 6 years, meeting criteria 3, 4 and 6 is considered sufficient for diagnosis.

Depending on the patient's age, changes to the criteria established for adults may be made. Thus, for example, an irritable mood rather than sadness may be considered for young patients. Although children may not suffer weight loss they may not fail to make the expected weight. Calles [6] distinguishes the following diagnostic characteristics in terms of age:

- Pre-school children: when assessing children in this age group it is important to be aware that a sad or irritable mood is a symptom with high sensitivity for MDD diagnosis and that anhedonia appears to show low specificity.
- School children: school children with depression tend to show symptoms that make them appear sad, although they may have difficulty in manifesting this feeling.
- Adolescents: same criteria as for adults.

Gender-related differences in symptoms of depression have also been described. Adolescent girls with depression show similar symptoms to those of depressed adult women, for example, anxiety disorders, eating disorders, adjustment disorders and at the same time show more florid symptoms. Compared to boys, depressed girls are more likely to experience guilt feelings, dissatisfaction with their body image, disillusion, feelings of failure, concentration problems, fatigue and concern about their health. On the other hand, depressed boys are more likely to have behavior problems as well as anhedonia and feel worse early in the morning, feeling weak and despondent [8].

Depressive disorders exist on a continuum and may be classified based on severity. Dysthymia is a milder form of depression than MDD, although longer lasting, and chronic, (during most of the day, almost every day, and at least for one year). It is characterized by depressed or irritable mood. Two of the following symptoms must also be present: changes in appetite, problems in sleeping, fatigue, low self-esteem, reduced concentration or difficulty in making decisions, and feelings of hopelessness. Children with dysthymia live in a gray world with no joy, sometimes accompanied by psycho-social abnormalities and poor school performance [5].

The most common depressive disorders in children and adolescents are adjustment disorders with depressed mood. Symptoms appear within three months of an identifiable stressful event which could be expected to interfere with social, occupational or school functioning. Symptoms should not meet criteria for other psychiatric disorders, not be caused by bereavement and not last more than six months after the elimination of the stressor.

For the diagnosis of minor depression, two of the MDD criteria must be met, one of which should be the presence of depressed mood, or diminished interest or pleasure. It should have a temporal course similar to that of major depression.

Atypical depression is characterized by hypersomnia, increased appetite, with compulsive consumption of carbohydrates, weight gain, interpersonal rejection sensitivity, leaden feelings in arms and legs, and mood reactivity.

Depressed mood, oversleeping, decreased appetite, and social withdrawal between October and February for two consecutive years suggests seasonal affective disorder.

Although less common, bipolar disorder is an important differential diagnosis. Risk factors for bipolar disorder are acute, early onset of depression, psychotic symptoms, significant psychomotor retardation, family history of bipolar disorder, any mood disorder in three consecutive generations of family members and mania induced by anti-depressants. Patients with this disorder have a high risk of suicide and may need hospitalization [1].

Depression can also be present as a subclinical condition, in which the individual may experience dysphoric moods and associated symptoms such as loss of self-esteem or pessimism. These subsyndromal conditions increase the risk for later major depressive episodes [12].

There are several scales that may be useful to guide the diagnosis of a depressive disorder. Those most commonly used are:

- *Children's Depression Inventory* (CDI): this contains 27 items and is for use with children and adolescents between 7 and 17 years. There is also a short version with 10 items. Each item is scored from 0 to 2 depending on the presence or absence of symptoms in the previous two weeks: 0 indicates absence of symptoms, 1 mild symptoms and 2 defined symptoms. The score is then plotted onto a graph and converted into a T-score. A score above 20 in the long version or 7 in the short version, or a T-score of over 65 are clinically significant. It requires a primary school reading age [1].
- *Beck Depression Inventory* (BDI): is the most commonly used inventory, it has 21 items each with four possible responses [5].
- *The Children's Depression Scale* (CDS): this self-administered questionnaire has 66 items, 48 focusing on depressed feelings (such as "I often feel lonely") and 18 on positive experiences (such as "I feel happy most of the time"). These items are interspersed so as to reduce the halo tendency in the responses, and to measure the "incapacity to experience pleasure or fun" as a component of depression. This enables two general, independent dimensions or scales to be obtained. A total depression dimension, with several sub-scales that assess mood, social difficulties, self-esteem, concerns about death/health, guilt and other depressive feelings and a total positive dimension with other subscales that assess mood-pleasure and other positive feelings. A score of 8 or more on a 1 to 10 scale indicates a diagnosis of depression. It has been validated for administration to individuals or to groups in children from 8 to 16 years. It has demonstrated excellent internal consistency (Chronbach's alpha of 0.96 and good test-restest correlation: 0.74) [3].

Comorbidity is the norm in children and adolescents with depression. Anxiety is often a precursor and could occur simultaneously with depression. Attention deficit/hyperactivity disorder often coincides with depression and they may be transmitted together in families. Alcohol abuse, drug abuse and smoking are associated with depression and a bi-directional

causality has been suggested. Behavior disorders are often comorbid with depression, particularly during puberty. Comorbidity with depression may be related to shared risk factors common to both conditions, as occurs in mood or behavior disorders and substance abuse which could be caused by common factors such as parents' substance abuse and criminality or exposure to family violence [2].

According to Calles [6] the following disorders may coincide with a depressive disorder:

- Anxiety disorder: depression and anxiety is a common comorbid pattern in young people, between 20 and 70% of young people with depression also have anxiety disorder. Anxiety tends to occur before the depressive disorder.
- Attention deficit/hyperactivity disorder: a 9 to 38% prevalence of depressive syndrome in children with this disorder has been reported.
- Behavior problems: which include oppositional defiant disorder and conduct disorders. The first is characterized by argumentativeness, loss of temper and disregard for authority, whilst in the second physical fighting, property destruction and other delinquent acts predominate. Given the disparity of symptoms related to depressive disorders the frequent co-occurrence with conduct disorders is noteworthy [10].

Forty five percent of children with depressive disorder meet criteria for oppositional defiant disorder, whilst 30% of patients with this disorder meet criteria for MDD. If oppositional defiant disorder is combined with a behavior disorder the prevalence of severe depression increases to 55% in these patients.

In the vast majority of cases, behavior problems develop prior to depression, although in some individuals the reverse could occur. It appears, therefore, that behavior problems have a predominant effect on depression and vice versa. Furthermore, although there are independent risk factors for both disorders, some are common (parent psychopathology such as depression, especially in the mother; antisocial behavior or substance abuse; emotion regulation defects and cognitive deficit and distortions), therefore co-occurrence of these two disorders could be explained by the overlapping of these risk factors. Wolff and Ollendick [10] have proposed a model for the development of comorbidity over time. This models suggests that risk factors, whether common to both disorders or just to one, could give rise to the development and interaction amongst them, thus risk factors exist that could lead to comorbidity. The model would explain the development and maintenance of behavior problems and depression, the interconnection of several factors and the direct and indirect relationship between these disorders over time. Behavior disorders and depression would be interrelated so that the presence of one disorder would increase the risk of developing the other. It seems more likely however, that behavior problems would increase the risk of depression rather than the other way round. Children could progress from depression to behavior problems as a way of exteriorizing interiorized feelings. Relating to the idea of "masked depression" depressed children may become aggressive as means of exteriorizing some of their feelings, particularly sadness.

It has also been suggested that depression may lead to behavior problems through cognitive weakening when faced with frustrating situations. Irritability and concentration difficulties may hinder the ability of depressed people to respond appropriately in certain

situations. For example, difficulty in perceiving the adverse consequences of their actions together with the affective barrier in their relationship with other persons, may foster interpersonal conflicts.

As regards the route from behavior problems to depression, the adverse consequences and life events associated with behavior problems may lead to a negative view of themselves, of the situation they are going through and the future. In this way, the combination of other people's negative reactions and personal functional impairment make them more vulnerable to depression.

- Substance abuse disorders: it has been demonstrated that adolescents with depressive symptoms start to consume alcohol and other drugs earlier than non depressed adolescents. There is also a link between the severity of depression and the severity of substance abuse.
- Developmental disorders: in several development disorders the prevalence of depression is higher than in the general population, such as in Down's syndrome, in children with neurological development deficiencies related to prenatal exposure to alcohol, girls with fragile X syndrome, Prader-Willi syndrome, velocardiofacial syndrome or neurofibromatosis.
- Finally, the coincidence with medical disorders such as diabetes mellitus and asthma is pointed out.

Although serious reservations have been expressed on the routine screening for depressive disorders in all children and adolescents, Cuijpers et al [14], in a systematic review and a meta-analysis of studies in which school children and adolescent students were screened for depression, and those with clinical depressive symptoms were treated, found encouraging results. The number of students that needed to be screened in order to generate one positive result was 31. This means that in a class of around 30 students, depression in one of them could be relieved by screening followed by early intervention.

PROGNOSIS

The duration of a depressive episode ranges from 3 to 8 months. Factors associated with longer episodes include comorbidity, either with dysthymia, anxiety disorder or substance abuse, greater severity at onset of depression, present or past suicidal ideation or conduct, chronicity and number of parental depressive episodes and family disputes. Approximately 20% of adolescents have persistent depression lasting 2 years or more. There is a high probability of relapses, ranging from 30 and 70% within the first and second year. Risk factors for these relapses are early onset of mood disorder in a parent, lack of complete recovery, preexisting social dysfunction, history of sexual abuse and family disputes [2].

As already pointed out there is a close relationship between past or present suicidal ideation or conduct and the persistence of a depressive episode. In the USA suicide is the third cause of death among youths between 15 and 24 years and of these 90% have suffered from depression, bipolar disorder and other psychiatric disorders. Most cases of depression are undiagnosed and untreated. Only 2% of the adolescents who committed suicide were

under medication [15, 16]. Around half of youths diagnosed with depression will attempt suicide at some time in their lives, and around 2% of them will complete it [17].

Depressive disorders may be persistent in around half of patients who suffered depression in childhood or adolescence, both in clinical and community groups. The results of a studied performed by Dunn and Goodyer [18] suggest that the probability of reoccurrence is high, even if the original depressive episode is moderate. In 40% of patients depression is recurrent, with significant periods of remission. A small but significant group (18%) remained persistently depressed into adulthood. Recurrence is more probable in females, where males are more likely to have persistent depression. For most of those with persistent depression the index episode was accompanied by at least one comorbid disorder. Complex diagnostic profiles developed, including a high rates of psychotic illness (30%), dependence (30%) and suicidal thoughts or attempts (87%). In over half (53%), the patients did not adhere to treatment. These individuals had been depressed for long periods of time before starting treatment and had been severely affected.

This study analyzed a group of patients from a clinical setting and another from a community setting. In the clinical group, those whose index episode remitted took more time to recover than those in the community group. Almost all adolescents assessed in the community group recovered from the first depressive episode: 68% in 3 months and 92% in 3 years. This may be explained by less severe index episodes with absence of suicide attempts and psychotic features, almost an absence of self-harm and only moderate impairment. Despite this, almost half (48%) experienced recurrence of their depression. The first recurrences were similar to those experienced in the clinical group. A small proportion of individuals from the community may suffer persistent depression, with no remission.

Remission of the index episode of depression in the clinical group was slower: only 5% had achieved remission at 3 months, less than a third at 12 months and only half at 3 years. The mean time to full remission was 2 years, with 12 (22%) having no remission during the follow up period. Longer index episodes were associated with more severe impairment, being depressed for longer before starting treatment and having an early psychiatric episode.

TREATMENT

There are four empirically supported interventions for depression in adolescents: antidepressants, cognitive behavioral therapy (CBT), interpersonal therapy (IT) and, to a lesser degree electroconvulsive therapy. It is reasonable to begin treatment of mild depression with family education, advice and support, case management and problem solving. In more persistent or more severe cases, the use of three empirically validated therapies would be indicated: selective serotonin reuptake inhibitors (SSRI), CBT or IT. Each of the three interventions is acceptable for initial treatment of moderate depression. Choice may initially depend on the patient, and it is advisable to set a time period (6-8 weeks) to assess treatment response and consider the use of combined treatment, a change or increase in medication [2].

Despite this simple plan of general treatment lines we cannot ignore the wide controversy on the efficacy of these therapies. Neither can we draw any conclusions on effective maintenance treatments for depression in children and adolescents, because long term care of pediatric depression is as yet unexplored. We must bear in mind that there are few long term

studies on depressive disorders in children and adolescents, therefore, information on treatment and its repercussions is scarce [19].

EFFICACY OF NON-PHARMACOLOGICAL TREATMENT

In view of the wide variability of clinical expression of depression, its multifactorial etiology, and the importance that emotional and environmental factors have for the origin and development of some types of depression, alternative non-pharmacological treatments must be considered. These treatments are especially important in mild and moderate depression in which environmental and psycho-biographical factors have most weight. In spite of the difficulty of applying evidence-based medicine to assess the efficacy of psychotherapeutic and psycho-educational techniques, there are some studies in a population of children and adolescents with mild-moderate depression that report favorable results. In some cases with a positive balance superior to pharmacological treatment [20].

The National Institute for Health and Clinical Excellence (NICE) [19] has published a clinical practice guideline to advise of the identification and management of depression in children and young people. The guideline recommends the use of psychotherapeutic techniques, and not antidepressants, as first line therapy in children or adolescents with mild depression. They also indicate that in cases where pharmacological treatment is recommended, it should always be used in combination with psychological therapies. They favor CBT, IT and short-term family therapy (FT) as first line treatment for children and adolescents with moderate to severe depression. Structured psychosocial therapy should include individual components of CBT: behavioral therapy, problem solving therapy, psychodynamic therapy and support therapy. Several studies in an adolescent population support its efficacy, however they also recognize that 40% were non responders and that accessibility is limited in many countries.

Most research on the treatment of adolescents in the acute phase has focused on CBT. There has been less research into IT, FT and group therapy in younger children, or into maintenance treatment and prevention of relapses [6].

Systematic reviews on the efficacy of psychotherapy, particularly CBT, have limitations. The available evidence for this treatment may be biased by an overrepresentation of small positive trials that reflect a probable publication bias. The studies do not reflect adverse effects, although these are well documented in the scientific literature, or data on the cost-effectiveness of psychotherapy in youth depression compared with no treatment, waiting list controls, attention-placebo or routine treatment. For this reason, evidence-based recommendations are still limited.

Having established the above, the evidence for the efficacy of psychotherapy for depression in children and adolescents suggests its superiority over control groups (waiting list or placebo patients). The evidence for efficacy of CBT and IT in adolescents with moderate to severe depression is more robust than that for children with mild to moderate depression [21].

In an study whose objective was to determine if SSRI treatment, mainly fluoxetine, plus CBT was more effective in the short term than antidepressant treatment alone in adolescents with moderate to severe major depression, Goodyer et al [22] did not find any differences

between the two treatment models. This suggests that CBT contributes little benefit in the short term, when combined with a SSRI.

In contrast, the Treatment for Adolescents with Depression Study (TADS) [23], a controlled trial that compares psychotherapy and pharmacological therapy, demonstrated that fluoxetine alone was superior to CBT alone, whilst the combination of both was significantly superior in response and remission than CBT alone, than fluoxetine plus clinical management and than placebo plus clinical management. Response rate for fluoxetine was only 60.6% and response rate for fluoxetine plus CBT was 71% in major depression in adolescents. It should be pointed out that in the TADS study, the CBT group showed less improvement than in other previous studies.

Other studies, however, have demonstrated that combined therapy is superior to either of the treatments alone and that CBT is superior to pharmacological treatment (using sertraline) in the acute treatment of adolescent mild to moderate depression [24].

Based on the conclusions of Watanabe's [21] review and the TADS [23] it may be maintained that the results of psychotherapy alone, especially CBT and IT, are probably superior to the results of waiting list or placebo patients, but inferior to the results of a combination of psychotherapy and drug therapy. The addition of antidepressant drugs increases the efficacy of psychotherapy in the short term, although studies are needed on what would be the outcome in the long term [25]. Both CBT and IT have demonstrated greater efficacy in adolescents of 12 to 18 years and with moderate to severe depression.

In spite of the positive impact of psychotherapy on depression in young people, its effects are lower than those on other psychological problems in young people. Using an intervention component profile the Weize et al meta-analysis [26, 27] demonstrated that some common treatment elements may be identified as beneficial, including activities to encourage competence, improve communication and relationship skills, teaching how to solve problems, changing imaginary negative thoughts, using strategies to increase activity and show feelings in relationships.

Trained professionals are needed to administer these specific treatments, and they are often scarce or simply inaccessible in many communities. Pediatricians can implement CBT techniques and thus contribute to decreasing depressive and anxiety symptoms [28]. CBT stimulates the patient to become self-aware and change thoughts that perpetuate depression. Clinics should encourage patients to take more physical exercise and do more fun activities (natural antidepressants) and to develop a repertoire for problem solving.

Other studies have assessed family therapy, such as the clinical trial performed by Trowell et al [29]. This trial compared 72 patients allocated to two treatment groups: Individual psychodynamic psychotherapy and FT. The authors found that 74% of the IT group were no longer depressed following therapy and 100% showed no signs of clinical depression 6 months later. IT appears to have been effective in MDD, dysthymia and "double depression". This efficacy appears to have been persistent, with no relapses during 6 months follow-up. Furthermore, for all patients who were still depressed after therapy, their depression was resolved at the next control visit. This suggests a continued response after discontinuation of therapy ("sleeper effect"). In the FT group 75.7% of patients were no longer depressed following therapy and 81% showed no signs of clinical depression 6 months later. FT also appears to have been effective in MDD, dysthymia and "double depression" as it was persistent and there were no relapses during the 6 months following therapy. Additional improvements were found in some of the remaining cases of depression at the next

control particularly dysthymia and "double depression". The response rates in the two treatment groups were not significantly different at the end of treatment. The differences at follow-up were attributed to having included 4 lost-to-follow-up cases in the FT group, without which the differences would not have been statistically different. In both groups a similar improvement was found in terms of deterioration and functioning levels. The authors conclude that both therapies were effective for the treatment of moderate to severe depression in children and adolescents, including cases of dysthymia and "double depression", which are generally considered more difficult to treat. They also found a reduction in comorbid conditions across the study.

EFFICACY OF PHARMACOLOGICAL TREATMENT

To be able to resort to more accessible effective treatments has become a significant, recent challenge for healthcare professionals in the management of major depression in children and adolescents. In this respect, in the last few years antidepressants have been considered as a plausible alternative, given the severity of the disease and the need to administer treatment that controls symptoms, mainly in the case of severe major depression with risk of suicide. Thus a spectacular increase in the use of antidepressant drugs has occurred in the last few years. This may be attributed to the relative ease and safety of use of the SSRIs compared to the previous tricyclic antidepressants.

Nevertheless, research in the last few years has questioned the use of antidepressants in children and adolescents. In fact, the increase of suicide-related behavior and the lack of evidence for the efficacy of these treatments have generated doubt on the safety of these drugs and social alarm is reflected in the actions of scientific institutions and government agencies.

In a meta-analysis published in 2006 by Papanikolaou et al [30], tricyclic antidepressants did not show a significant benefit compared with placebo in the treatment of adolescent depression, unlike the SSRIs which did provide significant benefit. The authors concluded that, in spite of some encouraging results on the use of SSRIs in the treatment of adolescent depression, caution is warranted until the long term safety of these agents may be demonstrated.

Therefore, the study of the efficacy of the SSRIs and the new antidepressants in children and adolescents is still at a relatively early stage. Only as regards fluoxetine is there any agreement on its efficacy and it is the only antidepressant approved for the treatment of depression in children [31]. Trials with other antidepressants are less conclusive although there are encouraging data for citalopram and paroxetine. In general, studies conducted so far do not provide any definite conclusions on the use of other SSRIs and of other antidepressants in children and adolescents [20].

It may be maintained that the few studies conducted so far do not provide conclusive results on the efficacy of the SSRIs as a group and even less for the more recent antidepressants. Furthermore, the notable differences in primary results amongst the studies, the heterogeneity in the definition and the methods used to determine the adverse effects and the lack of availability of non-published data further hinders the process of choosing the best treatment for each particular clinical situation [31].

The Chochrane group on depression, anxiety and neurosis [32] has conducted a recent review to determine the efficacy and adverse outcomes (definitive suicidal behavior and suicidal ideation) of SSRIs compared to placebo in the treatment of depressive disorders in children and adolescents. The authors report trials that found a treatment response at 8 to 12 weeks in children and adolescents, and a significant increase in the percentage of patients who improved when treated with an SSRI compared to patients in the placebo group. Most of the studies analyzed showed similar results, although they used different types of SSRIs and the age groups in the trials were different. The authors emphasized that the clinical importance of these results is not clear, due to the high drop out rate and doubts as to the appropriateness of the measuring instruments. There were also queries on the response to SSRIs in children and adolescents with co-existing diseases, and in those with risk of suicide, because in the majority of studies this group of patients was excluded. We must bear in mind that adolescents with MDD seen in clinical practice are characterized by having suicide-related behavior and comorbidity.

The review by Usala et al [33], which included studies with different SSRIs, such as fluoxetine, paroxetine, citalopram, escitalopram and sertraline, reported that treatment with these drugs in children and adolescents with depression may be more effective that placebo. This review also reported that treatment with fluoxetine and CBT is more effective than treatment with placebo alone, fluoxetine alone or with CBT alone. The authors report that the combination of fluoxetine and psychotherapy should be considered as a first line option in the management of depression in children and adolescents. The meta-analysis by Wallace et al [34] demonstrated that patients treated with these drugs have higher response rates than those treated with placebo and that both fluoxetine and citalopram could be safe, effective treatments in these subjects, even when used without psychotherapy. According to these authors, non published studies on the benefits of using SSRIs in children and adolescents with depressive disorders show similar results as those reported for published studies.

SSRIs have demonstrated to improve symptoms in pediatric patients with disorders other than depression. In a review of clinical trials by Hammerness et al [35] to compare the observed benefits against the risks of SSRIs and evaluate their safety in pediatric patients, the authors reported that subjects treated with SSRIs showed a reduction in symptoms of depression and anxiety. An overall improvement in young people with moderate or severe depression who were treated with SSRIs was also demonstrated. It was also reported that the most effective treatment for young people with depression is the SSRI and CBT combination.

However, do all SSRIs have similar efficacy? According to Hetrick et al [32], fluoxetine was the only SSRI for which there was consistent evidence from three clinical trials for its efficacy in reducing depressive symptoms in both children and adolescents. Patients treated with this drug scored lower on the Children's Depression Rating Scale-Revised (CDRS-R) that those treated with placebo. Therefore improvement was greater in those who took fluoxetine. It is not clear if this difference is meaningful for the treatment of depressive disorders in children and adolescents, since there was an improvement during treatment both in subjects treated with fluoxetine and those treated with placebo, but the patients treated with fluoxetine demonstrated a greater improvement. In the meta-analysis by Usala et al [33], that included studies with different SSRIs such as fluoxetine, paroxetine, citalopram, escitalopram and sertraline, a superior response was demonstrated in primary efficacy measures with fluoxetine. Kratochvil et al [36], in a study to evaluate the balance between benefits and risks of using SSRIs in children and adolescents with depression, also report that the combination

of fluoxetine and CBT is the best treatment for adolescents with major depression, especially when it is moderate or severe and there is a history of suicidal tendencies.

There were no clinically significant differences as regards response to treatment in children and adolescents who took paroxetine and those who took placebo [37]. In trials with sertraline or citalopram there were no statistical differences as regards the percentage of young people who responded to treatment between the treatment group and the placebo group. The percentage of subjects who responded to fluoxetine compared to placebo was greater than for sertraline and citalopram [32]. The safety and efficacy of venlafaxine in pediatric patients has not been demonstrated either [38].

Children and adolescent show different treatment response patterns. Donelly et al [39] determined the time to first response and time to persistent response of sertraline. The study was performed in subjects of different ages, and it was observed that time to persistent response to treatment was faster in adolescents although children had a faster time to first response than adolescents.

As emphasized by Findling et al [40], in many cases, the dosing strategies that were used in placebo controlled clinical trials in juvenile depressive disorders are not supported by the results of pharmacokinetic studies. Furthermore, in some clinical trials there is insufficient evidence to support or reject the dosing strategies used. The inappropriate medication dosing regimes may have contributed to the failure to detect efficacy in some studies with antidepressants. Likewise, the doses of medication may also have contributed to sub-optimal tolerability seen with some of these drugs. It should be borne in mind that there are limits to pharmacokinetic studies. Although these studies can provide vital information on how best to dose medications in a given population, the age-related differences in the pharmacodynamics are important considerations that can also substantially influence the tolerability and efficacy of the drugs. In addition, some of the pharmacokinetic studies are not designed to determine effective dose ranges for juveniles, instead they make comparisons based on pharmacokinetic parameters estimated in adults. Although such data may be used to provide rational dosing strategies for clinical trials, only rigorous, methodological, treatment studies can inform physicians of the safety, tolerability and efficacy of a given drug. Evidence-based dosing strategies should be developed before studying any drug in children. This is particularly important for antidepressants for several reasons. First, other methodological factors, such as high placebo response rates, could make it difficult to detect efficacy for an agent in the treatment of MDD. In addition, antidepressants could be associated with severe collateral effects when prescribed to children and adolescents. Therefore, in order to appropriately evaluate the safety and tolerability of a drug, empirically supported dosing strategies must be used. In view of the fact that children respond to medicines differently from adults, data derived from adults are not applicable to juveniles. It is important, therefore, that pharmacokinetic, pharmacodymanic and placebo controlled clinical trials are performed in children and adolescents.

SAFETY OF PHARMACOLOGICAL TREATMENT

The appearance of risks that accompany SSRIs may offset the benefits of their use in children and adolescents. In the last few years use of SSRIs has been linked to an increased risk of suicide and suicide attempts.

Moderate or severe psychiatric adverse reactions, although rare, do occur. However, suicide tendencies in pediatric clinical trials with antidepressants, although few, raise ethical concerns on their use in children and adolescents. Juveniles treated with antidepressants must be strictly monitored [31]. Such reactions could be related with dose. Also the concurrent psychotherapy may offer certain protection from these changes (CBT may help to prevent suicide ideation), but these assumptions need further study [41].

There is much concern therefore on the safety of SSRIs. The short follow up period in clinical trials makes it difficult to assess the serious adverse effects. Determining which children may have a greater risk of adverse outcome is a key factor for the safe use of these agents.

Headache, nausea and dizziness are common adverse effects reported in patients who have taken paroxetine. Other reported adverse effects are somnolence, insomnia and emotional instability [42]. The adverse effects reported for subjects who have taken fluoxetine are: headache (the most common), diarrhea, somnolence, insomnia, emotional instability and mania or hypomania [23]. Diarrhea, vomiting and insomnia may also occur with relative frequency. Sertraline is generally well tolerated, as demonstrated by Donnelly et al [39] in a follow-up of more than 34 weeks. However as there were numerous drop-outs and suicide-related events were reported, these authors recommend strict monitoring of patients treated with this SSRI in order to detect any possible risks. Furthermore, nausea was reported frequently in depressed children and adolescents treated with sertraline [43]. Escitalopram is another SSRI that has been studied to examine its efficacy and safety in the treatment of depression in the pediatric population. It was observed that this drug is well tolerated in adolescents, despite reports of adverse effects such as headache and abdominal pain [44].

Without doubt the most controversial aspect of the SSRIs is the rate of suicide-related events. The increase of suicide-related behavior and the lack of evidence for treatment efficacy have generated doubts on the safety of these drugs and social alarm is reflected in the actions of the scientific institutions and government agencies.

In 1989, a patient who had been taking fluoxetine for weeks, shot dead eight people and injured twelve others before killing himself at his place of work. This lead to a legal action against the manufacturers of fluoxetine. This raised the possibility of a link between the antidepressant use and violent acts [45]. Similar cases appeared in the following years in which aggressive behavior and suicides were related with taking antidepressants, both in adult and adolescent patients. This led to the appearance of the first neuropsychopharmacological task force in 1993, who considered that there was a possibility of an increased risk of suicidal behavior in the first stages of treatment, in dose changes and in the treatment withdrawal stage. As from 2003, the USA and UK healthcare agencies warned that the use of SSRIs could increase anxiety, agitation, panic attacks, insomnia, irritability, hostility, aggressiveness, mania and of the small but significant increase in suicide risk in children and adolescents. They publicly expressed their concern that SSRI treatment in children and adolescents could increase the risk of suicide ideation and suicide attempts. In September of

the same year a Task Force was formed on SSRIs and suicidal behavior in youths. It was appointed to evaluate the safety and efficacy of these drugs in patients under 18 years with depression. The Task Force considered three explanations for the frequent occurrence of suicidal behavior in depressed youth (whether in treatment or not). One is that SSRIs fail to relieve the suicidal behavior associated with depression because of a lag in antidepressant effect, or an incomplete response, or a treatment-resistant depression. A second possibility is that the SSRIs generate a novel set of suicidal emotions or behaviors. A third possibility is that SSRIs cause some patients to report already existing ideation or behavior, or clinical improvement leads to acting on existing suicidal feelings [46].

The evidence being accumulated through different clinical trials pointed towards an increase in aggressiveness, irritability and suicidal thoughts in the first month of treatment, in treatment changes and withdrawal of treatment [43]. However, they have serious limitations due to the absence of data from the patients' past, the heterogeneity of the populations, short duration and small samples sizes.

In a review of randomized clinical trials involving 1619 children and adolescents aged from 6 to 18 years, Sharp and Hellings [47] determined that fluoxetine, paroxetine, sertraline and citalopram were well tolerated and had some efficacy in the treatment of depression at these ages. However, reanalysis of published and unpublished studies by the US Food and Drug Administration (FDA) and the UK's Medicines and Healthcare products Regulatory Agency (MHRA) raised alerts regarding higher suicidal ideation rates from SSRIs in this population.

In the already mentioned TADS [23], that included depressed adolescents between 12 and 18 years allocated to four treatment groups (SSRI, CBT, combination of SSRI and CBT and placebo), an improved score for suicidal ideation was reported for all 4 groups. Fluoxetine alone did not significantly reduce the scores of the rating scale, used to measure suicide ideation, compared with placebo.

In a review of clinical trials Hetrick et al [32] found evidence for an increased risk of suicide ideation and behavior in patients prescribed SSRIs. However it is not clear if SSRI treatment will modify this risk in a clinically significant manner in children and adolescents, because the trials reviewed were not designed to appropriately measure any of the suicide-related outcomes.

A meta-analysis has been published that examines the risk of suicide-related behavior and suicide ideation in combination [48] or separately [49], which demonstrated a consistent and somewhat higher risk for SSRI use compared to placebo.

In a recent meta-analysis, Bridge et al [50] determined the efficacy and risk for suicide with antidepressants, differentiating between MDD, obsessive compulsive disorder and anxiety disorders. Studies and data were selected from PubMed (1988-2006), USA and British regulatory agencies (1998-2006) and clinical trial registries that included patients under 19 years. The authors came to the conclusion that relative to placebo, antidepressants are effective for the treatment of these disorders, although the effects are strongest in anxiety disorders, intermediate in obsessive compulsive disorders, and more modest in MDD. Benefits of antidepressants appear to be greater than risks of suicidal ideation and suicide attempts because, although the meta-analysis found an increased risk of suicide compared to placebo, the differences were not statistically significant.

There is new evidence to support the benefits of SSRIs for the reduction of suicide risk. The studies on this can be divided into toxicological and epidemiological studies. The former

are based on the determination of serum toxicology levels of antidepressants in persons who had just committed suicide. The results revealed that a very low percentage of patients who were treated with antidepressant had detectable plasma levels after their death. It may be concluded therefore that suicide occurred in those who did not follow the indications or in those who had discontinued treatment [15, 16].

Depression in itself is one of the strongest predictors of suicide-related events and a common factor in completed suicides. In moderate to severe depression the risk of suicide if not treated with antidepressants may outweigh the risk of self-harm associated with them [51]. Whilst the use of antidepressant drugs in children and adolescents has increased substantially in the last several years, the suicide rate amongst adolescents has gradually decreased: a 1% increase in adolescent use of antidepressants was associated with a decrease of 0.23 suicides per 100 000 adolescents per year [52]. In the light of available evidence, therefore, it may be concluded that the increased use of antidepressants has been a determining factor for the decline in suicide rates amongst adolescents [51, 53, 54]. This being so, changes in prescribing habits and the decrease in the number of antidepressants prescribed could place a significant proportion of depressed youths in danger and have serious consequences for public health.

All in all, data on the benefits and risks of the SSRIs in the treatment of depression in children and adolescents are not conclusive. In order to decide on how to treat depression in minors, the clinician must consider: the information available on the potential benefits and risks of pharmacological treatment; the fact that other forms of intervention, such as psychotherapy, have demonstrated efficacy for acute treatment; the possible risks of not treating and the particular needs of each individual [30]. Therefore, different treatment options should be considered after appropriately informing patients and their families not only of the potential benefits and risks of SSRI use, but also those of not treating depression (functional impairment, risk of suicide). In any event, the need for constant, close monitoring of children and adolescents treated with SSRIs in order to detect signs of suicide-related behavior cannot be repeated enough [55]. Doctors should not only ask about suicidal behavior, but also determine if there has been any history of such behavior [56]. Both doctors and families should watch for any unusual change in behavior, worsening of depression and suicide tendencies. It has been reported that the period of greatest risk is at the start of treatment when the patient begins to feel better, because the activator effect of the antidepressants appears before mood is improved. At this time the patient with suicide ideation finds the energy needed to carry out their proposal [57].

The decision to resort to pharmacological treatment can only be made on an individual basis and depending on the risk involved, the symptoms to be treated and on failure of psychological treatment. If antidepressants need to be administered, it should be borne in mind that fluoxetine has demonstrated a favorable benefit/risk balance and that if used the patient must be strictly monitored, especially at the start of treatment [20]. This pharmacological treatment must be started at low doses (equivalent to 5-10 mg of fluoxetine) and the dose increased every 2 weeks if response is not sufficient and no significant adverse effects occur. At the present time, the FDA recommends weekly personal monitoring for the first 4 weeks after the start of antidepressant treatment or after any dose adjustment [56]. As already pointed out, doctors and caregivers should watch for any unusual change in behavior, worsening of depression and suicidal behavior after initial treatment and, sometimes, after a change in dose. At the same time, patients and their families should be informed that

adjusting the medication without consulting the doctor could cause complications, and that all dose adjustments must be made by the doctor.

Therefore, the use of SSRIs is only recommended for adolescents with moderate or severe depression. Use must be individualized depending on the risk involved, the patient and family must be kept informed of the risks and benefits and the patient must be closely monitored, especially and the start of treatment [55].

To close this chapter on treatment we set out some general recommendations for the management of depression in children and adolescents.

TREATMENT ALGORITHM APPROACH FOR MDD IN CHILDREN AND ADOLESCENTS

The *Texas Consensus Conference Panel on Medication Treatment of Childhood Major Depressive Disorder* [58] has established a number of recommendations focused on subjects aged between 6 and 17 years who meet the DSM-IV criteria for MDD and whose depression is considered severe enough to justify medication: significant deterioration in psychosocial aspects, family relationships or school performance and/or risk of self-harm. There are not sufficient data to establish recommendations for children younger than 6 years old. Consensually agreed on medication algorithms based on available evidence were incorporated. These address acute treatment, recommendations for continued treatment and maintenance treatment for the prevention or relapses and recurrences.

Stage 0

Diagnostic Assessment

Includes an assessment of compliance to diagnostic criteria, assessment of family environment and initial monitoring. Indirect evidence suggests that depression in children and adolescents is influenced by psychosocial variables such as school friends and family members, as well as circumstances of their immediate environment and it appears to be more sensitive to non-specific treatment, such as the placebo effect. There is no evidence for determining the exact time period for this assessment and when to begin medication if necessary, which by consensus is established at 15 days. Psychoeducation and life-style training could be useful during this period.

As observed in intervention studies, families and patients need to be educated about the causes and symptoms of depression, the impairment related to it and the expected outcome of treatment, so that they can understand the disease and the steps to be taken to manage it. Doctors should be aware of the negative reactions of family members to a possible diagnosis of depression in an adolescent [59].

Assessment of Suicidal Tendencies

Tthe importance of evaluating past and present suicidal thoughts and behavior and also the need for close monitoring, especially in the first few weeks of antidepressant treatment when, as pointed out, there appears to be an increased risk of self-harm.

Non-Medication Treatment Against Medication Treatment

The decision must be based on appropriately informed parents and the available evidence on the efficacy and safety of the interventions. As already mentioned, there is consistent evidence for the efficacy of therapies such as CBT and IT in mild and moderate depression, both in adolescents and in children.

It is generally recommended that doctors maintain a good therapeutic relationship with the patient and family and incline towards continuity. The doctor should handle the interview with children and adolescents appropriately [60].

There is not sufficient evidence on the efficacy of other self-help or complementary treatments for depression such as glutamine, *S*-adenosylmethionine, St John's wort, vitamin C, omega-3 fatty acids, light therapy, massage, art therapy, bibliotherapy, exercise, relaxation therapy and sleep deprivation [61].

Stage 1

Use of SSRIs as Monotherapy

At this stage of intervention the use of antidepressants as monotherapy is determined. As a consequence of the warnings issued by Medicinal Products Safety Agencies, the use of antidepressants must be accompanied by the informed consent of parents or guardians. The recommended antidepressants are fluoxetine, sertraline or citalopram. At least one clinical trial has demonstrated the efficacy of these three SSRIs, however regulatory agencies such as the FDA require at least two studies that show consistent efficacy. In this respect, fluoxetine is the only antidepressant that has robustly and repeatedly demonstrated efficacy in the treatment of depression in children and adolescents [25, 60]. For this reason, it is the only antidepressant approved for this indication in this population group and is, therefore the drug of choice, unless there are reasons for selecting another SSRI (possible interactions, history of poor response with suitable dosing, family opposition). The SSRIs should be used at low starting doses, for example 10 mg of fluoxetine a day, and be gradually increased according to need and tolerability.

Stage 2

Switch to Another SSRI as Monotherapy

This stage is recommended in children and adolescents who have not improved with the stage 1 intervention, either due to lack of clinical response or intolerance of the drug (for example, nausea, excessive restlessness or agitation). The alternative would be another SSRI: fluoxetine, if it has not already been used, sertraline, citalopram, escitalopram or paroxetine (only in adolescents). Escitalopram and paroxetine are not included in stage 1 because they do not have grade A evidence, furthermore paroxetine appears to be associated with more abandonments due to adverse effects, such as agitation and hostility and it has been suggested that it entails a greater risk of self-harm in pre-adolescents.

Add Another Drug to the SSRI Treatment

In patients who show partial response another drug may be added. However, there is little evidence to support the efficacy of this intervention in children and adolescents. The majority of recommendations are extrapolated from available data on adults, such as combining an SSRI, with mirtazapine or bupropion, however these combinations have not been studied in children and adolescents. Therefore their efficacy and safety has still to be determined.

Stage 3

Switch to Another Antidepressant as Monotherapy

To pass on to stage 3 the patient must have experienced at least two failures with an SSRI. At this stage, the accuracy of the diagnosis, the presence of comorbidity and associated factors and the suitability of the psychotherapies administered, must be assessed and changes made if necessary. The recommended drugs as this stage are: bupropion, venlafaxine, mirtazapine and duloxetine. However, although these new antidepressants have shown to be effective in adults available evidence in the pediatric population is scarce and with no clinical relevance. Therefore, the recommendations are exclusively based on the extrapolation from results of studies on adults and these drugs are not recommended for initial treatment, although they could be considered at stage 3 in patients who have not previously responded to two SSRIs at an appropriate dose and treatment duration.

Other drugs such as nefazodone are not included at this stage due to their adverse effects that include hepatotoxicity. Tricyclic antidepressants are not included either, since apart from being ineffective in children and adolescents, their use entails a risk of cardiotoxicity. In this respect, venlafaxine has caused a significant increase in suicide ideation.

Stage 4

General Recommendations

There is no evidence to support other specific recommendations in a situation of continued lack of response. However, there are some general considerations, such as: reassessing the diagnosis and presence of comorbidity, including substance abuse; ruling out or addressing coadjuvant factors, such as family dysfunctions and the dose and duration of appropriate treatment and the patient's treatment adherence.

Specifically the following should be reviewed:

- If the patient had been having psychotherapy, its appropriateness should be assessed. If CBT or IT has not been used, they should be recommended.
- A combination of antidepressants should be considered, despite the fact that there is little available evidence for their use. The risks/benefits must be assessed beforehand according to the patient's clinical situation.
- If depression is severe and not responding to treatment, electroconvulsive therapy may be considered. There is sufficient grade B and C evidence for its use in children

and adolescents, respectively. In some US states its use is banned in children under 16 years.

In general, most patients respond to the first intervention stages in which treatment with the most evidence for safety and efficacy is recommended. For more resistant depression the other options may be considered.

MDD Treatment in Children and Adolescents

Acute Phase Treatment

Treatment objective at this initial phase is remission of symptoms. For patients showing minimum or no response it is important to continue treatment for 4 to 8 weeks from start of treatment before considering a change of treatment stage. If the patient shows a partial response, initial treatment may be maintained for up to 12 weeks and the dose may be increased, prior to changing the treatment stage. If there is still only partial response at week 12, moving to the next stage would be recommended because the presence of residual symptoms, without complete remission, considerably increases the risk of relapses. Frequency of visits is established by consensus, initial frequency being – each week during the first month and every 15 days during the second and third months. The objective of these visits is to assess the patient's clinical status, risk of self-harm, adverse effects of the drugs and dose adjustment if required. These visits also appear to encourage treatment compliance, enhance children and family participation in the treatment and provide opportunities for implementing educational measures. In any event, the frequency of the visits may need to be changed due to several associated factors: presence of suicidal ideation or behavior, adverse effects, severity of psychosocial stressors, level of family support, treatment response, etc.

The use of scales to measure and monitor treatment response may be evaluated.

Treatment should be started at low doses in order to minimize adverse effects, titration will be based on clinical response.

Children should be closely monitored, if adverse effects occur the dose should be reduced or treatment discontinued. The half life of the SSRIs and the new antidepressants is shorter in children, therefore they could suffer withdrawal symptoms at 8-12 hours of discontinuing the antidepressant (except with fluoxetine). These symptoms could be confused with lack of response or a relapse. The SSRIs are associated with the following adverse effects: headache, gastrointestinal disorders, sexual dysfunction, increased sweating, sleep disorders, and rarely serotonin syndrome. The occurrence of antidepressant-related suicidal thoughts or behavior, aggression, mania and motor hyperactivity or apathy will be monitored [28]. If these occur, closer follow-up will be implemented: more visits, telephone follow-up and family participation, including establishing a safety plan with the patient and family. There is no evidence for the efficacy of combining other drugs for the control of these symptoms. The multiple potential interactions of antidepressants with other drugs must be considered, and monitored.

Continuation Phase Treatment

The aim is to prevent relapse. Continuation of treatment is recommended for another 6 to 12 months after symptom remission (i.e. after successfully completing the acute phase of treatment) at the therapeutic dose used in the acute phase. Frequency of visits should be at least once every 3 months (preferably every 1 to 2 months) in order to evaluate the occurrence of symptoms indicative of relapse, the presence of suicide ideation or the onset of symptoms of mania.

If this was the first depressive episode patients should be evaluated for discontinuation of treatment at the end of the continuation phase treatment. If previous episodes have occurred patients should be evaluated for maintenance treatment. Amongst the factors to be considered are: severity of the depression episode, presence of residual symptoms, history of multiple episodes, family opinion and occurrence of adverse effects.

If none of the maintenance treatment criteria are met, treatment should be tapered slowly at not more than 25% per week over a period of 2 to 3 months. During this period and the months after treatment discontinuation, patients should be monitored for recurrence of symptoms. It depression reoccurs, treatment with previously effective medication should be reinitiated.

Maintenance Phase Treatment

The high rate of recurrence of MDD episodes in children and adolescents has already been pointed out. Some patients having a second episode and with related risk factors (family history, severity of episode, suicide conduct) and all those who have experienced 3 or more episodes, are candidates for maintenance treatment. The objective is to prevent recurrences. Maintenance treatment should carry on at full continuation treatment doses between 3 years and lifetime, depending on risk factors. In any event, optimal time has not been established in children and is extrapolated from available evidence in adults.

In a recent trial, a continuation of the TADS, 242 adolescents with MDD randomized into three groups: CBT, fluoxetine or a combination of the two, were followed-up from week 12 to week 36, in order to evaluate the efficacy of these three interventions in the continuation and maintenance therapy phases. The fluoxetine group were administered up to 50-60 mg depending on response, the CBT group, depending on response, had 6 to 9 sessions more than those prescribed in the acute phase, and in the third group the two therapies were combined. At week 12 response rates were significantly higher in the fluoxetine and combined treatment groups compared with the CBT group (67.5%, 70.9% and 42.1%). Nevertheless, the continuation and maintenance of treatment up to week 36 determined response rates of 70.4% for fluoxetine, 86.95% for combined treatment and 93.8% for CBT alone. Statistically significant differences were found between CBT compared to fluoxetine. Although more slowly, CBT was, in the end, more effective in maintaining treatment response [62].

Both fluoxetine and its combination with CBT appear to be at least as cost-effective in the short term as other commonly used treatments in Primary Care. Fluoxetine appears to be more cost-effective than combined treatment after 12 weeks of therapy [63]. However, another trial which determined the cost-effectiveness of SSRIs with or without CBT in 208 adolescents followed up for 28 weeks, appears to demonstrate that the CBT/SSRI combination is not more cost-effective than use of an SSRI alone in the short term. Although there were differences in favor of the SSRIs, these were not significant [64].

PREVENTION

Prevention may become an important way to reduce the enormous burden of mental disorders in the coming years [65]. Prevention is conventionally classified into primary, secondary and tertiary prevention. Primary prevention is focused on the reduction of new cases of a disease and is directed towards persons who are basically healthy. It could include treatment for persons who have known risk factors with signs and symptoms considered to be precursors of the disorder. Secondary prevention is concerned with detection and treatment of early signs of a disease, as well as treatment of a fully developed disorder. Tertiary prevention is concerned with minimizing the incapacity caused by a disease. Certain confusion has arisen as to whether early intervention should be classified as prevention or as treatment, i.e. as primary or secondary prevention. It is recommended that the term prevention be used when referring to those interventions that occur before the initial onset of a clinically diagnosed disorder, i.e., that are directed towards those children and adolescents with elevated symptoms of depression but who do not meet the criteria for a depressive disorder.

Prevention may be general, in which case the intervention is implemented for a certain population without regard to risk, or it could be targeted towards a population at high risk of developing the disorder. Specific preventive interventions may also be classified into selective interventions focused on populations with a risk factor for the disorder and indicated interventions that are targeted at populations who are already showing some signs and symptoms of a depressive disorder.

The two major benefits of a universal intervention are low stigma and the potential for widespread impact. That is, whilst targeted interventions involve a sub-sample of the population at risk, all participants stand to benefit from a universal intervention. However, universal interventions also have significant problems:

- Given the low prevalence of depression in an unselected sample, universal interventions require an enormous sample size, and this increases the cost. As pointed out by Cuijpers et al [65] one of the main problems in this type of investigation on prevention is the statistical power, with the large number of subjects that has to be included and the resultant high costs, especially when the incidence rates are low.
- Selecting an appropriate intervention given the different risk factors associated with depression that could potentially be addressed.

Targeted interventions have provided the most encouraging longitudinal results, particularly for diagnostic outcomes. However, they have some drawbacks. In particular, for selected intervention, because of the multitude of known risk factors only a fraction of the population who will eventually experience depression will be identified by a particular risk factor. Similarly, only a fraction of those who have a specific risk factor will experience depression. In order to increase the power, patients with multiple risk factors could be recruited. However, this increase in incidence comes at the cost of recruiting an even more specific sample, and may limit the generalizability of results. Furthermore, there are practical consequences to using a targeted design, such as the fact that the reliability of an “at risk”

classification based on a simple assessment may only be modest for some variables, or the arbitrariness involved in the selection of the cut off point to decide who is "at risk".

The programs are often focused on psychological interventions with an underlying emphasis on cognitive-behavioral therapy. Generally, the interventions are weekly and consist of between 12 and 15 group sessions: participants are taught how to overcome negative thoughts, solve problems and manage stress. The titles are generally positive [66].

Interventions based on exercise for reducing or preventing anxiety or depression in children and adolescents have been assessed. In a review by Larun et al [67] the authors concluded that whilst there appears to be a small effect in favor of exercise in reducing depression and anxiety scores in the general population of children and adolescents, the small number of studies included and the clinical diversity of participants, interventions and methods of measurement limit the ability to draw conclusions. It makes little difference whether the exercise is of high or low intensity. The effect of exercise for children in treatment for anxiety and depression is unknown, as the evidence base is scarce.

Both developmental and practical considerations make child and adolescent samples particularly appropriate for preventive interventions. From a practical sense, the use of schools for recruiting and implementing an intervention allows for a streamlined process of conducting research. Thus, targeting interventions to young people prior to a period of maximum risk for developing depression, between 13-14 and 18 years, will enhance the opportunity to reduce the incidence of major depression. However, the development of efficacious preventive interventions is still in its early stages. Different interventions have been used, such as cognitive reconstruction, problem solving skills, stress management, accessing social support, interpersonal skills and self-reward. From what we know at the present time it may be maintained that, despite some encouraging results, interventions of a more general nature obtain inferior short- and long-term results than more selective interventions. Interventions have generally shown small but positive short-term effects on symptoms and diagnoses [68].

As pointed out by Merry et al [66] psychological programs for prevention of depression have shown to be effective in preventing depression in the short term (some studies have demonstrated a decrease in depression at one year follow-up).

For Sutton [68] not all cognitive-behavioral interventions are equally effective. Whereas strategies focused on problem-solving skills do not appear to prevent occurrence of depression, programs focused on the development of cognitive skills appears to be more effective. This author highlights two prevention programs:

- The Penn Prevention Program consists of 10–12 sessions each lasting 90 minutes and has two primary foci: The first sessions focus on cognitive-behavioral concepts and tools (such as education about pessimistic explanatory style, cognitive restructuring, and decatastrophizing), and the second part of the program involves social problem solving skills (such as brainstorming, assertiveness and decision-making). The few studies that have assessed its efficacy show discrepant results. Although several studies have demonstrated at least small to moderate effects for this program on depressive symptoms at 1, 2 and 2.5 years post-intervention, in general the results are weak to date.
- FRIENDS program: this program was adapted from protocols designed to treat anxiety disorders in youths. It is a universal intervention focused on coping strategies

for anxiety and stress and includes tools such as cognitive reconstruction, relaxation and exposures with the help of parents. It consists of 10 weekly sessions lasting 75 minutes with students, 3 sessions with parents and two combined sessions at 1 and 3 months following completion of program. The results were less consistent for depression outcomes than for anxiety outcomes. In fact the FRIENDS program has acknowledged the substantial overlap of depression and anxiety and has demonstrated positive effects on symptoms of anxiety disorder. However, although the main foci of the program ostensibly overlap with those of other depression prevention programs, the FRIENDS intervention has not demonstrated a consistent nor long term effectiveness in preventing depressive outcomes.

Psychosocial interventions are not "inoculations" that are administered once and then offer protection either indefinitely or for many years. Ongoing, long-term programs are more likely to have longer-term effects than short-term interventions, therefore it has been recommended that booster sessions be included in the interventions.

To guide the decision on what type of prevention and on whether to even implement prevention, several variables must be considered: the cost of screening, sensitivity and specificity of the screening measure, the cost of the treatment and the prevalence of the disorder. Future research should pay particular attention to these considerations when evaluating the practicality and cost-effectiveness of implementing a particular intervention, when comparing competing interventions, and when considering whether to intervene preventively at all [68].

Widespread dissemination of preventive interventions is costly and such investment cannot be justified unless there is convincing evidence for their efficacy and effectiveness. Spence and Short [69] examined the evidence concerning the results of school-based, universal interventions for the prevention of depression amongst children and adolescents. The review focuses on school-based interventions specifically for depression rather than emotional well-being in general. In the last 20 years a growing number of studies have been conducted that investigated the impact of cognitive-behavioral approaches on the universal prevention of depression in children and adolescents, the majority in a school setting. However, scientific rigor was weak, making it difficult to draw conclusions on their efficacy and effectiveness. Despite the methodological concerns, some tentative conclusions may be established and some key areas for future research may be identified. The majority of the studies reviewed do not demonstrate positive effects on depression immediately after intervention. The results were marginally more robust around 6 to 10 months after intervention, but in those that had a longer follow-up period these effects were not maintained. There is insufficient evidence regarding efficacy and effectiveness that justify the widespread dissemination of school-based universal intervention for the prevention of depression in children and adolescents. Therefore, it may be premature to assume that investment in relative, brief, universal, school-based programs are likely to produce long lasting effects in the prevention of depression in the children and adolescents. Despite these reservations, research should continue in order to develop effective means of preventing the development of depression in youths. In particular, these authors suggest that current, brief interventions may not provide a sufficient "dose" to produce long-lasting benefits. We also need to ensure that the interventions are currently reaching those youths that are more likely to benefit from them. Finally, we need to make certain that those who administer the

intervention have sufficient training and supervision in order to ensure a high degree of reliability.

FUTURE RESEARCH

Zalsman et al [2] emphasize the following areas as needing more in-depth study in order to improve our knowledge on depression in children and adolescents:

- Genetic studies that would enable us to identify those factors related to the onset and course of depressive disorders, to help identify individuals at risk and to develop more precise treatment objectives. Likewise studies are needed to help clarify how factors linked to the development and to the environment can effect genetic expression.
- Neuroimaging is a promising field in which cerebral regions involved in the development of depressive disorders and recovery can be determined. This would help in the clinical classification of the disorders and in the selection of prevention and treatment objectives.
- We have little information on how the treatments currently available work. Further knowledge of their mechanisms of action may lead to a better treatment outcome and a more appropriate treatment assignment for the patient. Up to 40% of patients do not achieve full recovery after treatment. The most frequent sequelae is the occurrence of further episodes, therefore it would be useful to determine which treatment sequence and combination lead to complete remission.
- The most serious consequences of depression are suicide ideation and suicidal behavior. It is unclear if treatment of depression is sufficient to prevent suicidal tendencies or if another type of intervention is needed. Given the current concern that this could be an adverse effect of SSRI treatment it is important to have a better knowledge of the clinical, pharmacokinetic, pharmacogenetic and neurocognitive predictors of suicide in youths treated with these drugs.

Suicide-related events should be better characterized in the studies so that they can be better compared. This should also be generalized to other adverse effects for which the severity in each case should be better evaluated. Furthermore, prior behavior of the child or adolescent strongly correlates with present or future behavior, therefore, studies should not only report on the adverse effects but also on the history of similar behavior. Finally, adverse effects should be reported in a standardized manner in all clinical trials, so that rates can be compared amongst studies with a certain degree of confidence [54].

Other areas that need further research effort include:

- Little information on the economic burden of depression in childhood is currently available. More information is needed on the costs of depression and the cost-effectiveness of interventions to prevent and treat it [4].
- Future research should identify relationships amongst molecular, chemical and neurobiological processes and address questions on gender influences and

development. Furthermore, there is still a lot to learn on how such processes relate to the treatment responses and clinical presentations of childhood depression [12].

- Little is known on how the different cognitive vulnerability factors implicated in depression interact. Cognitive vulnerability in youths is probably a latent endogenous process, however there is a growing need for research on the evaluation of life stress and the incorporation of experimental paradigms of information processing and emotional saturation [9].
- More research is needed into the effectiveness of screening and early intervention. Before performing these interventions several questions should be answered on their use in routine practice, especially on the potential negative effects of these interventions [14].

Development of efficient preventive interventions is still in its early stages. Future research should investigate diagnostic outcomes, address protective factors, and explore variables of intervention. It will be especially important to establish how particular interventions work so that the active ingredients may be harnessed. Further, the effects of an intervention on different genders, ages, and cultures will be vital in the decision making of how to disseminate helpful interventions.

Few studies on the prevention of depression in children and adolescents have compared two active interventions. Future research could prove fruitful in that it may shed some light on those aspects of an intervention that are more strongly related to positive outcomes.

Careful training and supervision of group leaders who implement prevention programs may be an important factor in treatment efficacy, and is an area that merits further attention.

Additional studies on the ideal time for booster sessions and how to conduct these sessions are needed. Comparing interventions that include these sessions with those that do not would also be useful.

To guide the decision on what type of prevention and on whether to even implement prevention, several variables must be considered: the cost of screening, sensitivity and specificity of the screening measure, the cost of the treatment and the prevalence of the disorder. Future research should pay particular attention to these considerations when evaluating the practicality and cost-effectiveness of implementing a particular intervention, when comparing competing interventions, and when considering whether to intervene preventively at all.

Regarding the identification of risk factors, prevention research should attempt to capitalize on important advances in behavioral genetics research in order to identify at-risk participants in future preventive programs [68].

Future preventive approaches may need to be longer-lasting and more intensive, supported by booster sessions in order to achieve the acquisition of long-lasting skills. Furthermore, preventive approaches should be designed on ecological models of the etiology of depression in children and adolescents to provide a greater focus on that which reduces risk factors and increases protective factors within the child's environment, in addition to components focused on the individual [69].

The potential effectiveness of educational interventions has not been fully investigated. Given the gender differences in prevalence, and the change in these that occurs in adolescence with a disproportionate increase in prevalence rates for girls, it is likely that girls

and boys will respond differently to interventions, therefore a more definite description of gender-specific responses would be helpful [66].

CONCLUSION

Depressive disorders are frequent in children and adolescents with severe repercussions, which could include suicide.

Diagnosis of depression is difficult because it may present with atypical manifestations.

A large percentage of patients have a tendency for relapses and persistence of depression over time.

Treatment with SSRIs, mainly fluoxetine, is recommended. However if used, risks should be assessed individually and the patient must be closely monitored especially for suicide ideation or behavior, above all at the start of treatment.

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Chapter 7

THE RELATIVE LACK OF ATTENTION TO DEPRESSION IN YOUNG CHILDREN: A 'SAD' STATE OF AFFAIRS

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ABSTRACT

Historically, psychoanalytic theories suggest that it is not possible for children to be depressed. Today, using criteria from the *Diagnostic and Statistical Manual of Mental Disorders* (APA, 2000), children and adolescents can be diagnosed with depression. Further, developmentally appropriate modifications of these criteria are being described and validated for use in young children (Luby, Heffelfinger, Mrakotsky, et al., 2002). When age-appropriate symptom states are assessed, it becomes clear that young children who are between the ages of 3- and 6-years can experience stable and specific depressive syndromes (Luby, 2007). Even with these advances, however, relatively little attention is paid to the diagnosis of depression in young children, particularly in comparison to the occurrence of disruptive behavior disorders in young children (e.g., externalizing disorders and conduct problems often are cited as the most common reason for preschool referrals for mental health services; Luby & Morgan, 1997; Renk, 2005). As a result, this chapter intends to draw attention to the occurrence of depression in young children by discussing the criteria of available diagnostic systems, other considerations that may be helpful to mental health professionals, and available assessment measures and treatment interventions.

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INTRODUCTION

Historically, psychoanalytic theories suggest that it is not possible for children to be depressed. In psychoanalytic thinking, individuals can experience depression only after they have a fully developed superego. Given that the superego is not developed and internalized completely until adolescence or young adulthood (i.e., in psychoanalytic thinking), children cannot be susceptible to the symptoms of depression (Bemporad, 1994). It was not until the 1940s that thinking began to change. In particular, Spitz (1946) describes the changes in affect that occur when children are separated from their caregivers (e.g., with children 'in hospital'). In Spitz's (1946) paper, these changes in affect, along with a syndrome of other symptoms (e.g., whining, weight loss, impairments in social interactions, slowed or stunted growth), are referred to as *anaclitic depression*. Today, using criteria from the *Diagnostic and Statistical Manual of Mental Disorders* (APA, 2000), children and adolescents can be diagnosed with depression. Further, developmentally appropriate modifications of these criteria are being described and validated for use in young children (Luby, Heffelfinger, Mrakotsky, et al., 2002). In fact, some estimate that as many as 84,000 young children (i.e., those who range in age from 3- to 6-years; this estimate is based on the prevalence rate noted by Egger & Angold, 2006) may be experiencing clinically significant depressive symptoms (Stalets & Luby, 2006).

Even with these advances, however, relatively little attention is paid to the diagnosis of depression in young children, particularly in comparison to the occurrence of disruptive behavior disorders in young children (e.g., externalizing disorders and conduct problems often are cited as the most common reason for preschool referrals for mental health services; Luby & Morgan, 1997; Renk, 2005). As a result, this chapter intends to draw attention to the occurrence of depression in young children. In particular, this chapter will note the progressive nature of historical thinking about the occurrence of depression in young children (i.e., the previously accepted psychoanalytic thinking regarding symptoms of depression in children, the change in thinking promoted by Spitz's [1946] documentation of *anaclitic depression* in children following the separation from or loss of their caregivers, and current thinking regarding the acceptance of depression in children). Further, this chapter will survey current diagnostic systems (e.g., the *Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition-Text Revision* [*DSM*; APA, 2000], *Diagnostic and Statistical Manual for Primary Care* [*DSM-PC*] *Child and Adolescent Version* [Wolraich, Felice, & Drotar, 1996], *Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood: 0-3* [*DC: 0-3*; Zero to Three, 2005]) and the manner in which these systems describe depression in young children. In addition, this chapter will note modifications that have been made to diagnostic criteria in an effort to reliably diagnose depression in young children (Luby, Heffelfinger, Mrakotsky, et al., 2002). Finally, this chapter will survey possible assessment instruments and therapy interventions that may prove useful in addressing symptoms of depression in young children.

HISTORICAL OVERVIEW OF CHILDHOOD DEPRESSION

The existence of depression in young children has been debated for several decades and continues to be a source of contention in the field of psychology. In fact, there was little mention of childhood depression in medical texts before the 1800s (Haugaard, 2008). Around the mid-1800s, however, some physicians began to refer to melancholia in terms of childhood depression (Parry-Jones, 1995). In contrast, psychoanalytic theories, which were popular in the early 1900s, suggest that it is not possible for children to become depressed. In psychoanalytic thinking, the experience of depression can occur only after individuals have a fully developed superego. Given that the superego is not developed and internalized completely until adolescence or young adulthood (i.e., in psychoanalytic thinking), children cannot be susceptible to the symptoms of depression (Bemporad, 1994; Weller, Weller, & Fristad, 1984). Thus, in this line of thinking, young children are deemed cognitively and emotionally too immature to have or understand depressive symptoms (Luby, Heffelfinger, Mrakotsky, et al., 2003). Today, however, it is understood that young children are more sophisticated than once thought and can experience guilt and shame (Luby, 2007). Thus, developmental findings such as these negate these previous assumptions that suggest that children cannot be depressed.

In the 1940's and 1950's, thinking began to change when depressive symptoms were described in infants who were experiencing extreme circumstances of adversity (Luby, 2007). Based on the observations of the changes that occur in the emotional expression of institutionalized infants (Luby & Belden, 2006), pediatrician Renee Spitz (1946) describes the changes in affect that occur when children are separated from their caregivers (e.g., with children 'in hospital'). These changes include a transition from happy, outgoing behavior to weepy behavior during the first portion of infants' separation from their caregivers. Subsequently, the weepy behaviors that these infants exhibit transition into symptoms of withdrawal (e.g., ignoring the presence of other individuals). During this time, infants show many related symptoms, including weight loss, insomnia, as well as colds and other illnesses (Spitz, 1946). These changes in affect, along with a syndrome of other symptoms (e.g., whining, weight loss, impairments in social interactions, slowed or stunted growth), are referred to collectively as *anaclitic depression* (Spitz, 1946).

Suggesting that infants were capable of affective reactivity, even in a special population of institutionalized children, was not accepted readily. Instead, these assumptions were an impetus for decades of research and theory on depression in children. Despite Spitz' (1946) findings, many individuals continue to believe that childhood depression does not exist (Luby, Heffelfinger, Mrakotsky, et al., 2002; Stalets & Luby, 2006; Weller et al., 1984). This reluctance to accept depression in children is due to the fact that many view children as joyful and happy and consider childhood to be a time to be carefree. In the context of such attributions, depression would be impossible (Luby et al., 2002; Weller et al., 1984). Nonetheless, it should be recognized that young children experience rapid changes in their cognitive and emotional developmental that may serve as stressors and subsequently result in transient emotional and behavioral difficulties (Stalets & Luby, 2006).

Continued research in the 1960s, 1970s, and 1980s demonstrates that young children are more capable of experiencing and understanding depression than was once believed. Beginning in the 1960s, it is documented that children may experience "masked symptoms"

(e.g., acting out, running away, social phobia) that may be more characteristic of the clinical picture of childhood depression (Glaser, 1968; Weller et al., 1984). Then, in the 1970s, it was noted that children experience symptoms that are similar to those seen in adults (e.g., sadness, low energy levels, sleep disturbances; Kaufman, Martin, King, & Charney, 2001). They also experience additional behavioral manifestations of depressive symptoms (i.e., somatic complaints, social withdrawal, temper tantrums; Garber, 1984; Weller et al., 1984). In the 1980s, Kashani and colleagues (Kashani & Ray, 1983; Kashani, Ray, & Carlson, 1984) provide evidence that depression does occur in young children, albeit at a lower prevalence rate relative to other age groups. Thus, during this period, many new findings are described with regard to children's experience of depression.

Although many mental health professionals and researchers continue to believe that masked symptoms and behavioral manifestations characterize childhood depression, the use of the same diagnostic criteria for both children and adults is accepted. This practice began as early as the publication of the *Diagnostic and Statistical Manual of Mental Disorders-Third Edition* (Garber, 1984). Currently, mental health professionals continue to use the same diagnostic criteria, as assessed by the *Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition-Text Revised* (APA, 2000), when diagnosing children and adults with mood disturbances. These criteria, as well as those of other diagnostic systems, will be discussed next.

CURRENT DIAGNOSTIC CRITERIA FOR CHILDHOOD DEPRESSION

Childhood depression and related conditions are described in many of the current diagnostic classification systems. Although the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* is the most widely used and accepted diagnostic classification system in the United States, it is not the only system available to assist mental health professionals in conceptualizing childhood depression. The *Diagnostic and Statistical Manual for Primary Care (DSM-PC) Child and Adolescent Version* (Wolraich et al., 1996) and the *Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood: 0-3* (*DC: 0-3;* Zero to Three, 2005) are two other diagnostic systems that can serve as useful resources for the diagnosis of young children, particularly when mental health professionals are looking for developmentally descriptive criteria when diagnosing depression in young children.

DSM

The current edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*; APA, 2000), the *Fourth Edition-Text Revision*, uses a categorical classification system to identify psychological disorders. Of the different diagnostic groups included in the *DSM*, only one is dedicated solely to disorders occurring during childhood. This section, labeled 'Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence,' contains a number of disorders that typically manifest themselves during childhood. This section is not considered to be a complete listing of disorders that may be experienced during childhood,

however (APA, 2000). In other words, disorders that tend to have an onset in either childhood or adolescence are included in this section; however, children can be diagnosed with the majority of the disorders described throughout the *DSM*.

For example, depressive disorders are not listed in the section focusing on infants, children, and adolescents; however, they can be diagnosed in children, adolescents, and adults. It should be noted, however, that some have criticized the *DSM* criteria as being lacking in sensitivity to developmental variation (Stalets & Luby, 2006). All depressive disorders (regardless of their age of onset) are grouped under the Mood Disorders diagnostic category in the *DSM*. This section is further broken down into depressive disorders (i.e., Major Depressive Disorder, Dysthymic Disorder, and Depressive Disorder Not Otherwise Specified) and bipolar disorders (Bipolar I Disorder, Bipolar II Disorder, Cyclothymic Disorder, and Bipolar Disorder Not Otherwise Specified). There are other Mood Disorder possibilities as well (e.g., Mood Disorder Not Otherwise Specified; APA, 2000).

When diagnosing children with Major Depressive Disorder using the *DSM* diagnostic system, children must meet criteria for a Major Depressive Episode. For a Major Depressive Episode, children must meet five or more of the nine listed criteria, with one of these criteria being depressed mood or decreased interest or pleasure in usual activities (APA, 2000). The remaining criteria include significant weight loss or gain, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue or loss of energy, feelings of worthlessness or guilt, difficulty concentrating, and recurrent thoughts of death (APA, 2000). The *DSM* offers slight modifications in the diagnostic criteria for a Major Depressive Episode when such a diagnosis is being made in children and adolescents. For example, depressed mood may be manifested as irritable mood, and failure to make appropriate weight gains (as opposed to significant weight gain or loss) is considered a symptom specific to children (APA, 2000). In addition, children who are diagnosed with a Major Depressive Episode must not meet criteria for a Mixed Episode (an episode where symptoms of Major Depressive and Manic Episodes are being exhibited concurrently), must be experiencing clinically significant distress or impairment, must not be experiencing symptoms as a result of a substance or a general medical condition, and must not be experiencing symptoms that are better accounted for by Bereavement (i.e., the experience of symptoms following the loss of a loved one; APA, 2000).

Similar symptoms are noted when a diagnosis of Dysthymic Disorder is made using the *DSM* diagnostic system. In particular, to be diagnosed with Dysthymic Disorder, children must exhibit depressed mood over the course of one year (whereas the duration of symptoms is two years for adults). In addition, children can exhibit an irritable (as opposed to depressed) mood (APA, 2000). To make this diagnosis, children also must exhibit at least two of six depressive symptoms (i.e., eating too much or too little, insomnia or hypersomnia, fatigue, low self-esteem, difficulty concentrating, and feelings of hopelessness; APA, 2000). Finally, children cannot experience a Major Depressive Episode during the course of the first year of their Dysthymic Disorder diagnosis, must not meet criteria for other mood episodes (i.e., Manic, Hypomanic, or Mixed Episodes) or for Cyclothymic Disorder, must not meet criteria for a Psychotic Disorder, must not have symptoms from the effects of a substance or a general medical condition, and must experience clinically significant distress or impairment (APA, 2000).

It should be noted that the *DSM* diagnostic system does not define an age criteria for either Major Depressive Episodes or Dysthymic Disorder. For Dysthymic Disorder, however,

there is a subtype specification for age (i.e., either Early Onset, indicating an onset prior to the age of 21-years, or Late Onset, indicating an onset at or after the age of 21-years; APA, 2000). Thus, a diagnosis of Major Depressive Disorder or Dysthymic Disorder can be made for an individual of any age. Although research conducted with children and adolescents demonstrates the validity of *DSM* criteria for Major Depressive Disorder and Dysthymic Disorder, similar research has not been conducted on children who are 6-years of age and younger (Luby, Heffelfinger, Mrakotsky, et al., 2003). This lack of research is concerning, as children who are younger than 6-years of age are being diagnosed with depressive disorders based on diagnostic criteria that have no empirical validation for use in young children. Given this aspect of these criteria, other available diagnostic systems may be useful in identifying symptoms of depression in young children.

DSM-PC

The *Diagnostic and Statistical Manual for Primary Care* (*DSM-PC*) *Child and Adolescent Version* was developed in an attempt to bridge the gap between mental health and primary care medical settings (Wolraich et al., 1996). The *DSM-PC* is organized in an algorithmic fashion and includes brief information regarding the presentation of psychological symptoms in primary care settings (e.g., primary complaints that are likely to be seen by primary care physicians). Similar to the *DSM* diagnostic system, the *DSM-PC* provides a Major Depressive Disorder diagnosis and a Dysthymic Disorder diagnosis (using the criteria that are described above) as well as a Depressive Disorder Not Otherwise Specified category (Wolraich et al., 1996). In addition, however, the *DSM-PC* provides a code for Bereavement, which can be used when children experience sadness for a period of two months in relation to a major loss (Wolraich et al., 1996). Further, there also are categories for Sadness Variation, which is used to label transient depressive symptoms (e.g., withdrawal and sad affect in early childhood after losses) in response to stress that are considered to be normative, and for Sadness Problem, which is used to label mild depressive symptoms (e.g., depressed/irritable mood, diminished interest) that are not intense enough to qualify for a Major Depressive Disorder diagnosis (Wolraich et al., 1996). Thus, although the *DSM-PC* provides the same diagnostic categories as the *DSM*, this diagnostic system also provides other categories that note when children are experiencing normative varieties of depressive symptoms.

DC: 0-3

The *Diagnostic Classification of Mental Health and Developmental Disorders of Infancy and Early Childhood: 0-3-Revised* (*DC: 0-3R*; Zero to Three, 2005) was developed in response to the need for a diagnostic and classification system for infants and young children. Like the *DSM* diagnostic classification system, the *DC: 0-3R* uses a categorical classification system that provides descriptions of symptoms and syndromes. In contrast, the *DC: 0-3R* acknowledges the many challenges associated with accurate diagnosis of very young children. Given such concerns, the *DC: 0-3R* offers modified criteria to account for the developing cognitive and verbal skills of very young children (i.e., who are 0- to 3-years of age; Zero to

Three, 2005). It should be noted that the validity of these criteria has not been documented at this time (Stalets & Luby, 2006).

When using the *DC: 0-3R* diagnostic system, the depressive disorders are found in the 'Disorders of Affect' category (Zero to Three, 2005). The *DC: 0-3R* provides a Prolonged Bereavement/Grief Reaction diagnosis, which describes children who are experiencing at least three of eight symptoms (e.g., seeking out an absent caregiver, refusing the attempts of others to provide comfort, withdrawing emotionally, experiencing disruptions in eating, experiencing disruptions in sleep, exhibiting arrested development, showing a diminished range of affect, showing disturbance when faced with reminders of the caregiver) when they experience a significant loss. In addition, children who receive such a diagnosis experience a change in functioning and exhibit their symptoms over the course of at least two weeks (Zero to Three, 2005). Thus, this diagnostic category most closely resembles the Bereavement label described in both the *DSM* and *DSM-PC*.

The *DC: 0-3R* system also includes a subcategory of Depression of Infancy and Early Childhood. This subcategory is used to describe children who are exhibiting five general characteristics (i.e., children's disturbed affect and pattern of behavior should be a change from their usual mood and behavior, children's depressed/irritable mood must be persistent [i.e., occur for at least two weeks] and separate from sad experiences at least some of the time, children's symptoms should be pervasive, their symptoms should cause clear distress, and their symptoms should not be due to a general medical condition or the effects of a substance; Zero to Three, 2005). Once these criteria are met, a diagnosis of Type I: Major Depression is indicated for children who are exhibiting at least five other symptoms over the course of at least two weeks (i.e., from the same nine criteria described above for the *DSM*; Zero to Three, 2005). In contrast, a diagnosis of Type II: Depressive Disorder Not Otherwise Specified is indicated for children who meet criteria for only three or four of the nine criteria described above (Zero to Three, 2005). Thus, these categories are similar to disorders described in the *DSM* and the *DSM-PC*.

Other Modified Criteria

Because there have been questions about each of the diagnostic criteria described above, a task force examined empirically derived modifications that would allow the *DSM* criteria to be more developmentally appropriate for use with young children (Task Force on Research Diagnostic Criteria: Infancy Preschool, 2003). Based on the work of Luby, Heffelfinger, Mrakotsky, and colleagues (2002), these modifications suggest that the criterion regarding the experience of worthlessness or guilt and the criterion regarding the experience of thoughts of death and suicidal ideation or attempts may be evident in play themes. In addition, many of the criteria (e.g., depressed mood, lack of interest or pleasure, psychomotor agitation or ideation) can be met if other individuals observe them (i.e., instead of being reported by children themselves). Finally, the modifications suggest that the symptoms need to be present, although not necessarily persistent, over a two-week time frame (Luby, Heffelfinger, Mrakotsky, et al., 2002). This criterion may be particularly important, as 80 percent of young children who are depressed fail to meet a two-week duration for their depressive symptoms. In other words, the standard time frame of two weeks only captures the most severely affected young children (Luby, Mrakotsky, et al., 2003). These modified criteria demonstrate high

internal consistency as a cluster of symptoms, demonstrate sufficient specificity and sensitivity, and occur more frequently in a group of young children who are depressed (relative to a group of healthy controls and a group of psychiatric controls; Luby, Heffelfinger, et al., 2003). Thus, when diagnosing young children, these modifications to the *DSM* diagnostic criteria may prove to be particularly useful.

SUMMARY OF DIAGNOSTIC CRITERIA

Each of the diagnostic classification systems described above are valuable resources in the conceptualization and diagnosis of depressive disorders as experienced by young children. Although the *DSM* diagnostic classification system (APA, 2000) dedicates an entire section to disorders that usually occur in infancy, childhood, and adolescence, it does not include childhood-onset depression. Instead, childhood depression is diagnosed using the same criteria as adult depression, with some possible modifications in criteria for children (APA, 2000). The *DSM-PC* diagnostic classification system (Wolraich et al., 1996) conceptualizes depression in a manner similar to that of the *DSM* but allows primary care physicians in medical settings a structured, simple way to diagnose mental disorders. Further, this diagnostic classification system may facilitate communication and collaboration between medical and mental health professionals. In contrast to the *DSM* diagnostic classification system, the *DSM-PC* provides labels for the experience of depression that may not meet full criteria for a major diagnosis. Finally, the *DC: 0-3R* diagnostic classification system (Zero to Three, 2005) specifically classifies symptoms related to childhood depression in a manner consistent with the development of young children. Although these diagnostic classification systems are helpful in diagnosing the experience of depression in young children, some researchers are looking more closely at the use of these diagnostic criteria in young children and making suggestions for other important features that should be noted.

OTHER CONSIDERATIONS IN MAKING A DEPRESSIVE DISORDER DIAGNOSIS DURING CHILDHOOD

It is clear that attempts have been made to offer mental health professionals diagnostic classification systems that are relevant for infants (i.e., 0- to 3-years of age), children (i.e., 7- to 12-years of age), and adolescents (i.e., 13-years of age and older). In addition to the diagnostic criteria described above, it can be helpful to have information regarding the characteristics of infants, children, and adolescents who are experiencing depressive symptoms. Although some information has been provided for infants as well as children and adolescents, this information will be discussed only briefly here. Instead, the more substantial gap in the information relevant to young children (i.e., 3- to 6-years of age) will be the focus of this section. The new research that is being done with this population suggests that there are several important correlates for diagnosing depression in young children. This information will be discussed here.

Depression in Infancy

The majority of research on infants has been conducted with infants of mothers who are depressed. Relative to infants of mothers who are not depressed, infants of mothers who are depressed demonstrate less positive and more negative facial expressions (Murray, 1992). Physiological responses, such as decreased heart rate variability (Field, Pickens, Fox, Nawrocki, & Gonzalez, 1995b) and abnormal EEGs (i.e., similar to adults with depression; Field, Fox, Pickens, & Nawrocki, 1995), also are evident. These differences support the notion that depression does in fact have a biological or innate component that can manifest itself early in life (Luby, Heffelfinger, Mrakotsky, et al., 2002). Another longitudinal conceptualization of depression in infancy refers to temperament, or biological predispositions toward depressive affective states. Although a variety of hypotheses exist regarding the exact mechanism(s) relating temperament to later emotional functioning, most theories underline the importance of early interactions with caregivers for the expression and stability of depressive temperamental states. Further agreement exists, in that early expression of affective states is related to later social interactions and functioning in a social world (Trad, 1986). Overall, this research literature suggests that correlates of depressive symptoms can be present early in infancy and then be present throughout childhood, adolescence, and adulthood.

Depression in Childhood and Adolescence

The experience of depression in childhood and adolescence seems to have some similarities to the experience of depression in adulthood. As noted above, children and adolescents are believed to experience the same symptoms as adults (i.e., based on *DSM* criteria). Further, although prevalence rates for depression appear to increase with age (i.e., approximately 2% in children versus 6% in adolescents; Yorbik, Birmaher, Axelson, Williamson, & Ryan, 2004), the lifetime prevalence rates for children are similar to those for adults (i.e., 15 to 20%; Birmaher, Ryan, Williamson, & Brent, 1996). Birmaher and colleagues (2004) also report that, relative to adults, children and adolescents exhibit a similar duration and severity of depressive symptoms as well as similar rates of recovery and recurrence. In a study by Birmaher and colleagues (1996) examining the experience of depression in children (*M* age = 10.2-years) and adolescents (*M* age = 13.3-years), approximately 85% of both groups recover from their symptoms, and 40% experience a recurrent episode. Similar to adults, it appears that the experience of one depressive episode is highly predictive of future episodes. In fact, up to 70% of adolescents go on to experience a depressive episode in adulthood (consistent with a 70% recurrence rate for adults; Birmaher et al., 1996). One difference that did emerge in this study is that, similar to adults, female adolescents are two times as likely to experience depression as adolescent males. This difference is not noted for children (Birmaher et al., 1996).

In contrast, other studies find differences between childhood and adolescent depression and adult depression. These findings likely reflect developmental changes in emotions and cognitions. For example, children are more likely to demonstrate symptoms such as somatic complaints, separation anxiety, phobias, and behavior problems. As a result, children who exhibit these symptoms also are more likely to have comorbid diagnoses of Attention-Deficit/

Hyperactivity Disorder, Oppositional Defiant Disorder (Birmaher et al., 1996), and Separation Anxiety Disorder (APA, 2000; Yorbik et al., 2004). As children enter adolescence, they may be more likely to exhibit psychosis, suicide ideation, and suicide attempts and to experience more severe impairment (APA, 2000; Birmaher et al., 1996). Melancholic symptoms also may be more likely in adolescents relative to children (Birmaher et al, 2004), although this finding is not always replicated (Yorbik et al., 2004). More recent studies note an increase in the likelihood for adolescents to experience feelings of helplessness, hopelessness, decreased energy, weight loss, hypersomnia, and substance abuse (Yorbik et al., 2004). Despite these differences, both depressed children and adolescents experience difficulties with relationships and academic functioning. In addition, they both are more likely than children without depressive symptoms to have families characterized by high conflict, poor communication, less support and positive affect, and higher levels of abuse (Birmaher et al., 1996). Thus, although similarities in the characteristics of depression are noted across the lifespan, differences also are apparent.

Depression in Preschool Children

These and other studies using the established *DSM* diagnostic criteria seem to suggest that there are many similarities across the lifespan in individuals' experience of depression. As a result, the current *DSM* criteria (i.e., with possible suggested modifications for young children) may be a valid diagnostic tool for the diagnosis of depressive disorders for children as young as 7-years of age. It should be noted, however, that children under 7-years of age present additional challenges to the assessment and diagnosis of depressive symptoms due to their developing verbal, emotional, and cognitive abilities in relation to their exposure to different life experiences (Luby & Belden, 2006; Luby, Heffelfinger, Mrakotsky, et al., 2002; Luby, Heffelfinger, Mrakotsky, et al., 2003). Recently, attempts have been made to study young children's experience of depression in an effort to outline more age-appropriate diagnostic criteria (as noted above) and to identify other characteristics that may be relevant to young children's experience of depression.

One of the first questions that needed to be addressed with this line of research is whether or not young children even could experience depression. To address this question, Luby, Heffelfinger, Mrakotsky, and colleagues (2002) investigate whether or not children under 6-years of age can manifest depression. Based on the notion that some of the current *DSM* criteria and assessment protocols do not capture adequately the developmental level and life experiences of young children, modified criteria and assessment questions have been constructed. The modifications to the *DSM* criteria described above are used in diagnosing young children with depression in this study.

First, it was anticipated that young children who are depressed could experience anhedonia, or a lack of pleasure in activities and play or not having fun; demonstrate their feelings of worthlessness and guilt in themes of play; exhibit a diminished ability to think or concentrate and/or be indecisive over several days (i.e., to account for age-appropriate fluctuations in such behavior); and exhibit suicidal or self destructive themes in their play. Further, children were diagnosed and included in the study if they presented with only four (as opposed to five) of the nine core *DSM* criteria. Finally, children's symptoms did not need to be persistently present for a two-week period (i.e., also to account for age-appropriate

fluctuations in mood and behavior). To assist in diagnosing the young children in this study, the *Diagnostic Interview Schedule for Children-Version IV-Young Child* (*DISC-IV-YC*), a modified version of the *DISC-IV* that includes more age-appropriate questions regarding activities and play instead of work and school behavior, was used. Children who met the modified criteria constituted the depressed group and were compared to a psychiatric population (i.e., children who met criteria for Attention-Deficit/Hyperactivity Disorder and/or Oppositional Defiant Disorder) and a nonclinical control group (Luby, Heffelfinger, Mrakotsky, et al., 2002).

Luby, Heffelfinger, Mrakotsky, and colleagues (2002) indicate that 49 of the 136 preschool children in their study meet the modified diagnostic criteria for Major Depressive Disorder (i.e., *Preschool Diagnostic Criteria; P-DC-MDD*). Interestingly, 76% of the children in this study meet formal *DSM* diagnostic criteria with the exception of the two-week duration requirement, suggesting that up to three-fourths of young children who are clinically depressed may be overlooked in clinical settings. These children differ in many aspects of functioning when compared to the psychiatric and nonclinical control groups. For example, these children are noted to demonstrate typical symptoms (i.e., as opposed to masked symptoms) as described in the *DSM* when these symptoms are assessed with the translated, age-appropriate criteria. These children also display vegetative symptoms (e.g., sleep and appetite disturbances, changes in activity levels). Further, over 60% of these children display death or suicide related themes in their play. Overall, however, anhedonia emerges as a specific symptom of the children exhibiting depressive symptoms, as anhedonia is not seen in either of the other two groups of children that were examined. Overall, the modified constellation of symptoms continues to persist six months later, further supporting the validity of these modified criteria. Finally, greater social impairment, higher incidence of family history of mood disorders, and higher levels of self-reported depressive symptoms using a puppet interview further validate these modified criteria (Luby, Heffelfinger, Mrakotsky, et al., 2002).

In a subsequent study, Luby, Heffelfinger, and colleagues (2003) examine whether typical versus masked symptoms predominate the experience of depression in young children. A structured interview was constructed (i.e., *the Preschool Symptom Module; PSM*) to assess the presence of masked symptoms (e.g., sleep problems, whining/crying, being sad/grouchy, low energy) for this study. Results of this study suggest that, although some masked symptoms may be present, these symptoms are less frequent than might be anticipated. Results further indicate that typical symptoms of Major Depressive Disorder predominate the clinical picture of young children who are depressed. For example, 98% of the young children in this sample who are depressed demonstrate a sad or irritable mood. Further, relative to a psychiatric group and a nonclinical group, the young children who are depressed tend to experience the highly specific symptom of anhedonia. With regard to the masked symptoms that were examined in this study, the most common masked symptom in this sample is somatic complaints; however, this symptom is not specific to the young children who are depressed (i.e., the psychiatric group also experiences somatic complaints; Luby, Heffelfinger, et al., 2003). Thus, research suggests that young children can experience clinically concerning symptoms of depression. Further, previously accepted masked symptoms may be more typical of younger children but, based on the findings of Luby, Heffelfinger, and colleagues (2003), do not predominate the clinical picture. Therefore, it is

suggested that masked symptoms be considered by mental health professionals but not become the foundation of the formal diagnosis of depressive symptoms in young children.

Although the findings of this study minimize the utility of masked symptoms in diagnosing depression in young children, they also note that the presence of anhedonia should be particularly concerning, as anhedonia appears to be highly predictive of depression in young children (Luby, Heffelfinger, et al., 2003). In particular, Luby, Heffelfinger, and colleagues (2003) suggest that approximately one-half of the young children who are depressed exhibit symptoms of melancholy. Specifically, these children are experiencing anhedonia (i.e., a lack of reactivity to enjoyable activities) and psychomotor retardation. A further examination (Luby, Mrakotsky, Heffelfinger, Brown, & Sptiznagel, 2004) also indicates that anhedonia is a strong predictor of depression in young children. In other words, because anhedonia is not a typical characteristic of young children, the presence of anhedonia is highly suggestive of clinically significant depressive symptomotology. Further, the young children who exhibit anhedonia experience an even greater severity of depressive symptoms than those young children who are depressed but who do not exhibit anhedonia, have a greater family history of MDD, and exhibit more alterations in cortisol activity when introduced to stress (Luby, Mrakotsky, et al., 2004). Thus, symptoms of anhedonia may be a particularly important sign of depression in young children (Luby, 2007) and may, in fact, be the most specific symptom to indicate that young children are likely to meet the modified criteria of depression described above (Luby, Heffelfinger, Mrakotsky, et al., 2003).

Another evaluation (Luby, Sullivan, et al., 2006) of young children who are depressed examines whether more negative behaviors and less positive behaviors during observations can be indicative of symptoms of depression (i.e., beyond the information that is collected from structured interviews). Results of this study suggest that young children who are depressed (i.e., diagnosed with the modified criteria described above) can be identified through observation, further validating the use of these modified criteria (Luby, Sullivan, et al., 2006). In particular, results suggest that young children who are depressed demonstrate fewer positive and more negative behaviors when interacting with their caregivers. Further, young children who are depressed and exhibit anhedonic symptoms demonstrate the most negative behaviors (i.e., less enthusiasm, more avoidance and noncompliance) relative to young children who are depressed but do not exhibit anhedonic symptoms and relative to nonclinical young children (Luby, Sullivan, et al., 2006). Finally, young children who are depressed and who exhibit anhedonic symptoms but who do not exhibit comorbid externalizing behaviors can be distinguished from nonclinical young children based on observation alone (Luby, Sullivan, et al., 2006).

Further, during observations of dyadic tasks involving young children who are depressed and their mothers, the severity of these children's depressive symptoms account for a significant portion of their persistence, compliance, and enthusiasm. In addition, mothers' emotional support during these interactions mediates the relationship between young children's depression severity and their persistence and compliance. In other words, young children's depressive symptoms predict their mothers' emotional support, which then predicts these children's persistence and compliance (Belden & Luby, 2006). It also is worth noting that young children are more likely to include negative, sad, and dangerous themes in their play relative to nonclinical children and those who exhibit disruptive behaviors (Luby, 2007). Further, young children who are depressed experience higher levels of guilt and lower levels of guilt reparation relative to nonclinical children (Luby, 2007). Overall, these findings

suggest that young children's depressive symptoms may be critical in their interactions with other individuals.

Other factors also should be considered when diagnosing depression in young children. For example, Luby, Belden, and Spitznagel (2006) examine the role of having a family history of mood disorders and stressful life events in the occurrence of depression in 174 young children who range in age from 3- to 5.6-years. The findings of this study suggest that both a family history of mood disorders and stressful life events predict depression severity scores in young children six months later, with stressful life events serving as a mediator in this relationship (Luby, Belden, et al., 2006). Further, young children who experience depression are not likely to have temporary or spontaneously remitting symptoms. For example, Lavigne and colleagues (1998) indicate that 40 percent of 2- and 3-year old children who meet criteria for an emotional disorder are likely to continue to exhibit an emotional disorder up to four years later. Also during this time frame, 60 percent meet criteria for either a mood disorder or a disruptive behavior disorder. In addition, Luby, Heffelfinger, Mrakotsky, and colleagues (2002) suggest that the developmentally modified *DSM* criteria demonstrate six-month stability. Thus, it is likely that young children will continue to exhibit depressive symptoms once they are noted to have these symptoms.

Finally, when diagnosing young children who are depressed, it is also important to be aware of other difficulties that these children may experience. For example, young children who are depressed tend to experience other disorders as well. One study suggests that 42 percent of these children experience comorbid Attention-Deficit/Hyperactivity Disorder, 62 percent experience comorbid Oppositional Defiant Disorder, and 41 percent experience both of these disorders comorbidly (Luby, Heffelfinger, et al., 2003). Similarly, Kashani, Allan, Beck, Bledsoe, and Reid (1997) suggest that young children who are depressed tend to experience high rates of externalizing behavior problems (e.g., aggression, anger). In contrast, young children appear to experience lower rates of anxiety symptoms concurrently with their symptoms of depression relative to older children, adolescents, and adults (Egger & Angold, 2006). Researchers are beginning to speculate about the reasons that young children may be experiencing comorbid constellations of symptoms. For example, these symptoms possibly may occur as a result of their limited ability to express themselves and use coping skills (Kashani et al., 1997). It also may be the case that young children exhibit dysregulation in both their emotions and behaviors, resulting in symptoms of both internalizing and externalizing behavior disorders (Egger & Angold, 2006). Nonetheless, it is not uncommon for other disorders to co-occur with symptoms of depression in young children. As a result, mental health professionals should monitor these children for comorbid conditions.

Summary of Other Considerations

Although the last four decades have seen great changes in the acceptance and definition of childhood depression, it appears that more current conceptualizations have reached some consensus (Birmaher et al., 1996; Luby & Belden, 2006). The transition from infancy through childhood and adolescence to adulthood is a period of time marked by drastic changes in emotional and cognitive development. As a result, it makes sense that young children, school-age children, and adolescents will vary in their presentation of some depression symptoms and their ability to express their feelings. In particular, young children's experience of

anhedonic symptoms appears to be particularly important in diagnosing their symptoms of depression (Luby, Heffelfinger, et al., 2003).

Developmental considerations are beginning to be included in the criteria listed in the *DSM*, the *DSM-PC*, and the *DC: 0-3R*; however, more changes can be incorporated to make all criteria even more developmentally appropriate. For example, new research on age-appropriate diagnostic criteria for young children demonstrates that depression does in fact exist in childhood and demonstrates the great need for developmental abilities to be considered in the context of making an accurate diagnosis (e.g., Luby, Belden, et al., 2006). When age-appropriate symptom states are assessed, it becomes clear that young children who are between the ages of 3- and 6-years can experience stable and specific depressive syndromes (Luby, 2007). Thus, more research regarding diagnostic criteria for depressive disorders in children should be conducted.

ASSESSMENT AND TREATMENT OF CHILDHOOD DEPRESSION

Given the research described above, there is evidence that children as young as 3-years of age can experience clinically significant levels of depression (e.g., Luby, Heffelfinger, Mrakotsky, et al., 2002; Luby, Heffelfinger, et al., 2003). Further, the occurrence of depression in such young children is likely to have an adverse impact on their development (Luby, Heffelfinger, et al., 2003). It also is important to note that depression in young children may manifest itself in a manner different from that in adolescents and adults (Cooper, Hooper, & Thompson, 2005; Kaufman & Kaufman, 2005). Thus, accurately identifying and treating age-appropriate signs and symptoms of depression in young children is imperative. In fact, early identification of young children's difficulties can allow mental health professionals an opportunity to provide interventions at the earliest developmental point (Luby, Heffelfinger, Measelle, et al., 2002) and allow young children to return to a nonclinical developmental trajectory (Stalets & Luby, 2006).

Assessment

Traditional psychological assessment of young children focuses almost exclusively on adult informants, due to the belief that young children are developmentally unable to accurately report on their own psychological symptoms (Emslie & Mayers, 1999; Luby, Belden, Sullivan, & Spitnagel, 2007). Recent research demonstrates, however, that young children are capable of accurately reporting on their psychological symptoms when age-appropriate assessment tools are used (Luby et al., 2007; Stalets & Luby, 2006). Further, research suggests that relying solely on adult, or parental, informants is particularly problematic because parents may fail to recognize certain symptoms of depression in their young children (Kolko & Kazdin, 1993; Wu, Hoven, & Bird, 1999), as these symptoms are not robust observable behaviors (Stalets & Luby, 2006). For example, young children are more reliable in reporting on their own internalizing symptoms (e.g., depressed mood) when compared to their parents' reports (Kolko & Kazdin, 1993; Wu et al., 1999). Thus, child reporters have the potential to make a unique contribution to the identification of their

internalizing symptoms (e.g., depressed mood). Due to the unique contribution that young children make in identifying internalizing symptoms, using multiple informants is essential to the assessment of depression in young children (Kraemer et al., 2003; Wu et al., 1999). The methods most frequently used in screening and assessing depression in young children, as well as other methods that may prove helpful, are delineated below.

Rating Scales

The *Preschool Feelings Checklist* (*PFC*; Luby, Heffelfinger, Koenig-McNaught, Brown, & Spitnagel, 2004) is a 20-item parent report checklist designed specifically to screen for symptoms of depression in young children who range in age from 3- to 5.5-years. Luby, Heffelfinger, and colleagues (2004) indicate that the *PFC* is a valid measure for the identification of young children in need of clinical assessment for depression. Further, research suggests that the summary scores on the *PFC* accurately differentiate young children who are depressed from those who are experiencing other psychological disorders (Luby, Heffelfinger, et al., 2004). Thus, the *PFC* has the unique ability to screen for and identify those young children who are in need of further clinical evaluation for depression. Unfortunately, the *PFC* is one of the only valid screening measures for depression in children under the age of 6-years (Luby, Heffelfinger, et al., 2004).

The *MacArthur Health and Behavior Questionnaire* (*MHBQ*; Luby, Heffelfinger, Measelle, et al., 2002) is another measure that has been developed to assess the physical and mental health of young children. This measure has both a parent version and a teacher version and yields ratings for the functioning of young children in the domains of emotional and behavioral symptoms, physical health, social adaptation, and school adaptation. In a study examining the *MHBQ*, it is noted that the *MHBQ* identifies significantly more young children with internalizing symptoms than does the *DISC-IV*. Further, the *MHBQ* may be a more sensitive measure in that it yields higher ratings of symptoms and impairments on teacher reports relative to the *DISC-IV* (Luby, Heffelfinger, Measelle, et al., 2002).

Several other general rating scales can be helpful in the assessment of depression in young children. The *Child Behavior Checklist* (*CBCL*; Achenbach & Rescorla, 2000, 2001) is a reliable and valid self-administered rating scale used to solicit parental ratings of children's emotional and behavioral functioning. Parents complete either the 1.5- to 5-year old version or the 6- to 18-year old version, depending on the age of their children. This measure has good test-retest reliability (mean *r*'s of .85 and .88, respectively) for the preschool (Achenbach & Rescorla, 2000) and school-age versions (Achenbach & Rescorla, 2001), respectively. The *Caregiver-Teacher Report Form* (*C-TRF*; Achenbach & Rescorla, 2000) and the *Teacher Report Form* (*TRF*; Achenbach & Recorla, 2001) are versions of the *CBCL* that solicit the report of children's caregivers and/or teachers. The *C-TRF* is a valid measure of emotional and behavioral functioning in children between the ages of 1.5- to 5-years (Achenbach & Rescorla, 2000), whereas the *TRF* is used for children who range in from 6- to 18-years (Achenbach & Rescorla, 2001). Although there is a self-report measure of the *CBCL* for completion by adolescents, the *Youth Self-Report* (*YSR*; Achenbach & Rescorla, 2001), the *YSR* is not applicable to young children. Nonetheless, these measures allow for the utilization of multiple informants when examining children's emotional and behavioral functioning.

Additional empirically supported measures that utilize children's self-reports of their depressive symptoms are, at this time, applicable to school-age children. For example, the *Children's Depression Inventory* (*CDI*; Kovacs, 1985) is a reliable and valid self-report measure of children's cognitive, affective, and behavioral symptoms of depression. One study indicates that children's self-reported depressive symptoms as assessed by the *CDI* in the first grade are significantly more predictive of later child outcomes (e.g., suicidal ideation and depression) relative to adult reports of the same children's depressive symptoms. This study also suggests that the ratings provided by these children are relatively stable and related to current and future adaptive functioning (Ialongo, Edelsohn, & Kellam, 2001). Further, the *CDI* also has versions for parents and teachers to complete. In addition to the *CDI*, the *Hopelessness Scale for Children* (Kazdin, 1986) is a true/false self-report questionnaire that assesses children's hopelessness, a symptom that correlates highly with depression. Unfortunately, both the *Children's Depression Inventory* and the *Hopelessness Scale for Children* tend to be used with children who are of school-age or older. Further research should be conducted regarding the utility of these measures for young children, however.

Clinical Interviews

In addition to the rating scales described above, structured and semi-structured interviews are used commonly when assessing young children's experience of depression. One such interview is the *Kiddie Schedule for Affective Disorders and Schizophrenia – Present and Lifetime Version* (*K-SADS- PL*). The *K-SDAS-PL* is a semi-structured diagnostic interview used to assess psychopathology in children and adolescents (Kaufman et al., 1997). Although the *K-SADS-PL* is used commonly for children who are 6-years of age and older, some have used this interview for assessing the symptoms experienced by young children. This interview can be used with both children and their parents. Due to its semi-structured nature, mental health professionals have the ability to ask age-appropriate questions when assessing children's psychological symptoms. Research suggests that the *K-SADS-PL* is a valid and reliable method for assessing depression in children and adolescents according to the *DSM* criteria (Kaufman et al., 1997).

In addition to the *K-SADS-PL*, there are valid diagnostic interviews that are designed specifically for use in assessing the psychological symptoms of young children. For example, the *Diagnostic Interview Schedule for Children-Version IV-Young Child* (*DISC-IV-YC*) is a structured diagnostic interview that is administered to parents only (Lucas, Fisher, & Luby, 1998). The purpose of the *DISC-IV-YC* is to assess the psychological symptoms of preschool-age children (Lucas et al., 1998; Shaffer, Fisher, & Lucas, 1998). The *DISC-IV-YC* is a modified version of the *DISC-IV* (i.e., the version of this measure for older children and adolescents; Shafer et al., 1998) in order to account for developmental manifestations of symptoms in young children (Luby, Heffelfinger, et al., 2003). Similar to the *DISC-IV-YC*, the *Preschool Age Psychiatric Assessment* (*PAPA*) also is a structured diagnostic interview administered to parents of children between the ages of 2- and 5-years in order to assess for clinically significant symptoms (Egger & Angold, 2004). Thus, in general, mental health professionals administer each of the abovementioned diagnostic interviews (e.g., the *K-SADS-PL*, *DISC-IV-YC*, and *PAPA*) in order to gather information on the symptoms and experiences

of young children. Using a standardized process helps to improve the reliability of the information obtained.

Other Measures

The *Berkeley Puppet Interview* (*BPI*; Ablow & Measelle, 1993) is an additional assessment method that can be used with children who are 6-years of age and younger. The *BPI* is a measure of psychopathology specifically designed for children between the ages of 4.5- to 7.5-years that has minimal demands on children for expressive language. The *BPI* is a puppet interview that is a reliable and valid method of assessing young children's self-perceptions of themselves and their environment (Ablow & Measelle, 1993), including their academic, social, and emotional perceptions of themselves as well as their representations of their family environment and their relationships with their parents (Measelle, Ablow, Cowan, & Cowan, 1998). Further, the *BPI*-Symptom Scales (*BPI*-S) examines children's perceived emotional and behavioral symptoms in clinically relevant domains, including depression (Ablow & Measelle, 1993). The *BPI* engages young children with two identical hand puppets who make neutral but opposing statements (e.g., "I am a happy kid." versus "I'm not a happy kid."). The young children then are asked to choose which puppet most closely represents how they feel. Thus, the *BPI* uses age-appropriate assessment techniques to allow children to reliably report on their psychological symptoms (Ablow & Measelle, 1993).

When testing the validity of the *BPI*, Luby and colleagues (2007) report that there are significant correlations between children's self-report on core depressive symptoms and parents' report of depressive symptoms according to the *DISC-IV-YC* and the *CBCL*. Measelle and colleagues (1998) indicate that young children's reported depressive symptoms on the *BPI* also are correlated significantly with parents' and teachers' ratings of depressive symptoms. Further, children's responses on the depression-anxiety scale of the *BPI* appear to be relatively stable, with scores being correlated significantly over a one-year period from Kindergarten to First Grade (Measelle et al., 1998). Finally, the *BPI* is found to discriminate between community and clinic-referred children who range in age from 4- to 8-years old for both internalizing and externalizing disorders (Ablow & Measelle, 1993). In particular, Luby and colleagues (2002) note that young children who are depressed report more depressive symptoms on this measure than do children in a nonclinical control group.

Assessment Summary

Overall, the research conducted by Luby and colleagues (2007) suggests that young children may serve as useful reporters of their own basic symptoms of depression when age-appropriate assessment techniques are utilized. Unfortunately, there are still too few empirically validated assessment procedures for use with young children. If the measures that are available for use with young children themselves and with their parents are used in conjunction with other available tools (e.g., observations of young children's interactions with other individuals as well as observations of young children at play; Stalets & Luby, 2006), an accurate diagnosis of the difficulties that young children are experiencing in conjunction with depressive symptoms can be made.

Treatment

Several studies suggest that the depressive symptoms experienced by young children may follow a particularly severe course with a high level of relapse (Geller, Zimmerman, Williams, Bolhofner, & Craney, 2001; Kovacs, 1998). As a result, it is especially important to identify and treat depression when it first manifests itself in young children (Geller et al., 2001). Despite advances in the treatment of depression in older children and adolescents, there are still relatively few empirically supported treatments for the depressive symptoms exhibited by children who are 6-years of age and younger, however (Carr, 2008). Treatments that may be helpful in alleviating the symptoms of depression experienced by young children are described here.

Psychodynamic Psychotherapy

Traditionally, psychodynamic psychotherapy for children was the sole treatment for young children's depressive symptoms (Cooper et al., 2005). Traditional psychodynamic psychotherapy, however, is a time consuming and very expensive option. These characteristics of this therapeutic approach limit the number of children who could potentially benefit from this therapeutic approach (Cooper et al., 2005). Although traditional psychodynamic treatment is no longer the only treatment available for young children who are depressed, there are still very few options when choosing an empirically supported treatment for young children who are exhibiting clinically significant levels of depression.

Cognitive-Behavioral Therapy

One of the most common forms of treatment for depression in older children and adolescents is *Cognitive-Behavioral Therapy* (*CBT*). *CBT* focuses on elevating children's mood by challenging automatic thoughts, identifying negative thinking, and restructuring maladaptive thoughts (Kaufman & Kaufman, 2005). For example, Stark, Sweater, Jurkowski, Sommer, and Bowen (1996) describe a *CBT* program for adolescents who are depressed and their parents. This program focuses on the development of positive mood and seeks to teach adolescents to reframe their negative thought patterns. *CBT* has been modified in order to meet the needs of young children who are exhibiting clinically significant levels of depression (Kaufman & Kaufman, 2005). In particular, young children require a unique adaptation of *CBT* due to their cognitive and verbal developmental limitations (Knell, 1998). As a result, *Cognitive Behavior Play Therapy* (*CBPT*) has been adapted from traditional *CBT* programs and incorporates cognitive, behavioral, and more traditional play therapies (Knell, 1993, 1994, 1998; Knell & Moore, 1990).

In particular, *CBPT* is a developmentally based and integrated model of psychotherapy for children who are between the ages of 2.5- and 6-years (Knell, 1993, 1998). The focus of *CBPT* is to increase behavioral competence, to correct maladaptive cognitions, and/or to instill adaptive cognitions by using age-appropriate techniques (Knell, 1998). Due to young children's linguistic limitations, *CBPT* deemphasizes complex verbal approaches and uses age-appropriate items (e.g., pictures of faces depicting feelings, puppets and/or dolls that act

out feelings and behaviors; Knell, 1998). In addition, *CBPT* uses modeling as a way to acquire, strengthen, or weaken certain behaviors and as a way to demonstrate adaptive coping methods (Knell, 1994, 1998). Although *CBPT* is effective in the treatment of young children's depressive symptoms, its use as an efficacious empirically validated treatment of young children with depression is yet to be established (Knell, 1998).

Family-Focused Treatments

Family-focused treatments for childhood depression also are effective for both older children and adolescents (Carr, 2008). The efficacy of *family-focused treatments* of depression in children younger than 6-years of age has yet to be established (Carr, 2008). Despite the lack of existing literature on the familial treatment of depression in young children, it is nearly impossible to dismiss the relative importance that young children's parents play in the successful treatment of their children's symptoms (for a review see McCauley & Myers, 1992). The outcome research on *family-focused treatments* suggests that parents can be important agents for emotional and behavioral change for their children (Asarnow, Scott, & Mintz, 2002). For example, family factors predict both outcomes and treatment response among older children and adolescents with depression (Birmaher et al., 2000). Research also suggests that *family-focused treatments* are particularly helpful before adolescence, when children's primary relationships are with their families as opposed to their peers (Tompson et al., 2007). As such, *family-focused treatments* for young children may be particularly appropriate.

Psychotropic Medications

Lastly, studies assessing the use of *psychotropic medications* for the treatment of depressive symptoms experienced by young children are limited, despite evidence of increased use of psychotropic medications in this young population (Gleason et al., 2007; Zito, Safer, dosReis, et al., 2000). In general, the use of these medications with young children would be considered off-label (Stalets & Luby, 2006), with recommendations for psychopharmacological treatment of depression in young children based largely on studies conducted with older children and adolescents (Hughes, Emslie, & Crimson, 2007). Further, the impact of early exposure of young children to *psychotropic medications* is yet to be studied systematically (Gleason et al., 2007). Research demonstrates, however, that only Fluoxetine (Prozac) is beneficial over placebo for the treatment of depressive symptoms in children who are younger than 12-years of age (i.e., despite the potential side effects of selective serotonin reuptake inhibitors; Bridge, Iyenar, & Salary, 2007). Given the dearth of literature on *psychotropic medications*, more research is needed.

Treatment Summary

Overall, despite the advancements in the assessment and treatment of depression in older children and adolescents, assessment devices and empirically supported treatments for depression in young children is still relatively limited. As noted above, multiple informants should be used in the assessment of young children's depressive symptoms (Luby et al., 2007). Research shows that children can be reliable reporters of their basic depressive symptoms and are, in some cases, more accurate then their parents in reporting their own internalizing symptoms (Kolko & Kazdin, 1993; Wu et al., 1999). Thus, using the traditional approach of relying solely on parent report is less effective when compared to using parent, child, and even teacher reports.

Once properly assessed, it is important for young children to receive treatment for their depressive symptoms. Although recent research on the treatment of depressive symptoms in young children has moved away from the traditional psychodynamic treatment approach, there are still few empirically supported treatments for young children (Carr, 2008). Currently, variants of *CBT* (e.g., *CBPT*) and *family-focused treatments* are among the only treatments for young children with depression. Further, although some *psychotropic medications* decrease depressive symptoms in younger children, even the research on *psychotropic medications* for young children with depressive symptoms are based largely on older children and adolescent populations (Hughes et al., 2007). Thus, future research should focus on examining the efficacy of current treatments and expanding the variety of empirically supported treatments for depressive symptoms in young children.

CONCLUSION

Historically, psychoanalytic theories suggest that it is not possible for children to be depressed. Today, however, it is understood that young children are more sophisticated than once thought and can experience guilt and shame (Luby, 2007). Further, it is now accepted that it is not uncommon for young children to experience symptoms of depression. To diagnose depression in young children, the *DSM* diagnostic classification system is used generally. Because the *DSM* diagnostic classification system uses the same criteria for individuals of all ages, other diagnostic classification systems (e.g., the *DSM-PC* and the *DC: 0-3*) and diagnostic modifications (Luby, Heffelfinger, Mrakotsky, et al., 2002) can be used to assist in identifying developmentally appropriate descriptors for depressive symptoms in young children. When age-appropriate symptom states are assessed, it becomes clear that young children who are between the ages of 3- and 6-years can experience stable and specific depressive syndromes (Luby, 2007). As advances in diagnostic criteria are made, further advances also are needed in the assessment measures and therapeutic interventions that are available for use with young children and their families. Such advances are particularly important, as addressing the symptoms of depression experienced by young children can put them on a trajectory toward a happier and healthier life throughout their later development.

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Chapter 8

THE RELATIONSHIP BETWEEN DIMENSIONS OF PARENTING AND COGNITIVE AND INTERPERSONAL VULNERABILITY FACTORS TO DEPRESSION IN YOUTH

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INTRODUCTION

Childhood depression was once believed to be a rare or transitory developmental phenomenon (Shwartz, Gladstone, & Kaslow, 1998). Contrary to such assumptions, however, strong evidence has accumulated in recent years demonstrating the high prevalence and chronic course of childhood depression (Kessler, Avenevoli, & Merikangas, 2001). Epidemiological studies have shown that by age 14, approximately 9% of children have already experienced at least one episode of severe depression (Lewinsohn, Rohde, Seeley, & Fischer, 1993). In addition, a large proportion of children and adolescents with subsyndromal levels of depressive symptoms report significant functional impairment and seek treatment at the same or higher rates than their peers with clinically elevated levels of major depression (Kessler & Walters, 1998; Gonzalez-Tejera et al., 2005) Furthermore, the best predictor of depression in adulthood is childhood or adolescent depression (Kim-Cohen, Moffitt, Harrington, Milne, & Poulton, 2003) and youth onset of depressive episodes has been shown to be associated with chronic or recurrent episodes in adulthood (Weissman, Warner, Wichramaratne, Moureau, & Olfson, 1997). Such findings highlight the importance of identifying the factors that render certain youth vulnerable to the development of depression.

One factor that has been found to significantly increase the risk for depression in youth is parental depression (Beardslee, Versage, & Gladstone, 1998). More specifically, children with an affectively-ill parent are four to six times more likely than other children to develop

clinically significant depressive episodes (Beardslee, Keller, Lavori, Staley, & Sacks, 1993; Hammen, Burge, & Adrian, 1991; Weissman et al., 1997). Parental depression has been hypothesized to influence child depression through multiple pathways including through the mediating role of higher levels of cognitive and interpersonal vulnerability factors. Cognitive and interpersonal vulnerability-stress theories posit that individuals with pre-existing maladaptive cognitive styles are more vulnerable to developing depression when they experience negative life events. Multiple cognitive and interpersonal vulnerability factors have been proposed including dysfunctional attitudes (Beck, 1963; 1972), low self-esteem (Brown & Harris, 1986), ruminative response style (Nolen-Hoeksema, 1987), depressogenic attributional style (Abramson, Metalsky, & Alloy, 1989), the personality predispositions of self-criticism and dependency (Blatt & Zuroff, 1992), and negative attachment cognitions (Bowlby, 1969; 1980). Providing support for the applicability of such theories to youth samples, cognitive and interpersonal vulnerability factors have been found to prospectively predict increases in depressive symptoms in youth following the occurrence of negative events (Abela & Hankin, 2008; Hankin & Abela, 2005).

Numerous studies have shown that high-risk children (i.e., those with a depressed parent) possess higher levels of cognitive and interpersonal vulnerabilities to depression than do low-risk children (i.e., those with non-depressed parent; Jaenicke et al., 1987; Taylor & Ingram, 1999). More specifically for cognitive vulnerabilities, children of mothers with a history of unipolar depression have been found to possess more negative cognitions about themselves than children of medically-ill parents or controls (Jaenicke et al., 1987). Similarly, high-risk children have been found to report lower levels of self-esteem (Hirsch, Moos, & Reischl, 1985) and more negative cognitive styles (Garber & Robinson, 1997) than normal controls. Such differences in cognitive vulnerabilities have been found to remain even after controlling for children's current levels of depressive symptoms (Garber et al., 1997). With respect to interpersonal vulnerabilities, children of depressed mothers have been found to exhibit attachment styles characterized by greater insecurity as well as higher levels of interpersonal dependency (Abela, Hankin, Haigh, Adams, Vinokuroff, & Trayhern, 2005).

One way in which parental depression has been hypothesized to lead to higher levels of cognitive and interpersonal vulnerability to depression in youth is through the mediating role of parenting processes. Parental depression has been found to negatively impact parenting style (Cummings & Davies, 1994; Gerlsman, Emmelkamp, & Arrindell, 1990). For example, parental depression has been found to be associated with lower levels of warmth and acceptance as well as with higher levels of control in parents (Alloy, Abramson, Tashman, Berrebbi, Hogan, Whitehouse et al., 2001). Depressed mothers have been found to be less responsive to their children (Cox, Puckering, Pound, & Mills, 1987; Goodman & Brumley, 1990) and more critical (Conrad & Hammen, 1993) than non-depressed mothers. Depressed mothers have also been found to exhibit lower levels of involvement with their children (Weissman & Pykel, 1974). Last, greater severity of parental mental illness has been found to be related to more negative parenting practices (Kokes, Harder, Fisher, & Strauss, 1980).

Negative parenting practices, in turn, have been found to be associated with higher levels of depressive cognitions in children (Bruce, Cole, Dallaire, Jacquez, Pineda, & LaGrange, 2006; Garber & Flynn, 2001; Jaenicke et al., 1987; Mezulis, 2005; Whisman & Kwon, 1992). More specifically, lower levels of parental support and higher levels of parental rejection and over involvement, have been found to be associated with lower levels of self-esteem and more depressogenic attributional style in children (Tiggemann, Winefield, Goldney, &

Winefeld, 1992). Similarly, higher levels of maternal restrictiveness and rejection have been found to prospectively predict increases in children's levels of self-criticism even after controlling for children's temperament (Koestner, Zuroff, & Powers, 1991). Lower parental care during childhood has been found to predict higher levels of depressive symptoms in young adulthood through the mediating role of depressotypic attitudes and attributions (Whisman et al., 1992). Last, Bruce and colleagues reported that negative parenting and negative life events were associated with higher levels of depressive cognitions in children, while positive parenting was associated with lower levels of depressive cognitions in children (Bruce et al., 2006). The relationship between negative parenting and negative cognitions was stronger for older children (Bruce et al., 2006). Thus, overall, there appears to be an association between negative parental practices and the development of vulnerabilities in children.

The goal of the present study was to examine whether parental depressive symptoms predict child cognitive and interpersonal vulnerability to depression through the mediating role of parenting. Although several studies have examined the relationships between (1) parental depressive symptoms and parenting processes, (2) parental depressive symptoms and child vulnerabilities, and (3) parenting processes and child vulnerabilities, to our knowledge, no studies have examined whether impaired parenting *mediates* the relationship between parental depressive symptoms and child cognitive and interpersonal vulnerabilities. Participants included 140 children between the ages of 6 and 14 with a parent with a history of major depressive episodes. During an initial assessment, parents completed measures assessing parenting styles and current depressive symptoms, while children completed measures assessing depressive symptoms, cognitive vulnerability factors (e.g., depressogenic attributional style, response style, dysfunctional attitudes, dependency and self-criticism) and interpersonal vulnerability factors (e.g., insecure attachment). The following hypotheses were examined: (1) higher levels of depressive symptoms in parents will be associated with higher levels of cognitive and interpersonal vulnerabilities in children; (2) higher levels of depressive symptoms in parents will be associated with parenting styles characterized by lower levels of nurturance, consistency and responsiveness, and higher levels of restrictiveness; (3) lower levels of parental nurturance, consistency and responsiveness and higher levels of parental restrictiveness will be associated with higher levels of child cognitive and interpersonal vulnerabilities; (4) after controlling for the proportion of variance in child cognitive and interpersonal vulnerabilities accounted for by parenting style, parental depressive symptoms will no longer be significantly associated with children's cognitive and interpersonal vulnerabilities

METHODS

Participants

Participants were recruited in two phases. In the first phase, advertisements were placed in several English newspapers, as well as posters across the greater Montreal area. The ads and posters called for parents with a history of past depressive episodes who had children between the ages of 6 and 14-years old. One hundred and ninety five people responded to

these advertisements. These respondents were invited to participate in a telephone interview during which a trained diagnostician administered the affective disorders module of the Structured Clinical Interview for the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders: DSM-IV-Axis I (SCID-I; First, Spitzer, Gibbon, & Williams, 1995), in order to assess their past history of mental disorders. Out of the one hundred and seventeen parents who met criteria for either a current or a past major depressive episode, eighty-six parents with one hundred and twenty two children decided to participate.

In the second phase, diagnosticians called 55 parents who had participated in a previous study in our lab and also had a child who met the age criteria and invited them to participate in the current investigation. All of the contacted parents agreed to participate and completed the telephone interview. Fourteen of the 16 parents who met criteria decided to participate.

Thus, the final sample consisted of 140 (71 girls and 69 boys) high-risk children and one of their parents (88 mothers and 14 fathers). In all cases, the parent who participated was the same parent that completed the telephone interview and met criteria for either a current or past major depressive episode. Parents' ages ranged from 27 to 53-years (M=40.3, SD=6.4, Median=41) and children's ages ranged from 6 to14-years (M=9.8, SD=2.3, Median=10). Of the parents, 14.7% were single (n=21), 43.1% were married (n=60), 9.8% were separated (n=14), 27.5% were divorced (n=38), 1% was widowed (n=1) and 3.9% were none of the above (n=6). Participants' mother tongues included English for 68.7% (n=70), French for 9.8% (n=10), Spanish for 2.9% (n=3), and other languages for 18.6% (n=19). All children were fluent in English. The median per annum family income ranged from $30,000 to $45,000. The sample was ethnically representative of the English speaking Montreal community and consisted of 84.3% Caucasians (n=118), 4.9% Asians (n=7), 2.9% Hispanic (n=4), 1.9% African American (n=1), 1.1% Native American (n=1) and 4.9% of other descent (n=7). In regards to parents' level of education, 7.8% possessed an elementary school diploma, 14.7% a high school diploma, 39.3% a community college diploma, 22.5% a bachelor's degree and 15.7% a graduate degree.

Procedure

For the initial assessment, parent and child pairs came to the lab at McGill University. Participants were informed that their participation was voluntary and that they could choose to withdraw at any time during the course of the study. All parents and children decided to participate, and completed both consent and demographic forms. Subsequently, a trained research assistant verbally administered a series of questionnaires to the child, including: 1) Children's Attributional Style Questionnaire (CASQ; Seligman, Peterson, Kaslow, Tenenbaum, Alloy, & Abramson, 1984); 2) Children's Self-Esteem Questionnaire (CSEQ; Rosenberg, 1965); 3) Children's Response Styles Questionnaire (CRSQ; Abela, Brozina, Haigh, 2002); 4) Children's Depressive Experiences Questionnaire (CDEQ; Abela & Taylor, 2003); 5) Children's Dysfunctional Attitudes Scale (Abela & Sullivan, 2003); and 6) Inventory of Parent and Peer Attachment (IPPA; Armsden & Greenberg, 1987). While children were completing these questionnaires, parents independently completed the Parenting Dimensions Inventory (PDI; Slater & Power, 1987); and Beck Depression Inventory (BDI; Beck, Ward, Mendelson, & Mock, 1961). Families were compensated $60 for their participation.

Measures

The Structured Clinical Interview for the DSM-IV (SCID-I; First et al., 1995). The SCID-I is a semi-structured clinical interview designed to arrive at a current and lifetime DSM-IV diagnosis. The current study employed the mood disorders module and psychotic screen for the diagnosis of current and past history of DSM-IV mood disorders in parents. The psychotic screen was used to eliminate any parent with current or past psychotic symptoms. The SCID-I has been shown to provide reliable diagnoses of depressive disorders (Zanarini, Skodol, Bender, Dolan, Sanislow, Schaefer et al., 2000).

Diagnosticians completed an intensive training program for administering the SCID-I and for making DSM-IV diagnoses. Training consisted of approximately 40 hours of didactic instruction, listening to audio taped interviews, conducting practice interviews and passing a diagnostic exam with a minimum grade of 85%. Finally, three audio taped interviews were coded and diagnosticians were required to show 100% agreement with the principle investigator as to the presence or absence of symptoms and at least 85% agreement on severity ratings of present symptoms. In addition, weekly supervised sessions were held with the principle investigator where interview notes were reviewed and any discrepancies regarding symptom presence or severity were addressed.

Children's Attributional Style Questionnaire (CASQ; Seligman et al., 1984). The CASQ is a forced-choice self-report questionnaire with 48-items, each consisting of a positive or negative hypothetical event and two potential causes of the event. Children are instructed to choose the option that best describes how they would think if that particular event happened to them. The possible responses for each item hold constant two attribution dimensions (global-specific, stable-unstable, internal-external) while varying the third. CASQ scores range from 0 to 16 where a score of 1 is given to each global, stable and internal response and a score of 0 is given to specific, unstable or external response. As the hypotheses of the current study involve attributional styles for negative events, only negative event items were administered. CASQ scores have been shown to be fairly consistent over time indicating that attributional style is a stable individual difference among children (Seligman et al., 1984). Test-retest reliability and internal consistency of the negative events scale have been found to be in the moderate range (Gladstone & Kaslow, 1995). Furthermore, predictive validity has been confirmed for depression severity above and beyond initial levels by several studies (Abela, 2001; Hilsman & Garber, 1995).

Children's Self-Esteem Questionnaire (CSEQ; Rosenberg, 1965). The SEQ is a 10-item self-report questionnaire. Participants are asked to rate the degree to which they agree with 10 statements of self perception, with total score ranging from 0 to 30, where 30 corresponds to the lowest level of self-esteem. The SEQ has been found to have high levels of test-rest reliability (Allgood-Merten, Lewinsohn, & Hops, 1990), as well as to posses both convergent and discriminant validity (Silber & Tippett, 1965).

Children's Response Style Questionnaire (CRSQ; Abela et al., 2002). The CRSQ consists of 25-items that describe a specific response to symptoms of depression. The items are grouped into three dimensions 1) rumination, 2) distraction, and 3) problem solving. As the hypotheses of the current study focused solely on rumination, all distraction and problem solving items were omitted from the current analyses. The rumination subscale includes 13-items that describe individual's responses to depressed mood that are self focused. Scores range from 0 to 39 where a score of 39 indicates a greater tendency to ruminate in response to

a depressed mood. The rumination subscale of the CRSQ has been found to have high internal consistency in third through seventh grade children (Abela, Aydin, & Auerbach, 2007; Abela, Brozina, Haigh, 2002; Abela, Vanderbilt, & Rochon, 2004). Furthermore, the rumination subscale has been shown to be positively correlated with depressive symptoms (Abela et al., 2002, 2004, 2007) and to predict increases in depressive symptoms over time (Abela et al., 2002, 2007).

Inventory of Parent and Peer Attachment (IPPA; Armsden et al., 1987). The IPPA is an 18-item self-report questionnaire that assesses positive and negative cognitive/affective dimensions (e.g., "internal working models') of children's relationships with their parents and close friends. The dimensions of attachment assessed include 1) degree of mutual trust, 2) quality of communication and, 3) extent of anger and alienation. Children rate each item on a 5-point Likert scale. Total scores range from 0 to 48 with higher scores indicating higher levels of insecure attachment. As the current study focused on parent-child attachment, we only utilized the 12-items assessing attachment to parents. The IPPA has shown internal consistency, strong test-retest reliability and good validity (Armsden et al., 1987; Crowell, Fraley, & Shaver, 1999). High scores on parent attachment subscales have been found to be associated with lower scores on nurturance, responsiveness and consistency attachment styles of the Parenting Dimensions Inventory in both younger and older children (Abela, Zinck, Kryger, Zilber, & Hankin, 2009).

Children's Depressive Experiences Questionnaire (CDEQ; Abela & Taylor, 2003). The CDEQ is a 20-item measure that assesses dependency and self-criticism. For each item, children are asked to indicate whether the statement is *not true*, *sort of true*, or *really true* for them. Composite scores on both the self-criticism (CDEQSC) and dependency (CDEQD) subscales range from 0 to 20, with higher scores indicating higher levels of self-criticism or dependency. The CDEQ has been found to possess moderate levels of internal consistency in third through seventh grade children (Abela, Sakellaropoulo, & Taxel, 2007; Abela & Taylor, 2003). CDEQ scores have been shown to positively correlate with depressive symptoms (Abela et al., 2007; Abela & Taylor, 2003) and to predict increases in depressive symptoms over time (Abela et al., 2007; Abela & Taylor, 2003).

Children's Dysfunctional Attitudes Scale (CDAS; Abela & Sullivan, 2003). The CDAS is a 40-item self-report questionnaire designed to assess dysfunctional attitudes in children. The child indicates how much each statement applies to them on a 4-point Likert scale (e.g., *never true*, *just sometimes true, most of the time true,* and *always true*). Each item is given a score from 0 to 3 with total scores ranging from 0 to 120 where higher scores correspond to higher levels of dysfunctional attitudes. Past research using the CDAS has reported high levels of internal consistency in middle school children (Abela & Skitch, 2007; Abela & Sullivan, 2003). In addition, scores have been found to positively correlate with depressive symptoms and to predict increases in depressive symptoms over time (Abela Skitch, 2007; Abela & Sulivan, 2003).

Parenting Dimensions Inventory (PDI; Slater et al., 1987). The PDI is a 26-item self-report questionnaire that assesses four dimensions of parenting: nurturance, responsiveness, non-restrictive attitudes and consistency. For each item, parents are asked to rate the degree to which the statement reflects their parenting style. Respondent's scores range from 1 (*not at all descriptive of me*) to 6 (*highly descriptive of me*). Regarding validity, parents of children with high levels of social competency and low levels of behavior problems reported more nurturing, more sensitive, less restrictive, and more consistent parenting styles than the

parents of children exhibiting low levels of social competency and/or high levels of behavior problems (Slater et al., 1987). The PDI dimensions of nurturance, responsiveness, non-restrictive attitudes and consistency has been found to exhibit moderate levels of internal consistency (Slater et al., 1987).

Beck Depression Inventory (BDI; Beck et al., 1961). The BDI is a 21-item self-report instrument designed to asses the severity of depressive symptoms within the last 2 weeks. In the current study, the BDI was used to assess parents' current level of depressive symptoms. Respondents' scores are rated on a 0 to 3 scale with higher scores indicating more severe symptoms. Total scores range from 0 to 63. The BDI's test-retest correlations have been shown to be stable, ranging from .60 to .86 (Beck, Steer, & Garbin, 1988). Past research using the BDI has reported high internal consistency with alphas ranging from .89 to .93 (M=.91) (Abela, Webb, Wagner, Ho & Adams, 2006).

Table 1. Means, Standard Deviations, and Intercorrelations between All Measures

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. BDI	-								
2. PAR	-.38***	-							
3. CASQ	.32***	-.31***	-						
4. SEQ	.24**	.04	.29***	-					
5. CRSQ	.10	-.03	.31***	.25**	-				
6. CDEQS	.34***	-.31***	.38***	.35***	.24**	-			
7. CDEQD	.24**	.01	.06	.20*	.29**	.27**	-		
8. CDAS	.29**	.03	.33***	.30**	.30**	.57***	.36***	-	
9. IPPA	.21*	.25*	.44***	.23**	.24**	.37**	-.04	.14	-
Mean	19.07	118.58	4.34	10.83	15.67	8.28	10.39	38.69	27.66
SD	12.40	14.95	2.37	4.52	7.80	3.43	3.54	15.69	8.09

Note. BDI = Beck Depression Inventory. PAR = Dimensions of Parenting Inventory. CASQ = Children's Attributional Style Questionnaire. SEQ = Self-Esteem Questionnaire. CRSQ = Children's Response Styles Questionnaire, Rumination subscale. CDEQS = Children's Depressive Experiences Questionnaire, Self-criticism subscale. CDEQD = Children's Depressive Experiences Questionnaire, Dependency subscale. CDAS = Children's Dysfunctional Attitudes Scale. IPPA = Inventory for Parent and Peer Attachment.

* $p < .05$. ** $p < .01$. *** $p < .001$.

RESULTS

Descriptive Data

Means, standard deviations, and intercorrelations between all measures are presented in Table 1.

Overview of Mediational Analyses

In order to examine the hypothesis that parental depressive symptoms would predict children's cognitive and interpersonal vulnerability through the mediating role of parenting, linear regression analyses were used. According to Baron and Kenny, there is a logical sequence involved in testing a mediational model (Baron & Kenny, 1986). Following this logic, if parental depressive symptoms predict children's cognitive and interpersonal vulnerabilities to depression through the mediating role of impaired parenting, then the following conditions must be true: (1) higher levels of parental depressive symptoms will be associated with higher levels of child cognitive and interpersonal vulnerability; (2) higher levels of parental depressive symptoms will be associated with greater impairment in parenting; (3) greater impairment in parenting will be associated with higher levels of child cognitive and interpersonal vulnerability; and (4) after controlling for the effect of parenting on child cognitive and interpersonal vulnerability, parental depressive symptoms will no longer significantly predict child cognitive and interpersonal vulnerability. In condition four, if the effect of parental depressive symptoms is entirely eliminated, the results provide evidence for full mediation. If, however, in condition four, the effect of parental history of depression is significantly reduced, but not entirely eliminated, the results provide evidence for partial mediation.

Mediational Analyses

Regarding condition one, and in line with our first hypothesis, higher levels of parental depressive symptoms were significantly associated with higher levels of the following child cognitive and interpersonal vulnerability factors: depressogenic attributional style ($t(133) = 3.64$, $p < .001$), low self-esteem ($t(133) = 2.89$, $p < .01$), dependency ($t(130) = 2.76$, $p < .01$), self-criticism ($t(131) = 4.02$, $p < .001$), dysfunctional attitude ($t(118) = 3.16$, $p < .01$) and negative attachment cognitions ($t(128) = 2.31$, $p < .05$). At the same time, however, higher levels of parental depressive symptoms were not significantly associated with child rumination ($t(132) = 1.12$, *ns*).

Regarding condition two, and in line with the second hypothesis, higher levels of parental depressive symptoms were significantly associated with greater impairment in parenting practices ($t(132) = -4.09$, $p<.001$).

Regarding condition three, and in line with the third hypothesis, greater impairment in parenting practices was significantly associated with higher levels of the following child cognitive and interpersonal vulnerability factors: depressogenic attributional style ($t(136) = -$

3.58, $p < .001$), self-criticism ($t(134) = -3.19$, $p<.05$) and negative attachment cognitions ($t(131) = -2.658$, $p<.01$). At the same time, however, parenting practices were not significantly associated with child low self-esteem ($t(136) = .48$, *ns*), rumination ($t(135) = -.27$, *ns*), dependency ($t(134) = .12$, *ns*), and dysfunctional attitude ($t(121) = -.18$, *ns*).

As mediation analysis requires that condition one to three be met prior to examining condition four, these analyses were only conducted for the following child cognitive and interpersonal vulnerabilities: depressogenic attributional style, self-criticism, and negative attachment cognitions.

After controlling for the proportion of variance in child attributional style accounted for by impairment in parenting, parental depressive symptoms remained a significant predictor ($t(131) = 2.82$, $p < .01$). At the same time, controlling for impairment in parenting reduced the effect of parental depressive symptoms on child attributional style by 37.70%. This reduction was statistically significant (sobel's $t = -2.19$, $p < .05$). Thus, the association between parental depressive symptoms and child attributional style was partially mediated by impairment in parenting.

After controlling for the proportion of variance in child self-criticism accounted for by impairment in parenting, parental depressive symptoms remained a significant predictor ($t(129) = 3.21$, $p<.01$). At the same time, controlling for impairment in parenting reduced the effect of parental depressive symptoms on child self-criticism by 24.73%. This reduction was statistically significant (sobel's $t = -2.26$, $p < .05$). Thus, the association between parental depressive symptoms and child self-criticism was partially mediated by impairment in parenting.

After controlling for the proportion of variance in child negative attachment cognitions accounted for by impairment in parenting, parental depressive symptoms no longer were a significant predictor ($t(129) = 3.21$, $p<.01$). Controlling for impairment in parenting reduced the effect of parental depressive symptoms on child negative attachment cognitions by 40.30%. However, this reduction was not statistically significant (sobel's $t = -1.32$, *ns*). Thus, the association between parental depressive symptoms and child negative attachment cognitions was fully mediated by impairment in parenting.

DISCUSSION

Overall, the results of the current study provide support for our hypothesis that impaired parenting mediates the relationship between parental depressive symptoms and child vulnerabilities. These findings suggest that children with depressed parents may be more likely to develop both cognitive and interpersonal vulnerability factors due to the impaired quality of their parent's parenting practices. However, the strength of the relationship between parental depressive symptoms, parenting style and children's cognitive and interpersonal vulnerabilities varied depending on the specific vulnerability being analyzed.

Several findings warrant additional attention. First, in line with past research (Abela et al., 2005), the results of the current study indicated that higher levels of parental depressive symptoms were significantly associated with higher levels of both cognitive and interpersonal vulnerability factors in children. This finding extended to the majority of the vulnerabilities examined in the current study including depressogenic attributional style, low self-esteem,

dependency, self-criticism, dysfunctional attitude and insecure attachment. These results suggest that one possible mechanism that may account for higher levels of depression among high-risk children is higher levels of cognitive and interpersonal vulnerability. Specifically, these children are more likely to engage in maladaptive ways of interpreting negative life events and are subsequently more susceptible to developing depressive symptoms. At the same time, however, parental depressive symptoms were not significantly associated with rumination in children, suggesting that a ruminative response style in children was not influenced by the level of parental depressive symptoms.

Second, in line with past research (Cummings et al., 1994; Gerlsman, et al., 1990) higher levels of parental depressive symptoms were found to be significantly associated with greater impairment in parenting practices. More specifically, higher levels of parental depressive symptoms were significantly associated with a parenting style characterized by lower levels of nurturance, consistency and responsiveness, and higher levels of restrictiveness. The current study, due to the small sample size, focused on a composite score of parenting practices, however, there is some indication in the literature that various sub-dimensions of parenting are differentially associated with childhood depression (Mcleod, Weisz, & Wood, 2007). Hence, additional research is needed to examine which specific dimensions of parenting are most strongly associated with the development of vulnerability factors to depression in youth. At the same time, it is also possible that different dimensions of parenting are connected to different types of cognitive and interpersonal vulnerability factors.

Third, greater impairment in parenting practices were found to be significantly associated with higher levels of cognitive and interpersonal vulnerability factors in children. More specifically, higher levels of impaired parenting were significantly associated with a more depressogenic attributional style, higher levels of self-criticism, and more negative attachment cognitions in children. Such a finding is in line with a the growing body of literature suggesting that impairment in parenting practices is one of the pathways that likely leads to the development of cognitive and interpersonal vulnerability factors to depression in youth. For example, Mezulis and colleagues found that negative parental feedback, especially maternal anger expression, interacted with negative events to predict increased depressogenic attributional style in a sample of 11-year olds (Mezulis, Hyde, & Abramson, 2006). At the same time, it is important to note that we did not find a relationship between impaired parenting and dependency, a ruminative response style or self-esteem, suggesting that multiple pathways likely lead to the development of cognitive and interpersonal vulnerability factors in youth. Future research will likely benefit from examining a multitude of pathways hypothesized to lead to the development of cognitive and interpersonal vulnerability factors including emotional abuse (Gibb & Abela, 2008), the occurrence of negative events (Rose & Abramson, 1992), and experiencing periods of sub-threshold depressive symptoms (Nolen-Hoeksema, Girgus, & Seligman, 1992).

Last, expanding on past research, the results of the current study suggest that the association between parental depressive symptoms and child attachment cognitions was fully mediated by parenting practices. More specifically, after controlling for the proportion of variance in child attachment cognitions accounted for by parenting, it was found that the effect of parent's depressive symptoms on child attachment cognitions was no longer significant. At the same time, the association between parent's depressive symptoms and their child's levels of self-criticism and depressogenic inferential styles was only partially mediated by parenting practices. In other words, after controlling for the effect of parenting

practices, the associations between parental depressive symptoms and children's levels of self-criticism and depressogenic inferential styles were significantly reduced but not entirely eliminated. This is consistent with the fact that many pathways may lead from the presence of depressive symptoms in parents to the development of vulnerabilities in children. Furthermore, parenting may be the primary mediator for some but not all vulnerabilities in children. Other possible primary mediators in the association between parental depressive symptoms and child vulnerability may include experiences with depression and high stress levels.,

Several limitations of the current study should be noted. First, self-report measures were utilized to assess both depressive symptoms and parenting practices. Although these questionnaires show high levels of reliability and validity, future research would benefit from using clinical interviews and multi-informant procedures including observational measures of parenting. Second, the sample size for this study was relatively small. This limits statistical power and may account for why no significant relationship between parental depressive symptoms and children's ruminative response style was found. In addition, the relatively small sample size restricted the number of analyses that were conducted, particularly limiting the analysis to global composites. Future research with larger sample sizes would enable mediational analyses of specific dimensions of parenting. Finally, the overwhelming majority of the parents in this study were mothers, thus it is unclear whether these results can be viewed as generalizable to parents or if they are only applicable to maternal parenting practices. Lastly, this study utilized a cross sectional design, future research is needed to examine our hypotheses using a prospective design.

In sum, the results of the current study suggest that the relationship between parental depressive symptoms and children's cognitive and interpersonal vulnerabilities, specifically attributional style, self-criticism and insecure attachment is mediated by impaired parenting practices. As more research emerges that examines potential mediators of the relationship between parental depressive symptoms and children's vulnerabilities, a better understanding of the developmental origins of factors that put children at risk for depression is expected to materialize.

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Chapter 9

DEPRESSION VULNERABILITY IN MIDDLE AND LATE CHILDHOOD AND ITS RELATIONSHIP WITH GENDER, SELF COMPETENCE, AND COPING

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ABSTRACT

Depression vulnerability in children seems to be connected with cognitive and interpersonal vulnerability, like pessimistic thinking and insecure attachment to their parents (DesJardin, 2003; Richaud, 2006a). Negative self-perceptions are believed to result from the negative competency evaluations of significant others, such as parents and teachers. A child's self-perception of competency may interact with others' appraisals to influence vulnerability to depression (Richaud, 2006a). According to DesJardin (2003) children who are vulnerable to depression have a tendency to make pessimistic remarks; have low self-esteem; poor coping strategies such as chewing over their problems without solving them; rigid and extreme personality traits that include self-criticism and over-dependency on their parents. At the same time, although clear gender differences in depression only appear after adolescence, developmental perspectives have placed its origins in childhood socialization. Relatively little is known, however, about continuities and discontinuities between childhood and adult behavior.

The aim of this chapter is to study vulnerability to depression in normal boys and girls in middle and late childhood, and its relation with cognitive vulnerability, as expressed in children's self-perception of academic and social competency, controllability of perceived stressors, and coping strategies.

Method Argentine adaptation of Harter's Self-Perception Profile for Children, Argentine adaptation of Harter and Nowakowski's Dimensions of Depression Profile for children and adolescents, and Coping Questionnaire for children were administered to

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1,050 middle class children, aged 8 to 12, from four primary schools, two state-run and two public, residing in the city of Buenos Aires and various towns in the province of Buenos Aires.

Results Cognitive vulnerability expressed as negative self-perception of academic and social competence is a factor of risk that may offset depressive vulnerability in middle and late childhood. This cognitive vulnerability is also connected with threat appraisal related to close-family interpersonal difficulties. Coping with these difficulties follows a pattern characterized by non-problem focus, low approach, high avoidance and emotion-focused coping. Finally, greater gender differences in vulnerability to depression appear in middle childhood and diminish in late childhood, males being more vulnerable to depression than girls, probably due in part to changes in socialization practices.

INTRODUCTION

Major depressive disorder often begins during adolescence. Between 20% and 50% of adolescents report experiencing subsyndromal levels of depression (Kessler, Avenevoli, & Merikangas, 2001). Adolescence, then, represents a critical period of vulnerability. Patterns of depressive symptoms may differ over the course of development given the occurrence of cognitive, social, emotional, and biological changes (Weiss & Garber, 2003). In addition, the observed gender difference in depression emerges in early adolescence (Angold, Erkanli, Silberg, Eaves, & Costello, 2002*)*.

If from a developmental and cognitive science framework, cognitive vulnerability is related to the beginning of depression among youth, we expect children to be capable of demonstrating maladaptive cognitions, which, in interaction with the stress of the adolescent transition, result in depressive disorders.

Depression vulnerability in children seems to be connected with cognitive and interpersonal vulnerability, such as pessimistic thinking and insecure attachment to their parents (DesJardin, 2003; Richaud de Minzi, 2006a). Muris et al. (2001) assert that negative parental rearing behavior and a negative attribution style would be risk factors for developing depression, whilecoping styles and self-efficacy would play a mediating role in the formation of depressive symptoms.

DEPRESSION VULNERABILITY AND ATTACHMENT

The purpose of attachment behavior is to achieve proximity to one person in particular, in order to feel secure and thus deactivate the attachment system (Hinde, 1997) and allow for the activation of other important behavioral systems, such as the exploratory and sociable systems (Dwyer, 2005).

It has been shown that caregivers who respond positively to children's demands, increase the latter's commitment to social relations, and their desire to learn and comply with the norms of their social world (Ainsworth, Bell, & Stayton, 1974; Kochanska, 1993). In this way, they foster the development of social competence, which is essential for self-control and cognitive development (Richaud de Minzi, 2006a). Richaud de Minzi (2006a) has found that lack of father availability and reliance show the greatest connection with depression in middle and late childhood.

DEPRESSION VULNERABILITY AND COGNITIVE VULNERABILITY

Beck (1967) defined cognitive vulnerability as the presence of maladaptive self-schemes reflecting helplessness and non-lovability, which become activated by negative life events or negative moods. In fact, stressful life experiences predict depression among children and adolescents (Grant et al., 2004). Apparently it is a bidirectional relationship, as depressive symptoms also predict increases in objectively assessed stressors among young people (Grant et al., 2004). In view of the fact that exposure to mild uncontrollable stress during adolescence can impair cognitive functioning, it is crucial to assess stress in the study of cognitive vulnerability (Jacobs, Reinecke, Gollan, & Kane, 2008).

Negative self-perceptions regarding competence may serve as a cognitive vulnerability factor for depression (Cole, 1990). They are believed to result from negative competency evaluations by relevant others, such as parents and teachers. A child's self-perception of competency may interact with others' appraisals to influence depression.

Richaud (2006a) has found that in middle and late childhood, parent attachment is related to academic competence. The style of relationship with parents, on the other hand, accounts for social competence slightly more significantly than for academic competence. From the point of view of parental styles, acceptance and commitment by both parents, and especially the father, are very important for the child's academic achievement. In both cases — attachment and style of relationship — children allot importance to their confidence in the parent's love and his/her concern for them.

As to perceived social acceptance (social self-competence), mother availability and acceptance constitute a solid basis from which the child can dare approach others, whereas pathological control by the father seems to inhibit the child's perception of social acceptance, since threats of punishment and expressions of concern about the child's inadequate performance make him/her feel rejected by others (Richaud, 2006a).

COPING AND STRESSORS

According to DesJardin (2003) children who are vulnerable to depression tend to make pessimistic remarks, have low self-esteem, and deploy poor coping strategies, such as grumbling about their problems without solving them, as well as rigid and extreme personality traits that include self-criticism and over-dependency on their parents.

A maladaptive coping style is a significant risk factor for psychological development in children and adolescents (Compas et al., 2001; Seiffge-Krenke, 1995; Wolchik & Sandler, 1997).

Previous studies in children and adolescents found that problem-focused and approach coping were negatively related to depression, whereas emotion-focused and avoidance coping were positively correlated (Compas et al., 1988; Causey & Dubow, 1992). Furthermore, it was noted that adolescent depression was predicted by low approach and high avoidance coping (Seiffge-Krenke & Klessinger, 2000; Seiffge-Krenke & Stemmler, 2002). Further research suggested that if stressors were perceived as controllable, the efficiency of coping strategies increased (Compas et al., 1988). In studies with children and adolescents, academic stressors were perceived to be more controllable that interpersonal stressors (Causey &

Dubow, 1992; Compas et al., 1988). These findings support the hypothesis that the efficiency of coping is determined both by the coping strategies employed and the perceived controllability of stressors (Boekaerts & Roder, 1999; Causey & Dubow, 1992; Compas et al., 2001). Apparently children and adolescents employ more emotion-focused coping strategies with interpersonal stressors, and more problem-focused coping strategies with academic stressors (Hampel & Petermann, 2005).

GENDER DIFFERENCES

Although clear gender differences in depression only appear after adolescence, developmental perspectives have placed its origins in childhood socialization. Relatively little is known, however, about continuities and discontinuities between childhood and adult behavior.

According to developmental psychology, there are learning principles of socialization that account for gender differences in vulnerability to depression. It posits that sex-stereotypical socialization practices by caregivers lead to gender differences in depressive vulnerability. Ruble et al. (1993) hypothesize that parents' expectations for girls and boys differ. Among other notions, girls are expected to be more nurturing and concerned with social evaluations of others, while boys are expected to be more autonomous. Consequently, they hold, stereotypical gender socialization leads to a lower sense of mastery and control and a higher concern for external evaluation in girls than in boys (Blehar & Oren, 1999).

Although developmental learning theories view socialization agents that predominantly influence child behavior and gender differences as the outcome of primarily unidirectional processes, other evidence witnesses the inheritance of stable temperamental traits that impact on caregiver/child interaction and prompt characteristic responses (Kagan, Reznick, & Snidman, 1988).

According to Nolen-Hoeksema (1995) women's increased vulnerability to depression is based on the identification of a self-focused coping style in response to a depressed mood. Although she found relatively weak support for overall gender differences in personality characteristics of passivity and assertiveness, she did perceive differences in women's response to depression: they face depression with negative emotions while men use distracting responses (Nolen-Hoeksema, 1995). A woman's typically longer and more severe depressive episodes are generally linked to "ruminative style" and higher rates of depression in adolescent girls are triggered by their greater exposure to concerns about personal appearance, safety, and self-worth. Other research indicates that women experience more life events than men (Kessler & McLeod, 1984). Karp and Frank (1995) report more life events 6 months before the onset of a depressive episode in women than in men in a treated group and suggest that women experience higher rates of reactive depression than men.

Richaud de Minzi (2006b) found that girls had made wider use of coping strategies such as self-blame, fatalism, instrumental support, emotional release and seeking emotional support than boys. The latter, on the other hand, were more prone to turn to evasion through physical activity when facing difficult situations. It is clear, then, that all the strategies chosen by females are emotional and basically depressive, since they take responsibility for failures, they have no expectations as to any contingency between what they do and what they achieve

and they cannot control their emotions. These findings confirm Nolen-Hoeksema's statement that girls focus on negative emotions while men use distracting responses to cope with stressors. Compas, Orosan and Grant (1993) posited that this is due to the fact that there are gender differences in emotion-focused coping that also come up in adolescence.

Two adaptive strategies such as Seeking for instrumental and emotional support, however, might point at a relative lessening of these girls' depressive feelings. This would indicate that they do not feel socially isolated, which is one of the most risky strategies in adolescence (Richaud, 2006b).

Moreover, Ohannessian et al. (1999) have found that 11–12 year-old boys reported significantly higher levels of self-competence than did girls. In addition, boys were significantly less depressed and anxious than girls at 12, but not at 11. According to these authors, self-competence would be partially responsible for the emergence of gender differences in depression and anxiety during late childhood.

In order to contribute to clarify some of the results recorded above, the aim of this chapter is to study vulnerability to depression in normal boys and girls in middle and late childhood, and its relation with cognitive vulnerability, as expressed in children's self-perception of academic and social competency, controllability of perceived stressors, and coping strategies.

METHOD

Participants

This study has been carried out on a sample of 1,019 middle class children, aged 8 (n = 184), 9 (n = 201), 10 (n = 205), 11 (n = 224), and 12 (n = 205), (483 boys and 536 girls) from four primary schools — two state-run and two public schools — residing in the city of Buenos Aires and various towns in the province of Buenos Aires. The instruments were administered to groups of 15 children by three psychologists per group.

Ethical Procedure

We asked for an interview with the heads of the schools to discuss the work to be done with the children. We explained the characteristics of the research and submitted a letter asking for their collaboration and attached a copy of the research project. After that, a letter was sent to the father and mother of each child explaining the aims of our project and the work to be done with the children. They were expressly told that participation was voluntary and anonymous. We committed ourselves to give no individual information whatsoever to the school staff, unless both parents expressly asked us to do so. We received a written permission from each father and mother before launching the field work. Finally, we told the children that we needed to know their opinion on their parents' relationship with them and they were told yet again that they were free not to answer. There were no objections.

INSTRUMENTS

The Self-Perception Profile for Children (Harter, 1985; Richaud de Minzi, Sacchi, & Moreno, Argentine adaptation, 2001)

Our study of academic and social competence in middle childhood was based on the Self-Perception Profile for Children (Harter, 1985), a revised version of the Perceived Competence Scale for Children (Harter, 1979, 1982), that assesses children's judgments of their competence in three domains: cognitive, social and athletic, as well as a global perception of their worth or esteem as a person. The current version of the instrument — Self-Perception Profile for Children — contains six separate sub-scales covering five specific domains: Scholastic Competence, Social Acceptance, Athletic Competence, Physical Appearance, and Behavioral Conduct, as well as Global Self Worth.

For the present work we only used the Scholastic Competence Scale and the Social Acceptance Scale.

The former investigates the child's perception of his/her competence or ability within the realm of scholastic or academic performance (Harter, 1985).

The Social Acceptance Scale assesses the degree in which the child is accepted by peers or feels popular. The items do not refer to social skills.

Each of the sub-scales contains six items.

The internal consistency reliability for all six scales based on Cronbach's alpha was fairly acceptable (Harter, 1985). Cronbach's alphas for Scholastic Competence, across several samples, ranged between .80 and .87 and for Social Acceptance between .75 and .86 (Eapen, Naqvi, & Al-Dhaheri, 2000; Harter, 1985; Van Den Bergh & Marcoen, 1999).

The test-retest reliability of the sub-scales for 24 children over a 3-year period was also satisfactory (Granleese, 1994).

The Argentine adaptation of the scales was done on the sample of 1,421 children just described.

On the basis of the factor analysis of the 12 items (six corresponding to scholastic self-competence and six to social self-competence) in the original scales, we built a shorter, 8-item version.

As a result of a new factoring of these eight items, two factors were obtained: Academic ($\alpha = .75$) and Social Acceptance ($\alpha = .71$).

Argentine Coping Questionnaire for children (Richaud de Minzi, 2006c)

According to Folkman and Lazarus (1988), coping not only includes approaching-eluding behavior or defensive processes to cope with the complex and restrictive demands of a given stressor. It also comprises a wide range of cognitive strategies that fulfill the function of problem solving and emotion-regulation. Research on coping focuses on processes that are specific categories of behavior (such as confrontative coping, search for social support, flight-avoidance, planning problem solving) that modify the source of the problem (problem-centered coping) or emotional distress (emotion-centered coping). Billings and Moss (1981) distinguish cognitive from behavioral aspects and divide coping processes into three categories: focused on evaluation, problem-focused and focused on emotions. These categories have proved to be a valuable theoretical perspective (Schwarzer & Schwarzer, 1995).

Based on this perspective, 27 items were constructed that could elicit the following replies: Yes, Sometimes, No. Three items were included in each sub-dimension: a) focused on evaluation: logical analysis, cognitive restructuring and cognitive avoidance, b) problem-focused: search for support, action on the problem, search for alternative gratification, and c) focused on emotion: emotional control, generalized inhibition and emotional loss of control. Factor analysis has indicated two factors: Functional (α =.71) and Dysfunctional (α =.74) strategies.

The Coping Questionnaire expects children to focus on whatever worries them at a given moment. Situations described by children were included in one of the following categories: interpersonal difficulties with family and peers, worried about his/her health, worried about the health of his family, fear of the future, worried about his self-image, parents' divorce, studies, economic situation, insecurity.

Dimensions of Depression Profile for children and adolescents (Harter & Nowakowski, 1987; Richaud de Minzi, Sacchi, & Moreno, Argentine adaptation, 2001)

The initial version of Dimensions of Depression Profile for children and adolescents is a self-report instrument that operationalizes four dimensions of depression: affect/mood (the extent to which one feels cheerful and happy as opposed to sad and depressed), global self-worth (the extent to which one feels pleased with oneself), energy/interest (the extent to which one feels wide awake and energetic), and self blame (children feel that things which go wrong are their fault). It provides a profile of scores across dimensions, as well as a global depression score. Although, as stated above, the initial version of the instrument contained four scales, the present Profile includes a fifth sub-scale, suicide ideation (the extent to which one considers committing suicide (Harter & Nowakowski, 1987). Each of the sub-scales includes six items.

The internal consistency reliability for all five scales based on Cronbach's alpha was fairly acceptable (Harter, 1987). Cronbach's alphas for Mood Affect across several samples ranged between .84 and .88, for Self-Worth between .81 and .84, for Energy/Interest between .72 and .87, for Self-Blame between .76 and .86, and for Suicidal Ideation between .88 and .90 (Eapen, Naqvi, & Al-Dhaheri, 2000; Harter & Nowakowski, 1987; Van Den Bergh & Marcoen, 1999). The Profile study with the Argentine sample did not include the Suicidal Ideation sub-scale as we were working with children with no pathologies.

Factor analysis (N=1,421) showed four factors corresponding to dimensions posited by Harter and Nowakowsky (1987). We worked with a new scale formed by the three items with greater weight in each factor: a total of 12 items.

Cronbach's alphas for all four scales were: .76 (Self-Blame), .71 (Self-Worth), .75 (Energy/Interest) y .77 (Mood Affect).

RESULTS

Cognitive Vulnerability: Academic and Social Competence - Self-Perception

Upon analyzing the relationship between academic and social self competence and depression, I found that self competence explains 15% variance of depression. The analysis of standardized regression coefficients of each type of self competence indicates that both

academic and social self competence negatively predict depression in children (β = -.23, p < .000, and β = -.26, p < .000, respectively).

Considering the relationship between academic and social self competence and depression dimensions, self competence explains 4% of self blame dimension, 7% self-worth, 13% energy/interest, 2% mood affect. The analysis of standardized beta coefficients indicated that: both social and academic self competence negatively predict self blame (β = -.12, p < .001, and β = -.14, p < .000, respectively); social and academic self competences negatively predict self worth (β = -.19, p < .000, and β = -.15, p < .000, respectively); and social and academic self competences negatively predict energy/interest and (β = -.24, p < .000, and β = -.21, p < .000, respectively).

Coping

As to depression and coping style, a MANOVA comparing two extreme groups of global depression (lesser than Percentile 25 and higher than Percentile 75) in connection with coping results, there are significant differences in the way children in these depression-groups cope with problems ($F_{\text{Hotelling}}$ (9, 305) = 3.08, p < .001). *Post hoc* contrasts showed that these differences are seen in cognitive evasion-coping (F(1, 313) = 7.192, p < .008), alternative gratification (F(1, 313) = 7.25, p < .007), emotional control (F(1, 313) = 13.53, p < .000), generalized inhibition or paralysis (F(1, 313) = 10.71, p < .001), and emotional non-control (F(1, 313) = 3.71, p < .050), more depressive children witness the highest values in all these coping strategies (Table 1).

Table 1. Mean, standard deviations and F corresponding to coping strategies in vulnerable depression and non vulnerable depression children (N= 409)

Coping	Md	SDd	Mnod	SDnod	F(1, 313)	p
Logical Analysis	2.38	.04	2.45	.05	1.42	.235
Cognitive restructuring	2.62	.04	2.57	.04	.74	.389
Cognitive avoidance	1.94	.06	2.16	.06	7.19	.008
Search for support	2.28	.05	2.17	.06	2.01	.157
Action on the problem	2.50	.05	2.42	.06	1.05	.306
Search for alternative gratification	2.22	.05	2.40	.05	7.25	.007
Emotional control	2.09	.05	2.34	.05	13.53	.000
Generalized inhibition	1.92	.06	2.19	.06	10.71	.001
Loss of control	1.87	.07	2.08	.08	3.71	.055

$F_{\text{Hotelling}}$ (9, 305) = 3.08, p < .001

Perceived Stressors

When studying stressors perceived by children, there is a significant association between depression and type of stressors (χ^2 = 23,839 gl = 9, p < .005). Depression-vulnerable children witnessed more interpersonal difficulties with family, parents' divorce, and parents' fights, while non depressed children indicated more interpersonal difficulties with peers,

economic problems in their country, and others that included general difficulties such as friends' moving, feeling sorry for children in poverty, etc. (Table 2).

Table 2. Stressors of a sample of Argentine males and females 8-12 years

Stressors	Frequence	%	No vulnerable	Vulnerable
Interpersonal difficulties with friends	159	19	53	29
Concern about academic difficulties	146	17.4	35	34
Economic situation	131	15.7	50	20
Interpersonal difficulties with the family	128	15.3	31	41
Concern about health of family or death of a loved one	105	12.5	28	20
Others	64	7.6	18	9
Fear of the future	39	4.7	7	6
Insecurity	29	3.5	6	5
Parents rows and divorce.	24	2.9	2	10
Concern about own image.	9	1.01	3	2
Concerns about own health	3	.4	-	-
Total	837		233	176

Out of 1019 children in the whole sample, only 837 voiced their main concern

Frequency of stressors in vulnerable depression children and non vulnerable depression children corresponds to the two extreme groups (n = 409), as explained in Results

Gender

As to the influence of gender over depression in general, males witness higher values than females ($t = 4.318$ $gl = 797$, $p < .000$)

The profile that indicates the influence of gender over depression dimensions was studied through a MANOVA. I have found that there is a general and significant difference between males and females in depression ($F_{\text{Hotelling}}$ (4, 794) = 5.59, $p < .000$). An analysis of *post hoc* contrasts shows significantly more Self blame ($F(1, 787) = 15.07$, $p < .000$), and lower Energy ($F(1, 787) = 11.10$, $p < .001$), in males that in females (Table 3).

This marked difference in global depression between males and females is maintained (t = 3. 915 gl = 447, $p < 000$) in both age groups: 8-10 years of age and 11-12-year old (t = 2.305 gl = 347, $p < .022$). The difference between males and females diminishes with age, however, with 11-12 age- groups still showing higher values but less than in younger groups. (Mean difference males-females 8-10 = .50, Mean difference males-females 11-12 = .33).

Table 3. Mean, standard deviations and F corresponding to depression dimensions in males and females (n = 799)

Depression dimensions	**Mm**	**SDm**	**Mf**	**SDf**	**F(1, 787)**	**p**
Self blame	2.01	.63	1.84	.59	15.07	.000
Low Self worth	1.53	.57	1.47	.57	2.71	.100
Low Energy/interest	1.86	.59	1.73	.56	11.10	.001
Mood Affect	1.75	.35	1.71	.34	3.08	.079

$F_{\text{Hotelling}}(4,794) = 5.59$, $p < .000$

In the case of depression dimensions, there was an important general difference between males and females among 8-10 children ($F_{\text{Hotelling}}$ (4, 444) = 4.39, p = .002). *Post hoc* contrasts showed considerable differences in all depression dimensions: Self-Blame *(F*(1, 447) = 4.698, $p < .031$), Self-Worth (F (1, 447) = 4.774, $p < .029$), Energy/Interest (F (1, 447) = 11.013, $p < .001$), and Mood Affect *(F* (1, 447) = 4.864, $p < .028$) (Table 4).

Table 4. Mean, standard deviations and F corresponding to depression dimensions in males and females 8-10 years (n = 449)

Depression dimensions	Mm	SDm	Mf	SDf	F(1, 447)	p
Self blame	2.00	.04	1.87	.04	4.70	.031
Low Self worth	1.62	.04	1.50	.04	4.77	.029
Low Energy/interest	1.98	.04	1.80	.04	11.01	.001
Mood Affect	1.77	.02	1.70	.02	4.86	.028

$F_{\text{Hotelling}}$ (4,444) = 4.39, $p < .002$

As to 11-12-year old children, there is a significant general difference between males and females ($F_{\text{Hotelling}}$ (4, 344) = 3.21, $p < .013$) but post hoc contrasts showed only noteworthy differences in Self-Blame (F(1, 347) = 11.89, $p < .031$), with male values higher than females' (Table 5).

Table 5. Mean, standard deviations and F corresponding to depression dimensions in males and females 11-12 years (n = 349)

Depression dimensions	Mm	SDm	Mf	SDf	F(1, 347)	p
Self blame	2.01	.05	1.79	.05	11.89	.001
Low Self worth	1.42	.04	1.41	.04	.01	.915
Low Energy/interest	1.73	.04	1.64	.04	2.29	.131
Mood Affect	1.73	.03	1.72	.03	.02	.880

$F_{\text{Hotelling}}$ (4, 344) = 3.21, $p < .013$

DISCUSSION

Depression in adolescents and adults is an important problem in mental health that could be prevented if we had deeper knowledge about its precursors in childhood.

According to the literature available, depression vulnerability in children is accompanied by cognitive vulnerability expressed as child's negative self-perception of competency. In our study, we found that children with depression vulnerability perceived lesser academic and social self competence than non depressive children, and that low self competence was especially related to negative self-worth and low energy and interest. Apparently, self competence has little relation with mood affect, which is probably a more stable feature of personality, less affected by cognitive vulnerability.

According to previous literature findings, this maladaptive cognition related to depression vulnerability is accompanied by a characteristic form of stressors perception that influences the efficiency of coping strategies.

When analyzing perceived stressors, we can observe that children with depression vulnerability feel a deeper threat from events relating to their family, or close circle of acquaintances, and appear to have no special interest on external events.

The present study was performed during a period when Argentina experienced an unprecedented economic crisis, with most people concerned about the economic situation, job-loss, etc. While among non-depressive children the main problems perceived were more external and related to the general situation: the economy, father's job, own and friends' relocation, and interpersonal difficulties with peers; depressed children remained absorbed with internal family problems.

As we said before, negative self-perception results from an insecure attachment to parents and parental rearing styles (Richaud de Minzi, 2006a; Muris et al., 2001). These results confirm that insecure attachment to their parents and negative parental rearing behavior would be important risk factors in the development of depression (DesJardin, 2003; Richaud de Minzi, 2006; Muris et al., 2001).

At the same time, we have hypothesized that dysfunctional coping is a risk factor for depression-vulnerability in children. Our results indicate that children with vulnerability to depression use avoidance coping, such as cognitive avoidance and alternative gratification, and emotional-focused coping, such as emotional over-control, generalized inhibition or paralysis and emotional lack of control, when faced with a stressor. On the other hand, non depressive children show a tendency to use problem-focused coping and approach, such as seeking social support.

Compas et al. (1988) asserted that more functional coping strategies corresponded to stressors that were perceived as controllable, as is the case with academic stressors. In the present study with children 8 to 12-years old, however, there was no difference in the perception of academic stressors among depressive and non depressive children. The difference was established between close family stressors and outside family stressors, both being apparently uncontrollable events for children. Probably, at this age, close family stressors, such as the parents' divorce or arguing, upset them more profoundly than external stressors, such as economic problems or interpersonal difficulties with peers.

However, concerning the pattern of coping in children vulnerable to depression, our results are similar to those of others authors (Compas et al., 1988; Causey & Dubow, 1992; Herman-Stahl et al., 1995; Seiffge-Krenke & Klessinger, 2000; Seiffge-Krenke & Stemmler, 2002): low approach, high avoidance and emotion-focused coping.

Respecting gender differences in depression, contrary to the hypothesis that girls are more vulnerable to depression than boys, we found that between 8 and 10 years of age, males witness more vulnerability to depression than girls; whereas between ages 11 and 12, differences diminished. As observed in middle childhood, males have higher values in all depression dimensions: self blame, self worth, energy/interest, and mood affect, but in late childhood, boys have significantly higher values only in self blame.

In fact, apparently clear gender differences in depression only appear in early adolescence (Angold, Erkanli, Silberg, Eaves, & Costello, 2002).

Although developmental psychology hypothesizes that sex stereotypical socialization practices by caregivers lead to gender differences in depressive vulnerability, our results do not support this hypothesis. Having said that, it would seem that differences diminish with age and we could hypothesize that probably at a certain point, the difference reverses. However, following developmental theories about socialization practices, nowadays these

practices have changed a lot, at least in occidental urban societies. Girls have an increased sense of control and are more independent and less concerned for social evaluation. At the same time, boys have a decreased sense of mastery. These characteristics bring the behavior of boys and girls closer.

Summing up, cognitive vulnerability expressed as negative self-perception of academic and social competence is a factor of risk that may offset depressive vulnerability in middle and late childhood. This cognitive vulnerability is also connected with threat appraisal related to close-family interpersonal difficulties. This would corroborate the importance of parents as factors of risk in depressive vulnerability. Coping with these difficulties follows a pattern characterized by non-problem focus, low approach, high avoidance and emotion-focused coping. Finally, greater gender differences in vulnerability to depression appear in middle childhood and diminish in late childhood, males being more vulnerable to depression than girls, probably due in part to changes in socialization practices.

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Chapter 10

THE IMPORTANCE OF IDENTIFYING CHILD AND ADOLESCENT DEPRESSION IN THE MEDICAL SETTING

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ABSTRACT

Depression poses a significant risk to the mental health of children and adolescents, and with a prevalence rate of about 2-10% is not uncommon. While most pediatricians feel responsible for identifying depression in children and adolescents, successful recognition is estimated to be as low as 17%. Depression has been found to worsen health outcomes, as child patients with depression may be three times more likely to be medically noncompliant than their non-depressed counterparts. Fortunately, depression is also highly treatable with psychotherapy and psychotropic medication. Because depression causes emotional suffering and is a risk factor for medical noncompliance and poor health outcome, the pressing need to recognize childhood depression in the medical setting is clear. This commentary discusses the importance of identifying and treating childhood depression in the medical setting and suggests future directions for research.

"The incidence of untreated child and adolescent mental health problems constitutes a public health crisis for our nation" (American Psychological Association Task Force on Psychology's Agenda for Child and Adolescent Mental Health, 2003, p. 3). This statement highlights the current dismal state of affairs for children with mental health needs. It is consistent with the perspective of a relatively recent report by the Surgeon General (U.S. Department of Health and Human Services, 1999). Some estimates indicate that as many as 75% of children with mental health needs remain untreated (Mash & Dozois, 2003). Unfortunately, depression is common in children of all ages. It is estimated that 2%-10% of children and adolescents experience a depressive disorder (Birmaher, Brent & Benson, 1998;

Costello, Mustillo, Erkanli, Keeler & Angold, 2003; Dopheide, 2006). The adolescent lifetime prevalence rate for major depression is estimated to be even higher at 14% (Hammen & Rudolph, 2003).

Given this mental health "crisis," what role do medical professionals have? The medical setting provides both a unique opportunity and responsibility to recognize depressive symptoms and address them through treatment or referral. Young people suffering from "psychosocial dysfunction and/or chronic illness" see their primary care physician about four times a year (Bernal et al., 2000, pp.263-264). Many health care systems structure the primary care physician as the "gatekeeper" to specialty mental health care (Costello et. al, 1988). However, recognition of psychosocial problems in the pediatric setting is poor and has been estimated to be as low as 20% (Bernal et al., 2000). Other estimates find that childhood psychiatric illnesses are recognized about 17%-59% of the time in the medical setting (Goldman et al., 1999). In addition to psychological suffering, health care utilization and costs are elevated for children suffering from psychosocial problems (Bernal et al., 2000). Although numerous barriers to recognition exist, identification and diagnosis are crucial to obtaining treatment.

Depression in the medical setting may be complicated by other problems, such as the co-occurrence of chronic illness. Chronic childhood illness is an intensely stressful experience for the child as well as the family unit. The first author's clinical experience as a schoolteacher in a children's hospital has brought to her attention the unique stressors associated with seriously ill children and their families. The hospitalization and treatment process can create enormous stress, as family members can be separated by large distances while children are treated at specialty children's hospitals. Hospitalization or outpatient treatment may linger into months or years. With the family unit far from home, the comforting routine of school and work is absent. Chronic illness introduces a host of difficulties for young people that include body image problems, difficulties with peers, academic problems and stressful medical procedures (Jellinek & Snyder, 1998). In this environment, the first author has noted that depressive symptoms in pediatric inpatients and their siblings can go unrecognized and untreated. While the physical health of the child is imperative, mental health problems must be recognized as well.

In addition to this anecdotal experience, research has also established that chronic illness has a profound effect on the psychosocial functioning of children and adolescents. It is not surprising that depression has been found to be associated with chronic and serious illness (Scott et al., 2007). Depression is not only a devastating mental illness, but also may have a substantial impact on medical treatment adherence and even survival. In their meta-analysis examining the relationship between depression and medical non-adherence, DiMatteo, Lepper and Croghan found that depressed patients were three times more likely than non-depressed patients to be medically non-adherent (2000). Although the meta-analysis focused mainly on adult studies due to the lack of pediatric studies, their findings were compelling. A recent study on treatment adherence in adolescent oncology patients also found that non-adherence is correlated to elevated depression levels. Sadly, medication non-adherence was also correlated with mortality. Although the majority of adolescents exhibited subclinical levels of depression, this study highlights the need to identify even minor mood disturbances in seriously ill adolescents (Kennard et al., 2004). The connections between depression, medical adherence, and survival make detection and treatment of depressive disorders essential.

Clinicians in the medical setting need to be attuned to depressive symptoms, as medication and treatment side effects can mimic depression. Symptoms characteristic of depression--namely fatigue, appetite problems and cognitive changes--might easily be misinterpreted by clinicians because they are also common side effects of cancer treatment (Dejong & Fombonne, 2005). Some research has suggested that adolescents may be less willing to tolerate intensive drug regimes, such as immunosuppressant drug therapy, due to side effects such as obesity, infection and depression (Griffin & Elkin, 2001). Numerous medical conditions can imitate depressive symptoms in young people, including infection, epilepsy, diabetes and hypothyroidism (Jellinek & Snyder, 1998). Pharmaceutical drugs such as benzodiazepines, corticosteroids and oral contraceptives can also produce depressive symptoms in children and adolescents (Jellinek & Snyder, 1998). Thus, it is important to keep in mind that treatments for medical problems may actually create subsequent depression through either the side effects of medication or the social impact of coping with medical illness.

Numerous barriers to identification of childhood depression exist even for those who are mental health professionals. Although the symptoms needed to meet diagnostic criteria are the same for adults and children, there are developmental considerations that may affect symptom presentation. For example, irritability seems to be more common in children with depression. In addition, children's capacity to report their symptoms may be influenced by cognitive development and language considerations. Furthermore, given the high rates of comorbidity with disruptive behavior disorders, depression may be missed or "masked" by other more readily recognized disruptions (Hammen & Rudolph, 2003).

This diagnostic issue is complicated in the medical setting with the additional barriers mentioned above; medical illness causing/exacerbating depression and treatment side effects that appear similar to depression. In a study surveying pediatricians' thoughts about their role in diagnosing and treating childhood depression, 90% of pediatricians considered themselves responsible for diagnosing depression in youth. However, only 46% of the pediatricians felt they had the necessary skills to diagnose depression. "Inadequate time to provide counseling or education" and "incomplete training to diagnose or counsel" were some of the most commonly perceived barriers (Olson et al., 2001, p. 93). Indeed, lack of time appears to be the most commonly perceived obstacle for "identification and management of children's psychosocial issues" in the medical setting (Horwitz et al., 2007, p. e208). With the current health care crisis, it is alarming, but not surprising, that psychosocial issues go unrecognized in the already overburdened medical setting. However, given the evidence that depression can exacerbate illness and affect outcome, and that it can be successfully treated when identified, it is incumbent upon medical professionals to identify childhood depression.

The importance of diagnosing child and adolescent depression is particularly critical as successful therapeutic interventions and psychotropic medications are widely utilized. In fact, both intervention types can be helpful. Emslie and associates (2002) examined the effects of fluoxetine, a selective serotonin reuptake inhibitor (SSRI) in a randomized clinical trial with 122 children and 97 adolescents with major depressive disorder (MDD). They found that a protocol of 10 mg/day of fluoxetine for a week followed by 20 mg/day for eight weeks was associated with a significantly greater drop in depression scores compared to a placebo condition. For mild to moderate childhood depression, psychotherapy can be considered an alternative to psychotropic medication (Son & Kirchner, 2000). Although the evidence for various kinds of psychotherapy for children is limited and mostly based on studies with

adults, a variety of approaches are used with children, which should be tailored to children's developmental level (Son & Kirchner, 2000). The most compelling evidence exists for the effectiveness of Cognitive-Behavioral Therapy (CBT) in reducing depressive symptoms. In an 8 week study, adolescents provided with CBT showed significantly greater reductions in depression compared to those assigned to a waitlist control condition (Clarke, Rohde, Lewinsohn, Hops & Seeley, 1999).

The effects of psychotropic medication may enhance the effects of psychotherapy--at least for adolescents--as suggested by a randomized controlled trial conducted by the Treatment for Adolescents with Depression Study Team (2004). They found that the combination of fluoxetine with CBT was significantly more effective in alleviating depression among adolescents than was either CBT alone or the placebo condition. The combination of fluoxetine with CBT also showed the greatest reduction in clinically significant suicidal thinking. Effective treatments for child and adolescent depression are prevalent; therefore, the identification and treatment of such a debilitating and costly disorder is vital.

Pediatricians are the gatekeepers in children's healthcare. They can function as a unique bridge between mental and physical healthcare for young people. It is imperative that significant mood changes and depressive symptoms are recognized in the medical setting, and action is taken. Untreated child and adolescent depression not only leads to unnecessary suffering, but also is associated with comorbid substance abuse and other psychological disorders (Hammen & Rudolph, 2003). Indeed, high levels of depressive symptoms in adolescence have been found to be predictive of suicidal acts in later adolescence and early adulthood, making prompt identification critical (Nrugham, Larsson & Sund, 2008). The barriers are numerous, but because the alternative is unthinkable, identification of childhood depressive disorders in the medical setting must take precedence. Further research is necessary to improve understanding of how depression and medical illness interact to exacerbate or complicate children's health. In addition, research is required on how to accurately diagnose medically ill children with depression. Furthermore, greater knowledge is needed about how to improve and deliver treatments to address the complicated issues that are presented by children with medical illnesses and depression.

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Chapter 11

DEPRESSION IN CHILDREN WITH GILLES DE LA TOURETTE SYNDROME

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ABSTRACT

The Gilles de la Tourette Syndrome (GTS) is a neurodevelopmental disorder characterised by the presence of multiple motor tics and at least one phonic tic. GTS is increasingly recognized as a relatively common neuropsychiatric disorder, usually diagnosed in early childhood. Comorbid behavioral problems occur in approximately 90% of patients, with attention deficit hyperactivity disorder (ADHD) and obsessive-compulsive disorder (OCD) being the most common ones. Depression is also common, with a lifetime risk of 10% of patients. Moreover, converging evidence shows that children with GTS may have depression ratings that are higher than those in other school age children but lower than those found in children with primary depressive disorder. Whilst the association between GTS and depressive symptoms does not appear to be mediated genetically, social and environmental factors are thought to be of etiological importance. Children with GTS have involuntary movements and noises, which might amount to a chronic stigmatizing disorder in a school setting. This in turn could isolate the individuals, make them a subject for bullying, and ultimately lead to depression. The depression in children with GTS has been shown to result in a reduced health-related quality of life, and in severe cases it may lead to hospitalization and even suicide. Further research is needed to address factors of particular relevance to the etiology of depression in children with GTS and thus improve its recognition but also treatment and outcome.

INTRODUCTION

GTS is increasingly recognized as a common neurodevelopmental disorder characterized by the presence of both multiple motor and one or more vocal/phonic tics, although not necessarily concurrently, lasting longer than one year [1]. A tic is defined as a sudden, rapid, recurrent, nonrhythmic, stereotyped movement or vocalization [2]. The age at onset of motor tics is usually 6-7 years, whereas the onset of phonic tics is usually later, at around 11 years. Tic-associated symptoms include echolalia, echopraxia, palilalia, and coprolalia (in 10–15% of community samples) starting at around 15 years [3-7]. It may well be that a younger age at onset is associated with a more severe GTS [8]. Typically, tics occur many times a day, nearly every day, or intermittently with a waxing and waning course. It has been reported that their anatomic location, number, frequency, complexity, type, and severity usually changes over time [2]. The diagnosis of GTS is made from clinical and historical observations, as no specific laboratory or instrumental tests are currently employed. With respect to formal diagnostic criteria, there is a substantial overlap between the Tourette Syndrome Classification Study Group [9], the World Health Organization [10], and the American Psychiatric Association [11].

The most commonly used measure for tic severity is the Yale Global Tic Severity Scale (YGTSS) [12]. It was initially thought that GTS was lifelong, but more research [13] suggested that the prognosis was correlated with the onset age. Apparently the majority of symptoms disappear in half of the patients by the age of 18 years. With regards to disease evolution, Coffey et al. [14] assessed youngsters with GTS and found that ages at onset were similar; at baseline, 88% of subjects met threshold criteria for at least mild symptoms, but only 30% met criteria for impairment. At 2-year followup, 82% of the subjects met criteria for tic persistence (no significant difference from baseline), but only 14% met criteria for GTS-associated impairment that was significant. Bloch et al. [15] more recently studied 46 GTS children on follow-up after 7.6 years and reported that 85% had a reduction in tics during adolescence. Only increased tic severity in childhood was associated with increased tic severity on follow-up, whereas the worst tic severity was observed at 10.6 years.

Comorbid neuropsychiatric disorders occur in approximately 90% of patients, with attention deficit hyperactivity disorder (ADHD), obsessive-compulsive disorder (OCD) and depression being the most common ones. ADHD and OCD themselves are associated with depression, so determining the real relationship between depression and GTS can prove very difficult. Because of the high frequency of comorbid conditions, classification of GTS can be a problem too. DSM-IV-TR distinguishes GTS from chronic tic disorders, in which patients have either multiple motor or vocal tics (but not both) [5]. Moreover, it has been suggested [1,16] that it may be useful to clinically subdivide GTS into: (i) “pure GTS”, consisting primarily and almost solely of motor and phonic tics; (ii) “full blown GTS” which includes coprophenomena, echophenomena and paliphenomena; (iii) “GTS-plus” [17], in which an individual can also have depression, ADHD, significant obsessive-compulsive symptoms or OCD, or SIB. Others presenting with severe comorbid neuropsychiatric conditions may also be included in this group. In consideration of the clinical heterogeneity of this condition, it appears legitimate to question if GTS can be considered a unitary nosological entity.

With regards to psychiatric comorbidity, the worst ever obsessive–compulsive disorder (OCD) symptoms tend to occur a few years later than the worst tic severity. Increased

childhood intelligence quotient can be associated with increased OCD severity on follow-up. Although the prognosis of GTS is better than originally thought with regards to tic symptomatology, psychopathology, such as OCD, may persist longer and severely after the regression of GTS itself. Therefore, patients with GTS may be prone to depressive symptoms or depression later on in life, despite the fact that tics may have lessened (i.e., as OCD symptoms increase). However to the best of our knowledge no studies have explored this issue in close detail.

Depression is commonly reported by patients with GTS, with a lifetime risk of about 10% and with rates almost double among women. It is also common in young people, particularly in adolescent girls, occurring in about 8%. It may be a mild or a severe disorder when the lifetime suicide risk is about 15%. Depression is sometimes described as a spectrum disorder with a variety of clinical subtypes, and is well known to complicate physical illness [18], particularly neurological disorders like GTS or other psychiatric conditions like OCD. There are several ways by which depression and physical illness may be associated [19]. We will examine these in detail in the next two paragraphs.

The assessment of depression in the context of GTS may be complex. There are numerous diagnostic interviews, as well as physician-rated scales, that can be used to evaluate and diagnose depression, either specifically or as part of a larger psychopathological assessment. These include the Schedule for Affective Disorders and Schizophrenia, the Present State Examination, the Hamilton Depression Rating Scale, the Structured Clinical Interview for DSM-IV-TR [20,21], the Composite International Diagnostic Interview, the Clinical Interview Schedule-Revised, and the Schedules for Clinical Assessment in Neuropsychiatry, which are used primarily in research settings [22]. There are also many self-rating scales used to measure depressive symptomatology, including the Beck Depression Inventory (BDI), the Mood Adjective Checklist (MACL), the Crown Crisp Experiential Index (CCEI), the Hospital Anxiety Depression scale, and, in young people, the Kovacs Child Depression Inventory (CDI) and the Birleson Depression Self-Rating Scale (DSRS) [21]. In clinical practice, a full Mental State Examination (MSE) is important as many of the research interviews take time. The MSE may be supplemented by the use of self-report scales. The assessment and appropriate treatment of depression in this context are crucial. The aim of this chapter is to review the current understanding of the relationship between depression and GTS in children and its impact on diagnosis and treatment.

EPIDEMIOLOGY OF DEPRESSION IN PEDIATRIC GTS POPULATION

Following several early descriptions of depression in GTS [23], there have been numerous case reports, as well as both uncontrolled and controlled studies, examining depressive symptomatology and depression in pediatric patients with GTS. In 16 uncontrolled studies in specialist centers examining mood changes among 5409 GTS patients, depressive symptomatology, dysthymia, mood swings and/or MDD, or depressive illness was found in 13–76%. Many studies suggested that a significant number of patients with GTS have Major Depressive Disorder [24-39]. Using standardized measures, 13 controlled investigations have found both young people and adults with GTS (n=741) to be significantly more depressed than age-matched and gender-matched healthy control subjects [40-52]. Only one study

obtained differing results. Termine et al. [53] studied 17 GTS youngsters who were matched with healthy controls, and although GTS subjects scored higher than controls on the CBCL, there were no differences with regard to depression. However, since only few studies focused on this particular relationship, it seems appropriate to encourage for more controlled studies.

A pilot epidemiological study of tics and GTS by Mason et al. [54] studied 166 Year 9 pupils aged 13–14 years in a mainstream school in the United Kingdom. Thirty (18%) pupils were diagnosed as "tic possible", and five were diagnosed with GTS. Depressive symptomatology, as rated by the DSRS, was not significantly different between the tic-possible group and the total population. Of the five pupils with GTS, one was diagnosed with depression.

Hornsey et al. [55] reported the definitive investigation of a previous study in the United Kingdom and included 918 Year 9 pupils. When the total population (n=918) was compared to the tic-possible group (n=42), the tic-possible group's DSRS score was no different from that of the whole group. This means, therefore, that youngsters with tics in these studies undertaken in schools had more difficulties than their peers, but this did not include depressive symptomatology. Peterson et al. [56] reassessed a cohort of youngsters with tics in the community. They reported that years after the first assessment, which took place between the ages of 1 and 10 years, depression was significantly associated with the presence of tics.

Kurlan et al. [57] studied 1596 children between the ages of 9 and 17 in Upstate New York, using the Diagnostic Interview Schedule for Children to yield DSM-IV-TR psychiatric diagnoses. Parents and teachers also completed their versions of the Child Behavior Checklist (CBCL). Children with tics had a significantly higher frequency of psychiatric diagnoses, including major depression. The diagnoses clustered among four categories, including one of mood disorders. Among children with tics, psychopathology was common, ranging from 8.9% to 38.4%; mood disorders were less common (1.2–9.7%). However a major limitation of this study was the low response rate (only 11% of selected subjects agreed to participate).

Thus, in these community studies, individuals with GTS were rated as having significant depression in two of five studies. In the majority of studies, the youngsters who had tics had mild tic severity. Moreover, these data seem to suggest that the etiology of depressive symptomatology could be more related to the social impact of lengthened tics than GTS itself.

SUGGESTED CLINICAL ASSOCIATIONS AND CORRELATES OF DEPRESSION AND DEPRESSIVE SYMPTOMATOLOGY IN CHILDREN WITH GTS

First, most of the community studies mentioned above [58,54-57] failed to report depression or depressive symptomatology. This is important as the GTS subjects in those studies were mildly affected; it may thus be that mild tic severity is not associated with depression. Comings and Comings [43] found no differences in the frequency of depression in patients with or without ADHD, and found minimal correlation between the number of tics (i.e., a measure of severity) and depression. They suggested that depressive symptoms were integral to GTS rather than secondary to motor or vocal tics. Cardona et al. [35] studied 125 children and adolescent with tics and GTS who were drug-naive. They were of mild to moderate severity according to YGTSS scores, and statistical analysis demonstrated a

significant relationship between the duration of tic symptomatology and several CBCL scores, including the anxious/depressed subscale.

Both Carter et al. [49] and Sukhodolsky et al. [51] compared GTS-only versus GTS+ADHD patients and healthy controls. Carter et al. showed that GTS-only patients were significantly more depressed than controls, whereas Sukhodolsky et al. reported that GTS+ADHD, not GTS-only, patients were more depressed than controls.

Banashewski et al. [59] analyzed the GTS worldwide database (n=4833) and highlighted that the presence of obsessive compulsive symptoms was associated with impulsive and aggressive behaviors, as well as with depression and anxiety. Romano et al. [36] studied sleep disorders in 49 children with tics and GTS, and reported that sleep-wake transition disorders were the most frequent type of disorder; they were highly correlated with the severity of tics, internalizing problems, and anxiety/depression. Woods et al. [60] studied the psychometric properties of the Premonitory Urge for Tics Scale (PUTS) and showed that PUTS scores were correlated with tic severity (as measured on the YGTSS), and anxiety and depression (on the CBCL and YBOCS scores).

A study of depressive symptomatology in 72 moderately affected young GTS individuals (mean age 12 years, range 5-17) was undertaken by Robertson et al. [39], who reported substantial depression as measured by the BDSRS and CDI. Fifty patients had obsessive compulsive behaviors and 43 patients had ADHD. There was a significant difference in the two age-based subgroups (subjects aged 5–12 versus 13–17 years) in the distribution of ADHD, with the younger age group showing higher rates. However, age was not found to correlate with any of the other variables studied, and none of the variables correlated with gender. A positive correlation was found between the two self-report scales for depression and obsessive compulsive symptoms and ADHD. Obsessive compulsive scores also showed a positive correlation with depression scores, and obsessive compulsive behaviors were found to correlate with DCI scores. With regards to ADHD, there was a significant positive correlation with both BDSRS score and CDI-T score. Depression scores also showed a significant correlation with severity of GTS, as indicated by YGTSS and DCI scores. Finally, it is interesting to note that CDI scores correlated with Leyton Obsessional Inventory symptom scores, suggesting the link between depression and obsessive compulsive behaviors, as noted before by Eapen et al. [37].

Overall, it appears that the clinical association of GTS and depression increases along with tic severity, coexistence of OCB/OCD, SIB, coprophenomena and echophenomena, premonitory urges, sleep disturbance, aggression, childhood CD, and - in some but not all studies- older age and ADHD. However, the relationship between depression and social implications of GTS symptomatology and other related neuropsychiatric conditions has not been clearly established yet and more studies are needed.

GTS AND DEPRESSION: ETIOLOGY

While the etiologies of GTS and depression per se have been deeply investigated, few studies have addressed their correlation. Thus it is hard to correctly define the role of one over the other. Epidemiological studies can provide us with a few indirect clues on the best way to understand this relationship.

Current etiological theories for GTS include genetic influences in the majority of cases [61], and suggestions of infections via neuroimmunological mechanisms [62] and prenatal and/or perinatal difficulties [63]. Overall, there is good evidence from both controlled and uncontrolled studies in specialist GTS clinics to support the view that major depressive disorder, depressive illness, and depressive symptomatology are common in GTS patients. Depression in GTS is highly likely to be multifactorial in origin, as is depression in non-GTS populations.

First, GTS can be a distressing condition, particularly if tics are moderate to severe. Depression in GTS patients could therefore be explained, at least in part, by the fact that sufferers have a chronic, socially disabling, and stigmatizing disease. It has been clearly demonstrated that children who are bullied in school may also become depressed [81,82], and a significant proportion of children with GTS are bullied, teased, and given pejorative nicknames, potentially resulting in depression.

Another reason that might play a part in the etiology of depression in clinic GTS patients is that, in GTS, comorbidity with OCD is high. The most common complication of OCD (ranging from 13% to 75%) is depression; in one study, MDD was the most common comorbid disorder in OCD patients, occurring in 38% of subjects [64,65]. Next, ADHD is common in GTS and ADHD has been shown to have a high comorbidity with depression [66]; thus, many GTS patients could be depressed because of comorbidity with ADHD or OCD in the phenotype now named "GTS-plus" [17].

The depression in clinic GTS patients may also be due to the side effects of both typical and atypical neuroleptic medications (e.g., haloperidol, pimozide, fluphenazine, tiapride, sulpiride, and risperidone) as well as others used to treat GTS, such as tetrabenazine, the calcium antagonist flunarizine, mecamylamine, and clonidine [5].

Finally, it has been shown that depression is a common condition [61]. GTS is also more common than was previously recognized. Thus, the two disorders could coexist by chance in some instances. However, given how common major depressive disorder and depressive symptomatology are in GTS patients in specialist clinics (13–76%, compared to the lifetime risk of depression of about 10%) and significantly more so than in matched controls, this is an unlikely explanation in most studies.

In conclusion, the etiology of depression in GTS is highly likely multifactorial, as in primary depressive illness, and is less likely to be caused by a single etiological factor. The exact phenomenology and natural history of depression in the context of GTS, as well as the contribution of depression, if any, to the GTS phenotype, may deserve more research. As with OCD (obsessive compulsive behaviors in the setting of GTS being different in pure or primary OCD), the phenomenology of depressive symptoms may differ between GTS patients and patients with pure or primary depressive disorders [67].

DIAGNOSTIC AND TREATMENT IMPLICATIONS

In conclusion the relationship between GTS and depression is still not fully understood. Nevertheless it is widely accepted that children with GTS have a higher risk of developing mood disorders, but it is not proportional to the severity of GTS itself. As a matter of fact GTS symptoms may decrease during puberty while the risk of developing comorbidity

remains the same lifelong. The causes for this increased risk are not well determined but good consensus exists about the main role of environmental and social factors. Therefore, treating and caring for children affected by both GTS and depression can prove a quite difficult challenge. First, it is mandatory to perform a constant and careful follow-up in children with GTS looking for early signs of depression. Should this happen, it is recommended to investigate and, if possible, to remove any identified etiological factor and/or start appropriate therapy. In this regard, particular attention should be paid to pharmacological interactions and/or antagonisms (e.g. effects on mood of dopamine-antagonist agents). Beyond initial clinical improvement, long-term follow-up of children diagnosed with comorbid GTS and depression is usually required.

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