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Parental attributions concerning the causes and controllability of adolescent depression

Joan E. McDowell

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PARENTAL ATTRIBUTIONS CONCERNING
THE CAUSES AND CONTROLLABILITY OF ADOLESCENT DEPRESSION

by

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Dissertation

Submitted to the Department of Psychology

Eastern Michigan University

in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

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May 2007

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Abstract

The primary purpose of this study was to explore the relationship between parental attributions concerning adolescent depression and levels of negative expressed emotion (EE) within the home by breaking down attributional beliefs into two dimensions: the etiology of the disorder and the controllability of the symptoms. This study included 154 parents of adolescents who filled out an online survey. Participants were predominantly female and identified themselves as White/non-Hispanic. Among the teens, each gender was relatively well-represented. Mean age of respondents was 44.7, and mean age of the adolescents was 16.0. Of the entire sample, 101 were identified as the clinical group (having a teen with depression). The questionnaire included a section developed to measure specific attributions related to depression, the Children's Depression Inventory – Parent Version (Kovacs, 2002), and the Level of Expressed Emotion Scale (Cole & Kazarian, 1988). Results showed that controllability attributions are related to measures of expressed emotion ($p < .01$), while beliefs about causality had mixed results. It is suggested by this study that beliefs regarding controllability and causal beliefs may be less related than previous researchers suspected, and controllability beliefs may be more salient to theories of EE.

TABLE OF CONTENTS

Acknowledgements	iii
Abstract.....	iv
List of Tables	vii
List of Figures	viii
Introduction.....	1
Adolescent Depression	4
Distinguishing Between Depressed Mood, Depressive Syndrome, and Clinical Depression	5
Etiological Models.....	8
Interpersonal Links.....	14
Bi-directional Influences.....	15
Expressed Emotion	20
Attributions and EE	28
Purpose of the Current Study.....	39
Method.....	42
Participants.....	42
Measures and Procedure	46
Results	49
Preliminary Analyses.....	49
Analysis of Differences between Groups	52
Relationship between Causal and Controllability Attributions.....	57
Attributions and EE	58

Controllability Attributions and Individual Characteristics.....	62
Discussion.....	65
Relationship between Causal and Controllability Attributions.....	65
Attributions and EE	67
Differences between Groups.....	72
Individual Characteristics and Controllability	75
Limitations and Future Directions.....	77
Conclusions.....	78
References.....	81
APPENDIX A: List of Recruitment Websites.....	103
APPENDIX B: Recruitment Letter.....	104
APPENDIX C: HSRC Approval.....	105
APPENDIX D: Depression Attribution Questionnaire	106
APPENDIX E: Child Depression Inventory – Parent Version.....	109
APPENDIX F: Level of Expressed Emotion Scale – Relative Version.....	110
APPENDIX G: Factor Analysis of Controllability Items	113
APPENDIX H: Factor Analysis of Expressed Emotion Items	114
APPENDIX I: Mean Scores for Individual Controllability Items by Group.....	117
APPENDIX J: Correlations between Individual Controllability Items and EE by Group....	118

LIST OF TABLES

Table 1. Demographic Statistics	45
Table 2. Mean Ratings for Causal Attributions by Group.....	50
Table 3. Mean Differences in Causal and Controllability Attributions by Group.....	54
Table 4. Correlations between Causal and Controllability Attributions for the Whole Sample	57
Table 5. Correlations between Causal and Controllability Attributions for the Clinical Sample.....	58
Table 6. Correlations between Causal Attributions and EE for the Clinical and Comparison Groups.....	59
Table 7. Correlations between Causal Attributions and EE Subscales for the Clinical Group	60
Table 8. Stepwise Regression Analysis for Variables Predicting EE.....	62
Table 9. Stepwise Regression Analysis for Variables Predicting Controllability Attributions	63

LIST OF FIGURES

Figure 1. The 90% and 95% confidence intervals around the difference
between group means expressed as a percentage of clinical group means.55

Figure 2. Mean ratings for severity of depressive symptoms by chronicity of depression.64

Parental Attributions Concerning the Causes and Controllability of Adolescent Depression

Introduction

Although we know that there is a biological component to depression, relationships within the family may contribute to the development or maintenance of the disorder. One mechanism by which this may occur is through the beliefs that family members hold about depression.

This study measures the attributions that parents make concerning their depressed adolescents. This paper is important because previous work has shown that an individual's social network affects recovery from depression (Ezquiga, García, Bravo, & Pallarés, 1998). As investigators studied mechanisms within the social network, expressed emotion (EE) was linked to outcomes for depression (Asarnow, Goldstein, Tompson, & Guthrie, 1993; Butzlaff & Hooley, 1998; Greene, 1998; Hooley, Orley, & Teasdale, 1986; Licht, 2001). EE consists of criticism, hostility, and over-involvement of family members. Rates of relapse and time to recovery have both been shown to be related to the level of EE within a family. As investigators looked deeper into the construct, criticism was the element most consistently and significantly related to depressive outcomes (Hooley & Licht, 1997). Finding the mechanism that induces critical behavior within the family will prove helpful for developing treatment models as we examine depression from a developmental perspective.

The attribution literature suggests that the beliefs a family member holds about the depression leads to behaviors, such as criticism, within the family system that negatively affect the depressed individual. However, the link between attributions and outcomes has been inconsistent (Hooley & Licht; Licht).

Brickman et al.'s (1982) model of attributions provided the conceptual framework for this investigation. This study addressed the inconsistent relationship between attributions and depressive outcomes by scrutinizing attributions based on Brickman et al.'s attributional theory. This theory suggests that there is a difference between attributions for the origin of a problem and attributions concerning the solution to the problem. These beliefs might be thought of in terms of causal beliefs (the origin) and controllability beliefs (what can be done now to solve the problem). In the case of depression, attributions for the cause of the depression (e.g., biology, personality) may be unrelated to attributions regarding how much control the individual has over the symptoms of depression.

Previous research has linked attributions for controllability to criticism within the families of individuals suffering from mental illness (Hooley & Licht, 1997). In these studies, ratings for controllability and criticism were established from the same interview. Consequently, a measure of criticism was included in the current study to confirm the relationship between criticism and controllability beliefs.

The aforementioned theories were tested in the context of adolescent depression. Little research concerning EE and attributions has been conducted with this population. This research investigated causal and controllability attributions that parents hold for their adolescent with depression. A questionnaire posted on the World Wide Web obtained parental ratings of attributions for depression. This research tested the hypothesis that causal attributions are unrelated to beliefs about the controllability of the depressive symptoms. Second, relationships between parents' controllability attributions, the age of the adolescent, and the depressive symptomatology that is exhibited were explored. Parents' beliefs about the controllability of specific symptoms were expected to be negatively related to the severity

of symptoms and positively related to the age of the teen. Because adolescents commonly experience symptoms that are somewhat different from adults' symptoms, differences between internalizing and externalizing symptoms were explored. Parents' controllability beliefs were expected to be higher for acting out behaviors than the common depressive symptoms listed in the DSM.

Exploring these relationships may expand our understanding of how and when specific attributions begin and how they change for parents of adolescents. This information will be useful in developing treatments for depression that help not only the teen suffering with the disorder, but also other family members who are affected.

Adolescent Depression

Because of resistance to recognizing depression in adolescence as a significant clinical problem until the mid-1980s, little data exist regarding depression in this age group compared to the amount of information available concerning adult depression (Asarnow, Jaycox, & Tompson, 2001). Empirical studies of adolescent depression barely existed as recently as 25 years ago. The flood of interest in adolescence during the past decade led to numerous new journals devoted to publications concerning issues of this age period, as well as a substantial increase in articles on adolescence in existing journals and in the numbers of members of associations dedicated to adolescence (Steinberg & Morris, 2001). Along with this interest in adolescence generally comes an interest in the developmental course of many psychological disorders that have an etiology in adolescence, including depression.

Depression rates increase between childhood and adolescence (Fleming & Offord, 1990). The prevalence rate of depression among primary school age children is estimated at only 2% (Lock, 1996), while a review of epidemiological studies of adolescent clinical depression reported an average of 7% (Petersen et al., 1993). One explanation for this increase in depression is that parents' expectations for individuation and responsibility increase as the child enters adolescence. Parents may be critical of normal teenage mood swings and behaviors, expecting the adolescent to maintain control, which may lead to deeper depression. It is also possible that changes that occur during puberty create neurochemical imbalances that lead to depression or that the developmental needs of adolescents are at odds with societal demands and expectations placed upon them during this period.

Diagnosis of depression in adolescents is difficult due to developmental considerations. Because of the turmoil common during this period, teens often report depressed mood (Steiner & Feldman, 1996). It may be difficult to differentiate between an individual experiencing a mood change versus someone with an affective disturbance. In addition, teens may express negativistic or antisocial behavior during a depressive episode that makes diagnosing depression even more complex (Oster & Caro, 1990). Depression researchers studying adolescents may both under- or overreport rates of depression by failing to ask about symptoms such as irritability and acting out behaviors, or by over-interpreting responses about mood changes that indicate depression in adults but are more normative in adolescents.

Distinguishing Between Depressed Mood, Depressive Syndrome, and Clinical Depression

The array of constructs used in depression research includes mood changes that are common during adolescence and complicate interpretation of results. Various researchers have used constructs such as depressive mood, depressive syndrome, or clinical depression to study this topic. Depression as a mood or symptom refers to a dysphoric mood state (Merrell, 2003). It is a subjective state that is typical across the lifespan and considered transient. Such sadness is common to all individuals at some point and is considered a natural reaction to certain events. Depressed mood is often normative, and there are no conditions on the length of time for classification (Avenevoli & Steinberg, 2001). In addition, no assumptions are made about the presence or absence of other symptoms. However, depressed mood is likely to be linked with other problems, such as anxiety and social withdrawal (Petersen et al., 1993).

Depression as a syndrome is more than a mood state and is associated with additional indicators such as changes in psychomotor functioning, cognitive performance, and motivation (Merrell, 2003). A depressive syndrome is viewed as a group of behaviors and emotions that have been found statistically to occur together in an interpretable pattern (Petersen et al., 1993). The depressive syndrome approach assumes behaviors and emotions are deviations from the norm, while clinical depression is based on assumptions of a disease model (Petersen et al.).

Clinical depression, or depression as a disorder, is the presence of an identifiable syndrome for a specified amount of time that causes significant functional impairment (Merrell, 2003). Although many of the symptoms of depressive syndrome and clinical depression are similar (e.g., depressed mood, low self-esteem, guilt, crying), a clinical diagnosis of depression also requires somatic problems. Measures of depressed mood and depressive syndrome identify large numbers of adolescents who do not meet diagnostic criteria for major depressive disorder (Petersen et al., 1993).

Diagnostic criteria for major depressive disorder (MDD), according to the *Diagnostic and Statistical Manual of Mental Disorders-IV-TR* (American Psychiatric Association, 2000), include the presence of depressed mood or loss of interest in activities for more than two weeks, as well as four additional symptoms from the following list: changes in appetite or weight, sleep, or psychomotor activity; decreased energy; feelings of worthlessness or guilt; difficulty thinking, concentrating, or making decisions; or recurrent thoughts of death or suicidal ideation, plans, or attempts. It is suggested that adolescents may present with an irritable mood instead of a depressed mood. In addition, the symptoms must cause clinically significant impairment in functioning.

Symptom prevalence appears similar in adolescents and adults (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993), but manifestations may differ, such as a greater likelihood that adolescents with depression will present as irritable. In addition, there is an unusually high prevalence of depression and depressive symptoms in adolescents compared to adults. It is unknown whether this may be due to teens' tendencies to overreport, real experiences, or a result of normal adolescent turmoil (Allgood-Merten, Lewinsohn, & Hops, 1990). The range of intensities and combinations of symptoms make diagnosis difficult. In addition, some teens have an internal expression of depression in which they turn their anger inward, while others express their anger externally, directed at family and friends (Oster & Caro, 1990). Comorbid diagnoses are common in depressive disorders, with major depressive episodes in adolescents frequently associated with disruptive behavior disorders, attention-deficit disorders, anxiety disorders, substance-related disorders, and eating disorders (American Psychiatric Association, 2000).

Besides problems with diagnostic criteria and comorbidity, diagnosis may be difficult because teens have varied rates and patterns of development, and an individual's development may differ across domains and contexts (Steiner & Feldman, 1996). Therefore, to determine if there are significant impairments in functioning, there must be consideration of the level of development across contexts. For example, a teen who is high achieving academically may threaten suicide because of relationship problems. Although the adolescent has developed rapidly in the intellectual domain fostering academic achievement, in the social domain, development may be slower, resulting in relationship problems. In order to overcome this problem, there is a need for multiple observers and gathering of information on functioning in all domains. Other observers may have somewhat different views and

contribute to a more accurate picture of the teen's level of functioning. Problems in multiple domains (health/hygiene, interpersonal, mental health, academic/vocational, recreation/leisure) may provide evidence for psychopathology rather than normal development (Steiner & Feldman, 1996).

Because of the range of mood states, differences in development, and differences in depressive symptomatology among adolescents, parents may be likely to have difficulty determining which behaviors are normative and which are symptomatic of depression. Indeed, research has shown that many parents were unable to identify symptoms of depression in their teens (Logan & King, 2002). Among the parents of 44 adolescents identified as having a depressive disorder using a clinical screening instrument, only 11% reported symptoms sufficient for diagnosis. Seventy-nine percent failed to endorse any depressive symptoms in a structured clinical interview. In addition, the presence of a comorbid substance use disorder appeared to mask depressive symptoms even further, making identification of adolescent depression more difficult.

In sum, diagnosis of depression in adolescents is difficult. Symptoms can vary from those expected among adults with depression, and developmental stages may need to be taken into account.

Etiological Models

The biological model for depression suggests that there are genetic and neurobiological abnormalities in the brain leading to an affective disorder (Raichle & Drevets, 1996). According to this perspective, the neural mechanisms in the brain may have a disruption in both structure and function. For example, this may manifest itself as an imbalance of neurotransmitters responsible for communication between cells in the brain.

The most commonly researched biochemicals relating to depression are serotonin and norepinephrine (Oster & Caro, 1990). Other body chemicals such as the hormone cortisol have also been linked to episodes of depression. Some symptoms of depression, including mood problems and physical functioning, suggest that there may be problems with regulation of the hypothalamus. Finally, researchers have provided evidence that there are genetic vulnerabilities since depression appears to run in families (Oster & Caro). Other biological correlates include the hormone changes associated with adolescence. All teens experience these changes, yet the majority do not develop depression. Some have attempted to explain this phenomenon by proposing a developmental approach that stresses how hormonal changes may influence interpersonal experiences (Avenevoli & Steinberg, 2001). Such work looks at links between pubertal timing and depression.

The cognitive model (Beck, 1991) attributes depression to distorted reasoning. Individuals with depression experience a preoccupation with negative attributions and thought processes, as well as exaggerations of interpretations of situations. Self-evaluation, attributions, expectancies, inferences, and recall are permeated by automatic negative thoughts that lead to negative affect and depression. A similar theory based on attributions was originally called the helplessness theory of depression (Seligman & Nolen-Hoeksema, 1987) and later reformulated into the hopelessness theory (Abramson, Metalsky, & Alloy, 1989). Hopelessness theory is much closer to cognitive theories of depression than the original model and purports that negative outcome expectancies and expectations of helplessness about changing outcomes produce hopelessness that leads to depression. Adolescents' increased capacity to reflect on the self and the future is thought to play a role

in the possibility of experiencing depressed mood (Petersen et al., 1993) and may provide some insight into why prevalence increases at adolescence.

The interpersonal model is an extension of learning theories in which it is believed that depression is a failure to receive positive reinforcement from the environment (Oster & Caro, 1990). It includes the idea that an individual may lack the skills for obtaining maximum rewards and minimum punishment through interpersonal behaviors. Based on work such as Bowlby's attachment theory, the importance of social relationships is considered of paramount importance to depressed individuals and should be the focus of treatment (Klerman, Weissman, Rounsaville, & Chevron, 1984).

Each of the preceding models focuses on a specific area of study regarding depression. However, due to the number of factors related to adolescent adjustment and the reciprocal relations among them, efforts to partition out the variability into genetic and environmental components fail to capture the complexity (Steinberg & Morris, 2001). Although a genetic predisposition has been consistently linked to recurrence of depression, it is often argued that social-contextual influences combined with a variety of factors such as a genetic/biological predisposition, a cognitive predisposition, life changes (Avenevoli & Steinberg, 2001), a history of loss and trauma, or earlier developmental problems (Oster & Caro, 1990) may trigger depression. Adolescence is a time of intense change with many developmental hurdles to cross including bodily changes, identity clarification, sexual maturity, and separation from parents (Oster & Caro). In addition, teens face desires for group acceptance, peer pressure, initial exposure to alcohol and drugs, and concerns about academic performance. Ambivalent feelings about these changes may lead to erratic behavior, fluctuations of mood, inconsistency in relationships, and nonconformity (Oster &

Caro). A fully developmental model for depression suggests that the disorder should be looked at from a multidimensional integrative perspective in which biological, cognitive, interpersonal, family, and environmental factors must be taken into account equally (Cicchetti & Toth, 1998). The development of the adolescent is thought of in terms of how these dimensions affect depression and how depression affects further development.

Cicchetti and Toth (1998) proposed an ecological transactional model that recognizes many dynamic and reciprocal relationships between the individual and the environment that affect the likelihood of a depressive disorder. These mutual influences shape the course of child development over time. In this model, vulnerability factors paired with ecological challenges may increase the likelihood of depression. Conversely, the presence of protective factors and ecological buffers may explain why some children do not develop depression even when conditions that have been linked to depressive illness are present. In addition, this developmental model suggests that when children fail to resolve early developmental tasks, it may lead to future difficulties since the adaptation that should have been acquired in the past is required for future developmental challenges. Handling developmental challenges well contributes to preparedness to handle future challenges. In other words, early problems in development jeopardize future development and put children at risk for mental illness. This model takes into account biological systems by taking the position that activation of genes may differ in various developmental periods and at different times for different individuals. Thus, the relative influence of genes and environment at different stages must be considered for each individual.

A developmental view such as this is helpful in interpreting the results of the multitude of studies that have explored various factors by assuming that depression can arise

through a variety of pathways. In this model, various components of depression may develop in biological, cognitive, affective, and interpersonal domains and are integrated within the social ecological system to create a pathway for the development of depression. The concept of multifinality suggests that one source of influence does not necessarily result in one specific outcome (Cicchetti & Toth, 1998). For example, although children of depressive parents are considered at risk, not all children of depressed parents develop the disorder. In contrast, equifinality suggests that the same outcome may stem from diverse sources of influence. For instance, males and females may develop depression due to different causes. Viewing the development of depression in terms of these concepts allows for various pathways for the development of depression.

This model is consistent with the diathesis-stress view of mental illness (Zubin, 1978). This vulnerability model suggests that an individual will develop a mental disorder when there are sufficient stressors to exceed the level of tolerance for that particular individual. Stressors can be either endogenous or exogenous, ranging from biological disruptions within the individual to difficulties in the psychosocial domain (Zubin, 1984). From this point of view, we can identify an unlimited number of combinations of internal and external environmental demands. These may overwhelm a person who has an unlimited number of possible combinations of internal and external events that create the particular level of tolerance the individual possesses. This level of tolerance is made up of the balance of protective factors versus risk factors. The family as a potential source of risk and protective factors suggests the importance of exploring family factors involved in the development or maintenance of depression.

Family approaches to adolescent depression recognize the family at the core, while at the same time appreciating a full range of influences that affect the individual, including both biological and individual psychological processes (Robbins, Mayorga, & Szapocznik, 2003). This paper addresses the topic of adolescent depression from this perspective. While recognizing the impact of a large number of variables, focusing on family factors appears to offer a promising avenue for treatment of the disorder because of the relationship between depression and social support, which is discussed in the following section.

Interpersonal Links

It is well known that life events and the social environment affect mood. Depression often follows major negative life events such as divorce, death of a loved one, or loss of a job (American Psychiatric Association, 2000). These events can cause changes in the individual's social environment and, therefore, an individual's mood. The strong inverse relationship between the quality of close relationships and depressive symptomatology has been demonstrated, with research encompassing many types of subjects and many disciplines.

For example, social support, particularly the size of the social network and everyday psychological support from a partner, has a strong relationship to recovery in adults with major depression (Ezquiga et al., 1998). Likewise, Cronkite, Moos, Twohey, Cohen, and Swindle (1998) found that an individual without social resources is at considerable risk for partial or non-remission. While most of this branch of research has focused on adults, there are some studies that suggest similar patterns in teens and children. Among adolescents, Field, Diego, and Sanders (2001) identified parental relations, affection, and intimacy as important contributors to teen psychological health. Critical communication within the family has also been associated with poorer outcomes for depressed youth (Asarnow et al., 1993).

Although the interpersonal aspects of depression have been the subject of investigations for many years, the social support mechanisms for development and maintenance of the disorder are still poorly understood. Attributions and expressed emotion (EE) are two recent areas of inquiry that show promise in furthering the understanding of depression in the context of the social support system. The theory behind these concepts is

that beliefs about the disorder affect the manner in which family members behave toward the depressed relative and the likelihood of reacting with criticism, hostility, and over-involvement. This chapter reviews the interpersonal aspects of depression and then focuses on the literature concerning attributions and EE.

Bi-directional Influences

The strong association between depression and social factors raises the question of whether there is a causal relationship. There may be a direct causal effect of interpersonal dysfunction on depression, the depression may trigger relationship difficulties, or there may be a bi-directional influence between the disorder and the dysfunction. The research literature indicates that a reciprocal model of effect for depression and interpersonal factors is most promising. The environment affects mood; however, mood also influences social functioning and one's environment (Markowitz, 1998).

Coyne, Kahn, and Gotlib (1987) describe an interactional theory that explains how families affect and are affected by depressed family members. This interactional theory suggests that the demands of depressed persons for reassurance and support contribute to rejection. For instance, depressed individuals may be preoccupied with thoughts of others leaving them. Or reassurance-seeking behavior is an attempt to offset feelings of hopelessness. Their excessive demands for reassurance may serve to decrease empathy from nondepressed family members which, in turn, is perceived as a lack of support, worsening the depressive symptoms. Research has demonstrated that excessive reassurance-seeking and depressive mood contribute to negative attitudes from those around the depressed individual (Benazon, 1998). In a study of dating relationships, for example, men were more likely to exhibit relationship discord when partners reported depressive symptoms, reassurance-

seeking, and interest in negative self-relevant feedback (Katz & Beach, 1997). The reciprocal effect of environment and mood serves to maintain the illness. Living with a depressed individual creates an environment in which patterns of negativity, hostility, and criticism may become pervasive.

For example, the environment can affect mood when the actions of an overprotective spouse create threats to self-esteem and reduce feelings of self-efficacy (Holahan, Moos, & Bonin, 1999). Conversely, mood influences the environment as a depressed individual's mood and the mood of those around him/her become correlated. Coyne (2001) found that subjects who had spoken to depressive patients were more depressed, anxious, hostile, and rejecting.

These findings suggest that the environmental response to an individual with depression may play an important role in the maintenance of depressive symptoms. Even when there are no existing causal factors, familial factors that exacerbate or maintain depression may develop over the course of the illness. The link between depression and interpersonal factors has important implications for treatment and signifies the need for further exploration into the reactions of family members.

The reactions of family members. The burden associated with having a family member with a mental illness has been well documented (Baronet, 1999; Perlick et al., 1999). Many studies have shown that significant others of patients with depression have an increased risk for distress themselves (Benazon, 2000; Coyne, Thompson, & Palmer, 2002; Coyne, Wortman, & Lehman, 1988). The effects on quality of life for family members in terms of family relationships, friends, social life, and work prospects are generally negative (Fadden, Bebbington, & Kuipers, 1987; Hill, Shepherd, & Hardy, 1998; Yarrow, Schwartz,

Murphy, & Deasy, 1955). When children are involved, parenting roles may shift, with parents having to assume more responsibility for the depressed child in addition to taking care of regular responsibilities. Caregivers report stress, anxiety, and depressive symptoms of their own, as well as negative effects on their self-esteem and confidence that often fluctuates with their relative's mood state (Hill et al., 1998). Similarly, Coyne et al. (1987) found that living with a person in a depressive episode produces numerous burdens in response to the patient's symptoms that lead to psychological distress for the caregiver. Tessler and Gamache (1994) reported that the greatest caretaking burdens fall on spouses and parents who live with mentally ill individuals.

After finding out that a person they love has been diagnosed with a mental illness, family members must learn to cope with intense emotional responses to the illness (Karp & Tanarugsachock, 2000). A qualitative study that explored the manner in which family members respond to mental illness described several processes that families often experience as they adjust (Rose, Mallinson, & Walton-Moss, 2002). After initial diagnosis, one of the first processes the family faces is confronting the ambiguity of their situation. This includes limiting other regular family experiences as their time is consumed by caregiving. Families in this study reported intense disruptions in the family at this stage.

Family members in the Rose et al. (2002) study attempted to gain control of the illness and influence its course. This process included deciding who was responsible for managing the symptoms of the illness and what aspects could be managed. In this study, there were wide variations concerning the assignment of responsibility for symptom management. Some family members described instances in which they had reached a breaking point and felt they could not tolerate certain behaviors from the ill family member,

while others had accepted the idea that their ill relative would never be normal or completely responsible for their behaviors. These responses would undoubtedly engender a variety of reactions to the ill family member, ranging from sympathy to hostility.

In effect, caretakers have to monitor their relative's behavior while being extremely sensitive to their own reactions. Family members often find that they have to "walk on eggshells" because of the depressed person's sensitivity to criticism (Hill et al., 1998). Attempts to support and care for the individual with a mental illness can fail as emotional overinvolvement breaks down proper functioning within the family (Coyne et al., 1988). Families may also find that they are caring for the individual without much help, education, or training (Hill et al.).

These burdens and the resulting distress may create dysfunctions that subsequently affect the course of the illness. Coyne, et al. (1988) found that the quality of others' involvement and support deteriorates over time. New patterns of relating within the family may be formed, resulting in a situation that exacerbates or maintains the depressive symptoms.

Family correlates for adolescent depression. During the 1970s, researchers began taking context into account in asking what effect family members' behaviors have on adolescents with depression (Oster & Caro, 1990). Consistent with interactional theories, family systems theories suggest that the teen is affected by and in turn affects the family unit. Family variables have been linked with adolescent depression and can be viewed as having a causal relationship, a maintaining relationship, or a consequence of the disorder (Foster & Robin, 1998; Wamboldt & Wamboldt, 2000). Some of the family factors that have been

associated with depression include criticism, parental warmth, support, negativity, and conflict. Results of several studies regarding these variables follow.

One study designed to explore the specificity of risk factors for depression compared a group of adolescents with MDD, a group with a substance use disorder (SUD), and a control group with no disorder (Lewinsohn, Gotlib, & Seeley, 1995). This study found that social support from family was specific to depression. A variable was considered specific to depression if it differentiated between the depressed and control groups, but not between the SUD and control groups. Another investigation showed that teens that scored above the clinical cutoff for depression on the Center for Epidemiological Studies Depression Scale (CES-D) had poorer parental relations than those below the cutoff (Field et al., 2001).

Although studies such as these demonstrate the importance of family relationships, further investigations have identified specific aspects of the family that are related to adolescent depression. A 2-year longitudinal study of adolescents and their mothers used teen and mother reports, as well as participation in two problem solving interactions, to examine the current and prospective nature of the relationship between family functioning and adolescent depression (Sheeber, Hops, Alpert, Davis, & Andrews, 1997). Less supportive and more conflictual family environments were associated with greater symptomatology, both concurrently and prospectively, which strongly suggests a causal influence for depression of low support and high conflict within families. Similarly, a four-wave longitudinal study of 451 families found that parents of 10th graders with depressive symptoms were more hostile, less warm, and less effective disciplinarians when the adolescents were in 7th, 8th, and 9th grades compared to parents of adolescents without depression (Ge, Best, Conger, & Simons, 1996).

A study of 708 families that included a large proportion of twins examined family relationships, depression, and the impact of nonshared environments (Reiss et al., 1996). The investigators used self-report measures and videotaped problem resolution exercises to rate parenting variables. After adjusting for level of conflict/negativity toward a sibling to remove shared variance, regression analyses showed that 37% of the variance in depressive symptoms was accounted for by conflictual and negative parental behaviors directed specifically at the depressed adolescent. Conversely, there was less pathology in teens when conflict/negativity was directed at their sibling, suggesting this may be a protective factor. Warmth and support were also important correlates with depression in adolescents, but effects were lower than those for conflict and negativity. Monitoring and control had no significant effects on adolescent symptoms.

These studies indicate the importance of family functioning, in particular conflict and negative parental behaviors, as it affects adolescent depression. However, analyzing these factors further will pinpoint areas for improving the model of adolescent depression and targeting interventions.

Expressed Emotion

One of the patterns of behavior in a family that may be counterproductive to recovery from mental illness has been labeled expressed emotion (EE) (Brown, Birley, & Wing, 1972). This concept was developed in the United Kingdom in studies examining the influence of family life on schizophrenic outcomes. Research has also explored the relationship of EE to depression (Asarnow et al., 1993; Butzlaff & Hooley, 1998; Hooley et al., 1986), as well as a wide range of other illnesses such as eating disorders (Butzlaff & Hooley; le Grange, Eisler, Dare, & Hodes, 1992), obsessive-compulsive and panic disorders

(Chambless, Bryan, Aiken, Steketee, & Hooley, 2001), intellectual difficulties (Sabarese & Todman, 2005), and alcoholism (O'Farrell, Hooley, Fals-Stewart, & Cutter, 1998).

Hooley and Campbell (2002) describe EE as having three components: criticism, hostility, and emotional over-involvement (EOI). High levels of EE correspond to critical, hostile, and over-involved behaviors. Low EE is characterized by empathic, calm, and respectful conduct (Leff, Kuipers, Berkowitz, & Sturgeon, 1985). Criticism is the factor emerging as the most predictive of mental illness outcomes (Hooley & Teasdale, 1989; le Grange et al., 1992; López, Nelson, Snyder, & Mintz, 1999).

Early EE studies chiefly utilized the Camberwell Family Interview (CFI) to assess expressed emotion between patients and caregivers (Kazarian, 1992). The CFI is a semi-structured interview conducted with the relatives of patients and is designed to elicit feelings and reactions about the illness and the patient. This interview is taped for later coding by trained raters. Because the interview takes 60-90 minutes and months of training for administrators and raters, alternative methods of measuring EE have been developed. Self-report methods admittedly rate perceived EE rather than objectively measured behaviors. However, the concurrent and predictive validity of several of these measures appears to be good (Donat, 1996), and a factor analysis of the EE construct reported that the criticism component of EE appeared to measure the same construct whether assessed by self-report, relative, or behavioral observations (Chambless, Bryan, Aiken, Steketee, & Hooley, 1999).

Criticism is based on negative comments about the patient or the relative-patient relationship and typically takes into account content and voice tone in interviews with the relatives (Brown et al., 1972). Critical content includes a clear statement of resentment, disapproval, or dislike. Hostility is so closely correlated with criticism that many researchers

have dropped it from consideration, and EOI has shown equivocal relationships with outcomes (Chambless et al., 1999). EOI is a troublesome concept to measure due to its complex nature. Although emotional overinvolvement is considered a negative factor for illness outcomes due to the presence of controlling or intrusive behaviors, it is possible that it also measures relatives' concerns for the patient. In other words, we would expect that relatives who are very caring would exhibit greater involvement with their ill relative, that is, greater EOI. The patient then interprets this in a positive rather than a negative manner. This may help to explain the lack of consistency regarding the predictive validity of EOI. In addition, when exploring EE among families with ill children or adolescents, involvement with the child may be developmentally appropriate rather than pathological. The following review of EE research points out some promising areas for study as well as difficulties in this field.

The evidence linking EE with depressive outcomes among adults appears to be strong whether assessed with the CFI (Licht, 2001) or an alternative measure (Donat, 1996). In one 9-month follow-up study linking high levels of EE with relapse rates among depressive inpatients, Hooley et al. (1986) found that high EE, marital distress, and the patient's perceptions of criticism by spouses were associated with depressive relapse. In fact, none of the patients in a family with low levels of EE relapsed, whereas 59% of persons living in a family with high levels of EE relapsed. A meta analysis of 27 studies confirmed that EE is a reliable predictor of psychiatric relapse (Butzlaff & Hooley, 1998). In this study, EE was an even stronger predictor for mood disorders than for schizophrenia.

The EE-outcome association holds across many types of relationships. Studies have shown that outcome can be predicted by EE levels between spouses (Gerlsma & Hale, 1997;

Hooley & Teasdale, 1989), by levels of EE in parent-child relationships (Asarnow et al., 1993), and parent-adolescent relationships (McCleary & Sanford, 2002). There is a sizeable body of literature concerning the effects of EE on mental illnesses among adults (Hooley & Licht, 1997; Hooley & Teasdale; López et al., 2004). The number of studies concerning adolescents is much lower, which may be due to the origins of EE research among schizophrenic spectrum disorders, which typically begin after adolescence. The dearth of studies regarding adolescent depression and EE may also be due to the lack of interest in adolescent depression until recent years, or due to the belief that adolescent depression is similar to depression among adults and, therefore, not necessary to study. However, we may be able to draw some conclusions from a few recent studies that have examined EE or aspects of EE among youth.

One study of 273 children aged 6-19 showed that maternal-expressed emotion was associated with a three-fold increase in the likelihood of having either a depressive disorder, substance abuse, or conduct disorder (Schwartz, Dorer, Beardslee, Lavori, & Keller, 1990). Another study used a family problem-solving task to investigate the interaction styles of families with psychiatric inpatients aged 7-14 (Hamilton, Asarnow, & Tompson, 1999). Data indicated that parental attitudes were more critical toward the child among parents of depressed children than among the normal control group. The children in this study exhibited more negative and guilt-inducing behaviors than did schizophrenic-spectrum children or controls. The investigators concluded that family members with depressed children are subjected to higher levels of stress than other families. This raises the question of whether the behaviors of the depressed child invite negative parental responses such as criticism. Another study that linked EE with mental illness among children aged 6 to 18 revealed that EE was

specific to depression as compared to a control group and a group with attention-deficit hyperactivity disorder (ADHD) (Asarnow, Tompson, Woo, & Cantwell, 2001). Several studies have examined EE among depressed children with comorbid disruptive behavior disorders (Asarnow, Tompson, Hamilton, Goldstein, & Guthrie, 1994; Hirshfeld, Biederman, Brody, & Faraone, 1997). One study revealed that high maternal criticism was associated with externalizing behaviors as well as the total number of mood and behavior disorders (Hirshfeld et al.). One group of researchers found not only that EE was significantly higher in families of depressed children compared to families of children with early-onset schizophrenia spectrum disorders or control families, but also that depressed children with comorbid disruptive behavior disorders were most likely to have high levels of EE in the home (Asarnow et al., 1994).

The predictive nature of EE has begun to be established in the child literature as well. A study examining 26 psychiatric inpatient children aged 7-14 diagnosed with a mood disorder found that children returning to high EE homes were unlikely to recover during the year following hospitalization (Asarnow et al., 1993). In the only study found that investigated the relationship between EE and adolescent depression, McCleary and Sanford (2002) found that high EE within a family was not predictive of a worse clinical course for depression. However, when participants with comorbid ADHD were removed from the sample, low EE predicted remission among the depressive adolescent sample. This finding again shows specificity for the relationship between EE and depressive disorders.

In addition, patient characteristics appear to be a factor in EE. A study of adult schizophrenic patients revealed that the association between EE and relapse was stronger for more chronically ill patients (Butzlaff & Hooley, 1998). Similarly, a study of patients with

bipolar depression revealed that patients in families with more criticality had more symptoms after ten months than those in families with low EE (Greene, 1998). In the child literature, high EE was associated with a greater number of symptoms among depressed adolescents (McCleary & Sanford, 2002). A metaanalysis of 27 studies also confirmed that the association between EE and relapse is stronger when the sample includes more chronically ill patients (Butzlaff & Hooley). This indicates that high levels of EE may develop over time as family members are exposed to depressive symptomatology.

Indeed, this idea was put forward in the earliest EE literature regarding schizophrenic patients (Brown et al., 1972). Although the association between EE and outcomes may have led to a conclusion that familial EE caused illness, the investigators cautioned against presuming that parental childrearing practices had a causal effect, suggesting instead a reactive model in which EE develops in response to illness. In depressed individuals, a depressive communication style (hostile, argumentative, and demanding), self-disclosure, and negative facial expressions have been shown to evoke unfavorable reactions (Schwartz, Fair, Salt, Mandel, & Klerman, 1976). Therefore, inappropriate self-disclosure and complaining guarantees that the family attends to and confirms the individual's negative qualities. It becomes easier for family members to deliver negative feedback as they react to depressive symptoms, thereby raising the level of EE within the family. The fact that EE appears to have as strong an influence in married couples as in parent-child dyads argues against EE as a factor in a developmental etiological model.

Interventions to help friends and relatives caring for people with depression generally share the core idea of reducing levels of EE (Leff et al., 1985). Family-based treatments that reduce EE have shown an associated reduction in relapse rates (Asarnow, Tompson et al.,

2001). Thus, understanding and recognizing the causes of EE in families with a depressed loved one may help in developing treatment programs that target specific beliefs and behaviors.

Assessment of family factors. Although most of the early EE literature was conducted using the Camberwell Family Interview, more recently investigators have searched for alternative measures due to the difficulty in administering and scoring the standard interview method. With the establishment of simpler valid measures for EE, assessment of this construct may become more widespread, including use in clinical settings for which the interview method is not feasible.

Researchers began to search for new measurements for the EE construct (Hooley & Richters, 1991). However, some warned of the dangers in assuming the predictive validity of new measures and called for research establishing predictive as well as convergent validity (Hooley & Richters). Initial attempts to create new methods relied on identifying aspects of the interview that provided the most information and abbreviating the interview to include only these features. A coding system was developed for the Five-Minute Speech Sample (FMSS) to create an alternative to the CFI (Magaña et al., 1986) that has been broadly used for the study of EE. Administration of the FMSS involves instructing participants to speak for five minutes about the patient relative and how they get along together. Recordings of this speech sample are coded by trained raters in a manner similar to the CFI. Although this substantially cut down the time required for administration, this method still requires the use of a trained rater. Later, researchers sought to identify self-report questionnaires that measure the EE construct (Hooley & Richters). Pre-existing questionnaires of family functioning and newly created tests have been explored for convergent validity with the CFI and for

predictive validity for mental illness outcomes with several promising possibilities emerging. The Level of Expressed Emotion Scale (LEE) (Cole & Kazarian, 1988) is one such measure that has demonstrated convergent (Kazarian, Malla, Cole, & Baker, 1990) and predictive validity (Cole, 1992; Donat, 1996; Gerlsma & Hale, 1997).

Most rating scales associated with the measurement of EE have the patient evaluate the level of family functioning, although in earlier research, ratings of interviews were generally based on family members' reports. The convergence of family member ratings of EE with the CFI were tested with the LEE in a sample of schizophrenic patients and their families (Kazarian et al., 1990). The investigators reported that the relatives' self-report measure did evaluate the family environment, with the highest correlations occurring between the LEE and measures of criticism on the CFI.

One difficulty with the measurement of EE is that relative and patient reports may not converge. The standard measure of EE (the CFI) is based on parent verbalizations concerning events within the family, while more recently developed rating scales tend to measure patients' perceptions of family events. However, several scales that measure relatives' perceptions have been studied and demonstrate similar correlations with the CFI (Kavanagh et al., 1997; Kazarian et al., 1990).

Another concern is that the reports relatives make concerning their relationship with their ill relative may not relate to actual behaviors between family members. In other words, we cannot infer that relatives direct criticism at ill family members based on the fact that relatives express frustration or criticism to researchers. Investigators exploring patterns of behavior in families having an adult member with schizophrenia compared results of the FMSS with observational ratings of family interactions during a family problem-solving

discussion to explore this possibility (Hahlweg et al., 1989). Data indicated that relatives rated high in criticism exhibited more negative affect and behavior than relatives low in criticism. In addition, these families had extreme negative escalation patterns. If one member responded negatively, the probability was high that another member would continue the pattern of negativity with up to 20 behaviors in the sequence, while families low in criticism did not extend any negative sequences beyond six.

Similarly, a study of parent-child interactions among families with a child between 7 and 17 who had been referred for outpatient treatment indicated that the critical component of EE predicted negative parental behaviors (McCarty, Lau, Valeri, & Weisz, 2004). Parents rated as highly critical as measured by the FMSS exhibited harsher behaviors towards their children during the problem-solving task than did parents who were rated lower. These behaviors consisted of antagonistic, negative, and disgusted interactions with the child. On the other hand, EOI was not linked to observable behaviors in this study. This lends support to the idea that this construct may not apply to children and teens since involved behavior of parents may be developmentally appropriate for these populations.

Attributions and EE

The adjustment for families of individuals with depression is difficult because of enduring social and cultural stigma and the tendency to perceive mentally ill persons as weak, dangerous, and unpredictable (Crisp, Gelder, Rix, Meltzer, & Rowlands, 2000). Many people view depression as a sign of weakness, not an illness. Embarrassment, shame, and denial create a poor environment for adjusting to living with a mentally ill family member. Coyne et al. (1987) assert that while depression initially evokes sympathy and support, it subsequently produces impatience, frustration, and withdrawal by those in the environment

as they come to see the symptoms as willfully unpleasant. Benazon (2000) found that those around the patient become hostile and critical and withdraw from what they see as morally weak behavior.

In addition, many practitioners deal with families on the basis of etiological assumptions about their role in the causation of the illness. One study found that almost half of social work practitioners believe that the aim of therapy for severe mental illness should be to get family members to recognize their own culpability in their family member's illness (Rubin, Cardenas, Warren, Pike, & Wambach, 1998), even though there are indications that doing so would be ineffective, create stress and burden for the family, and perhaps be harmful to the individual with the disorder (Lefley, 1998).

Investigators have proposed that the attributions that caregivers hold about the illness and symptoms affect the level of expressed emotion between the caregiver and the patient (Hooley & Licht, 1997). Researchers have confirmed a link between attributions and EE in studies of depression (Hooley & Licht), as well as other disorders such as schizophrenia (Barrowclough, Johnston, & Tarrier, 1994; Brewin, MacCarthy, Duda, & Vaughn, 1991; López et al., 2004; Weisman, Nuechterlein, Goldstein, & Snyder, 1998), bipolar disorder (Wendel, Miklowitz, Richards, & George, 2000), and Alzheimer's disease (Tarrier et al., 2002). High levels of expressed emotion (EE) are related to familial attributions in which mentally ill persons are perceived as being in control of and accountable for their illness or their symptomatic behavior. In a study of 43 spouses of psychiatric inpatients who met criteria for major depressive episode, Hooley and Licht found that criticism was the dimension of EE that was most strongly linked to these negative attributions. This finding is consistent with the important role that criticism has taken in EE research.

Because attributions are related to EE, we would expect measures of attributions to have predictive power for relapse similar to EE measures. Yet previous research results are inconsistent regarding the predictive validity of attributions. Research has been conducted that suggests a link between attributions and the course of illness (Barrowclough et al., 1994; Casten et al., 1999; López et al., 2004). In Casten et al.'s study of geriatric psychiatry inpatients, having fewer depressive symptoms that the caregiver perceived to be within the patient's control predicted remission of the depression at discharge. Another study of patients with schizophrenia showed that attribution variables were better predictors of relapse than were EE measures (Barrowclough et al., 1994). However, there are also studies that have found no link between attributions and risk for relapse (Hooley & Licht, 1997; Licht, 2001).

These conflicting results may be due to differences in the composition of the samples. Casten's (1999) sample was composed of caregivers for geriatric inpatients, while the two studies that did not show predictive power used participants related to depressed individuals with a much lower mean age, of whom only about half had been hospitalized. Therefore, severity of depression and the level of exposure participants had to the depressed individuals may account for the difference in results. Barrowclough et al.'s study (1994) lends support to this possibility by reporting that the attributions that were made concerning the patients varied by illness chronicity and severity.

Another possible explanation is that the attributions under study were somewhat different. Are attributions concerning the cause of the illness or attributions concerning the controllability of symptoms more related to the course of depression? Different constructs of causal attributions and attributions of controllability of symptoms may have important implications for caregiver and patient outcomes.

Many previous studies in this area of research have used a five dimensional model of attributions composed of internal/external, global/specific, stable/unstable, personal/universal, and controllable/uncontrollable dimensions, or some combination of these (Barrowclough et al., 1994; Hooley & Licht, 1997; Licht, 2001). Beliefs that fall in the internal, global, stable, personal, and controllable dimensions are generally considered negative attributions in which relatives assigned responsibility to the patient for the event. Part of the difficulty in interpreting the results of these studies is that the attributions coded into these dimensions incorporate beliefs concerning past events and present events. In most studies, participants' reports of causal attributions for the disorder and attributions of responsibility for current symptoms are not distinguished. In other words, when a person attributes controllability to an individual with a mental illness, it could mean that the individual should have been able to control the onset of the disorder, or that the individual should be responsible for recovery from the illness or maintenance of the symptoms. This distinction may prove important considering that one attribution is for a past event and the other is for a present or ongoing event.

Furthermore, attributions are sometimes blurred by definition. Individuals are assumed to be responsible for internal causes, while external causation relieves the person of responsibility for causing the problem. However, in some studies, biological causes are included in the internal dimension because the cause originates within the individual (Bolton et al., 2003; Hooley & Licht, 1997). In this case, the assumptions of the internal/external dimension break down because the individual may not be blamed for a biological cause. In order to be rated as an internal cause, the attribution is based on the belief that the cause is a feature (psychological, physical, or behavioral) of the patient (Bolton et al.). By definition,

this combines intrapersonal and genetic causation into the same category. Some authors have noted difficulty in coding statements regarding the illness as a cause of problems because it may be unclear whether family members have an internal or external model of the illness (Tarrier et al., 2002). A remark concerning etiology would normally be coded as internal. However, family members may be blaming the illness for a negative outcome, not the individual, which would prompt the rater to code the comment as external. Likewise, the assumed relationship between negative dimensions breaks down when we consider the case of laziness. Stability is assumed to correlate with attributions of uncontrollability (Brickman et al., 1982). Traits such as habitual laziness may be considered a stable attribution; however, most people would assume an individual had control in this case.

Brickman et al.'s attributional theory (1982) suggests four models regarding personal responsibility that take into account the difference between blame and control. Blame is when an individual is held responsible for the origin of a problem, a past event. Controllability is holding a person responsible for the solution to a problem, a future event. People can believe that individuals are responsible for future solutions regardless of whether they are responsible for the cause of the problem. The first model, the moral model, proposes that an individual is responsible for causing and solving problems. The compensatory model suggests that responsibility for solving, but not causing, problems lies with the individual. Responsibility for causing problems, but not for the solution to the problems, is the theme of the enlightenment model. Finally, the medical model suggests that an individual has no responsibility for causing or solving their problems. Kleinke and Kane (1997) researched how people assigned responsibility for mental disorders to individuals with mental health problems. In general, people assigned more responsibility for solving problems than for

causing them, consistent with the compensatory model. In the context of depression, the etiological beliefs of caretakers are the assigned responsibility for causing the problem, or the causal attributions. Beliefs concerning the amount of control an individual has over symptoms of depression are equal to the expected responsibility for solving a problem, or controllability attributions.

It appears that causal beliefs change over time. Previous research by Robinson (1996) found that biogenetic causal attributions were the most highly endorsed responses for clients and family members who had been involved in a program emphasizing a strong biological and genetic cause for depression. Likewise, more clients were found to hold etiology beliefs similar to their therapist after counseling than before counseling (Atkinson, Worthington, Dana, & Good, 1991). Read and Law (1999) found that undergraduate students' etiology beliefs changed after a series of four lectures presenting the causes of and solutions to mental health problems. In other words, beliefs about the cause of a disorder can change in response to training. Similarly, evidence suggests that beliefs change with exposure to the mentally ill. Volunteers working with the mentally ill were given Rotter's Internal-External Locus of Control Scale before their volunteer work began and three months later (Miller, 1974). Their pre-work scores did not differ from a control group. After working with the mentally ill for three months, the volunteers had higher measures of internal control than did the control group. This contradicts the current thinking that people maintain one attributional style.

Only one study was found that divided attributions among causal and controllability beliefs for the purpose of finding out whether attributions for the illness in general (cause) and the symptoms (controllability) were similar (Brewin et al., 1991). Other studies broke down attributions by type in order to compare the number of statements made in each

category or to compare the differences in attributions by type of symptom (positive versus negative). However, exploring whether the relationship between attributions and EE changes by type (causal versus controllability) may provide a greater understanding of the specific beliefs that affect EE and illness outcomes. In the Brewin study, spontaneous comments during interviews with 26 relatives of patients with schizophrenia were divided into four classes: onset or exacerbation of the illness; negative symptoms (slowness, apathy, self-neglect); antisocial behavior (anger, rudeness, self-harm); and interpersonal problems (resentment, lack of assertiveness, dependency). Repeated measures analyses of variance for these categories indicated that interpersonal problems were rated as more stable and more personal to the patient than the category for onset of illness. Since depressive symptoms would often fall into the category of interpersonal problems (as defined here), we can presume that we would find differences in causal and controllability attributions (as defined by this current study). Thus, the inconsistency in the research on attributions may be due to confusion about whether attributions are for the cause or solutions for problems, as well as the belief that attributions are a stable style or trait.

A more recent study (Hooley & Campbell, 2002) sought to isolate and investigate only controllability attributions. The investigators measured these beliefs in families with a depressed member and families with a schizophrenic member and found that in both groups the relatives rated as high EE attributed more controllability to the patient than the relatives rated as low EE. Similar results have been found in other recent studies of families with a schizophrenic member (López et al., 2004; Weisman, Nuechterlein, Goldstein, & Snyder, 2000) or a family member with bipolar disorder (Wendel et al., 2000).

It appears that caregivers' attributions about the cause of depression, as well as their beliefs about the controllability of various behaviors associated with depression, can be crucial in helping the individual recover from depression. Further study of attributions that family members make concerning depressed persons is necessary to clarify the unexpected results in the literature regarding the predictive validity of caregivers' beliefs. It is probable that there are various dimensions that affect how much impact relatives' attributions have on a depressed person. Although a relationship between high EE and negative attributions has been established, Hooley and Campbell (2002) discovered that, even among relatives with a negative attributional style, most believed the patient was genuinely ill. Furthermore, the relatives believed that not all of the depressive symptoms were under the patient's control. This suggests that family members may have different attributions for the disorder in general than they have for specific symptoms of the disorder. In other words, causal attributions may be independent from the controllability of symptoms. Furthermore, studying whether attributions change based on individual characteristics regarding the illness may provide some insight into better treatments for depression and shed light on the nature of attributions as causal, mediating, or moderating variables.

Attributions regarding youths. This study examined these constructs in the context of adolescent depression. There are a few studies (described above) that confirm the robust link between EE and illness outcomes among youth, but research is needed concerning the role that attributions may play. At this time, attributional research concerning adolescents has not been conducted. In addition, only one study regarding younger children was identified that explored this topic directly (Bolton et al., 2003). The study was designed to examine the relationship between EE and attributions in mothers of children referred for problem

behavior. Consistent with the adult literature, the investigators found that high EE mothers were more likely to make attributions for the problem that were personal to and controllable by the child. As for the predictive validity of attributions among youth, researchers in an early study regarding parental attributions and treatment outcomes in Australia (Watson, 1986) interviewed 42 parents of children referred for psychiatric treatment. The data suggested that attributions internal to the child predicted worse clinical outcomes based on a standardized behavior checklist than attributions that were external to the child or considered situational. However, this study only speculated about the effect that attributions may have on parents' emotions and the role that this would play in family dynamics.

For further data, we can turn to the literature on parental attributions of children's normal behavior in order to make inferences about how these processes may play out when there is a psychological disturbance. In a study designed to measure mothers' attributions regarding their children's social behavior and personality characteristics (Gretarsson & Gelfand, 1988), investigators interviewed 60 mothers randomly selected from a pool of mothers with children between 4 and 12 years old in public school in a western city. Mothers generally viewed positive characteristics of their children as stable and inborn and negative characteristics as transitory. However, mothers with children that they rated as difficult to manage reported that their child's negative characteristics were stable and inborn. In other words, mothers with a difficult child have a more negative view of the child than do mothers who do not have a difficult child.

As enumerated above, the effects of caring for a depressed family member include stress and depression. The behaviors of the depressed individual are often viewed in a negative manner. This seems even more likely among youth, considering that children often

exhibit irritability or acting out behaviors in the midst of mental illness. Taken with the results of the study described above, we can expect parents to view their ill child as difficult and have corresponding negative attributions.

Another study of parenting behaviors in the nonclinical population examined types of discipline that parents employ in relation to the attributions they make for a child's behavior (Dix, Ruble, & Zambarano, 1989). The data suggested that the more strongly mothers believed that a child had the capacity to act appropriately and was responsible for their behavior, the more likely the mothers were to become upset and use negative affect while disciplining the child. In addition, it was found that mothers were more likely to be upset and to use negative affect while disciplining if the child was older rather than younger. Although this study used a survey method, the mothers reported the behaviors that they expected to use in such situations. This provides evidence that not only do attributions affect parents' emotions, but parental beliefs also may affect their actual behavior.

The implications for the current study are that negative attributions for a child's behavior appear to have an effect on parental emotions and may lead to negative parental behaviors (high EE). When depressive symptoms occur in adolescents, parents may exhibit critical behaviors linked to their beliefs that the symptoms should be under the control of the teen, rather than identified as behaviors related to a mental disorder. Because adolescence is typically perceived as a period of turmoil, it seems likely that parents may not identify teen behaviors as symptomatic of depression, but rather attribute them to the developmental stage. The resulting frustration or irritation at the teen's behavior could engender criticism and other behaviors associated with high EE. This change in family functioning may lead to the development of high levels of EE that serve to maintain the illness.

In sum, changes associated with depressive symptomatology may set the stage for negative familial behavior. Depressed persons may exhibit depressive behaviors such as complaining, social withdrawal, and fatigue, which are attributed as personal to or controllable by the ill individual, thereby inducing negative affect and rejection from others. Ultimately, this pattern of responding may result in increased distress for both the patient and their social network by increasing family functioning difficulties such as EE and negative attributional style. These interpersonal dysfunctions may have a cumulative effect over time, turning into ongoing patterns of behavior. Such cycles lock depressed persons into longer or more serious episodes of depression, as well as increase the risk for future episodes. The fact that there are differences in the length of time symptoms are remitted suggests the presence of mediating or moderating factors. Further exploration of these factors may lead to a better understanding of the course of depression, as well as a means for improving the well-being of family members living with a depressed person.

Purpose of the Current Study

This study examined new aspects of the attribution-EE relationship by breaking down attributional beliefs into a dimension regarding the etiology of the disorder and a dimension regarding the controllability of the symptoms. Secondly, the relationship between each dimension of attributions and EE was explored. In addition, EE is generally assessed by interview with the family members of the patient or by self-report of the patient. This study examined self-report responses from the family members of the patient. EE research has largely ignored the adolescent population, while the current study focused on a sample of this population. Finally, the majority of EE research has been conducted with schizophrenic patients. This study investigated the construct in the context of depressive disorders.

Two dimensions of attributions were explored in this study. The first dimension of attributions explored participants' beliefs about the etiology of their child's disorder. Researchers have found that causal attributions may be modified through psychoeducation about mental disorders (Read & Law, 1999), contact with mental health professionals (Atkinson et al., 1991; Robinson, 1996), and so on, rather than through any specific experiential variables. The second dimension of attributions explored participants' beliefs about the controllability of depressive symptoms.

Attributions for causality and attributions regarding controllability are not necessarily related, even though previous researchers often interpreted attributions in that manner. In other words, an individual can believe that depression occurs because of bad luck, yet also believe that personal control can effect change. Therefore, controllability attributions were measured separately from causal attributions. Furthermore, differences in causal and controllability beliefs were examined in families with a depressed adolescent and in

comparison families in order to determine how associations between these factors among those in the clinical group are similar or different from a comparison sample. Previous studies have demonstrated that, in general, family members rate many symptoms of mental disorders as uncontrollable (Hooley & Campbell, 2002). It seems likely that the severity of the behaviors of a depressed individual would lead family members to view these symptoms as less controllable, compared to similar but less severe behaviors in comparison individuals.

Therefore, the first set of hypotheses examined causal and controllability attributions. It was hypothesized that:

- causal attributions about the etiology of depression would be unrelated to controllability attributions about symptoms,
- there would be no difference between the clinical and comparison groups on causal beliefs (etiology of depression),
- controllability beliefs were expected to be higher in the comparison group.

The second set of hypotheses concerned the relationship of the dimensions of attributions and EE. Researchers have confirmed a link between attributions and EE in studies of depression (Bolton et al., 2003; Hooley & Licht, 1997). However, these studies were based on a five-dimensional model of attributions that may obscure differences between attributions for the etiology of the illness and attributions concerning current symptoms. This study expected to support the link between attributions and EE, but only for the controllability of symptoms dimension. It was hypothesized that:

- controllability attributions would be positively correlated with measures of EE for both the clinical and comparison families,

- causality attributions were not expected to be reliably related to EE measures for either the clinical or comparison families,
- EE was not expected to differ between the clinical and comparison samples.

The third set of hypotheses related to the relationship between controllability attributions and patient characteristics. Attributions appear to vary based on characteristics of the family member such as type of symptom (Brewin et al., 1991; Gretarsson & Gelfand, 1988), age (Dix et al., 1989), and illness severity (Hooley & Licht, 1997). Based on such findings, the controllability attributions participants make concerning depression were expected to be associated with patient and illness characteristics, such as depressive symptomatology, the severity of the depression, and the age of the adolescent. Specifically,

- higher age of the adolescent was expected to predict greater attributions for controllability, while greater chronicity and severity of illness were expected to predict lower attributions for controllability,
- controllability ratings for internalizing behaviors were expected to be lower than those for externalizing behaviors.

Expanding our understanding of family factors as they relate to adolescent depression can offer avenues for improving treatment models and assessment methods. Family-based treatments for depression in youths is limited and has shown contradictory evidence for the effectiveness of such methods (Asarnow, Jaycox, et al., 2001). However, in many studies comparing family therapy with other modalities, some component of family intervention is often in the competing model, thereby limiting the ability of the study to find differences (Asarnow, Jaycox, et al.). Likewise, many treatment packages that have been shown to be effective integrate some form of family treatment or education that may add to the beneficial

effects of the treatment (Coyne & Fechner-Bates, 1992). On this basis, and because family factors appear to have importance as causal or maintaining factors for depression, family assessment and treatment are an important avenue to pursue.

Accumulating information concerning factors such as EE will lead to better methods of assessment that can be utilized to make treatment decisions and better methods of treatment that focus on individuals' problems. If assessment measures suggest family dysfunction such as EE, clinicians may want to include family interventions, whereas if no negative family functioning is found, clinicians can focus on other treatment goals. The information collected in this study suggests the use of such instruments and treatment programs.

Method

Participants

Participants included individuals who accessed a survey posted on the Internet. Therefore, only those individuals who had access to a computer and an Internet connection were able to participate. Recruitment was via an invitation to participate posted to discussion forums on websites that target parents of adolescents with depression. Eighteen websites were solicited that included topics such as depression, anxiety, bipolar disorder, suicide, and mental health (see Appendix A). An introductory letter described the research and contained informed consent information before the participants clicked on a link to access the survey. Filling out the questionnaire constituted informed consent. A copy of the introductory letter is contained in Appendix B. This research and procedures for treatment of human subjects was approved by Eastern Michigan University's Human Subjects Review Committee. A copy of the approval is in Appendix C.

No requirement was made for the respondent to be the mother or for both parents to participate. This decision was made in order to minimize respondent burden and to maximize the number of responses to this study. In addition, it was expected that the parent responding to the survey may be the most involved with the adolescent due to their interest in obtaining information about parenting on the Internet.

In addition to parents of depressed teens, a comparison group was recruited in a similar manner from other websites that provide parenting information and advice. Eighteen websites were solicited for the comparison group that included topics such as general parenting advice, problem behavior, and conduct disorder (see Appendix A).

Each participant anonymously and voluntarily filled out a questionnaire regarding their attributions for the cause of their child's disorder, the amount of control that they expected their adolescent to have over the symptoms of depression, a measure of symptom severity, and a measure of expressed emotion. Branching rules in the questionnaire screened for the presence of an adolescent in the family with depression and omitted items relating to the adolescent's depression if the case was to be included as a comparison case.

Participants included 154 parents of adolescents. One case was removed because the teen was reported as 20 years of age, and two cases were removed because the respondent's age was reported in the teens (apparently the adolescents had responded themselves). Participants were predominantly female. Among the teens, each gender was relatively well-represented. Mean age of respondents was 44.7, and mean age of the adolescents was 16.0. In terms of ethnicity, the majority of respondents identified themselves as White/non-Hispanic.

Of the entire sample, 101 were identified as the clinical group (having a teen with depression) and 53 were identified as the comparison group. The classification was based on

the parent's response to a question of whether the teen had ever been diagnosed with clinical depression, hospitalized, or placed on antidepressants for depression by a professional and was confirmed by scores on the Children's Depression Inventory – Parent Version (Kovacs, 2002).

Demographic statistics for both the clinical and comparison subgroups were similar to those of the group as a whole: predominantly female and White/non-Hispanic, with a mean age in the forties and adolescents in the mid-teens. Adolescent gender was split relatively evenly in both groups, with somewhat more females in the clinical group and somewhat more males in the comparison group. By parent report, adolescents in the clinical group had a considerable number of comorbid conditions. Although adolescents in the comparison group did not have diagnosed depression, parents noted a considerable number of other mental illnesses. Approximately 40% of the parents reported that their teen had been diagnosed with some other disorder besides depression. Demographic statistics for all groups are reported in Table 1. It should be noted that the clinical and comparison groups did not differ with respect to age or sex of teen or respondent, with the exception of teen's age, $t(140) = 2.14, p < .05$. The depressed teens tended to be a little older. The effect size equals .39, which is considered small.

Table 1

Demographic Statistics

	Whole sample (<i>n</i> = 154)	Clinical (<i>n</i> = 101)	Comparison (<i>n</i> = 53)
Respondent gender ^a			
Male	5.8%	6.9%	3.8%
Female	92.2%	90.1%	96.2%
Teen gender ^b			
Male	44.2%	38.6%	54.7%
Female	55.2%	60.4%	45.3%
Respondent mean age ^c	44.7 (36)	45.3 (34)	43.7 (24)
Teen mean age ^d	16.0 (7)	16.2 (7)	15.5 (5)
Ethnicity ^e			
White/non-Hispanic	90.9%	90.1%	92.5%
Black	2.6%	3.0%	1.9%
Latino/Hispanic	1.9%	3.0%	0.0%
Asian	3.2%	3.0%	3.8%
Other	0.6%	1.0%	1.9%
Other disorders ^f	59.1%	69.3%	39.6%
Anxiety disorders	30.5%	40.6%	11.3%
ADHD/ADD	29.2%	31.6%	26.4%
CD/ODD	14.9%	14.9%	15.1%
Other	14.3%	12.9%	15.2%

Note. Numbers in parentheses denote range.

^aTwo missing data. ^bOne missing data. ^cTwo missing data. ^dTwelve missing data. ^eTwo missing data. ^fThe parent reported that their child had any other disorder besides depression. Parents could select more than one other disorder. Therefore, percentages under specific types of disorders will not add up to the total percentage for the group.

Hypothesizing a moderate effect size on dependent measures and conducting analyses with alpha set to 0.05, an 80% chance of detecting a significant effect could be achieved with a sample size of 85 for each set of data that was analyzed using bivariate correlation (Cohen,

1992). This also fulfilled the power requirements for regression analyses with the clinical group. Although the n for the comparison sample was not quite this large, the number was much larger than necessary for finding a large effect size for correlations run with this subsample. The number in the comparison subsample approached the recommended 64 necessary for testing for differences between the two groups with a moderate effect size and was more than adequate to detect differences with a large effect size.

Measures and Procedure

A self-report questionnaire was developed to gather specific attributions related to depression, (see Appendix D for sample questionnaire). A modified version was provided for the comparison sample by means of a branching format within the questionnaire. Unlike many of the previous studies of attributions, this study asked participants directly about their beliefs rather than inferring from an interview. Existing measures were also included to gather information concerning expressed emotion and for assessment of depressive symptomatology. Questionnaires were posted on a website that participants accessed through the Internet. Responses were returned via Internet to the survey web host and compiled before being provided to the researcher.

Participants were asked to indicate their age, sex, and ethnicity as well as the age and sex of their teen. In order to limit the study to attributions about clinical depression, the survey screened for adolescents who have been diagnosed with clinical depression, hospitalized, or placed on antidepressants for depression by a professional. Other items assessed for comorbidity, severity, and length of time the adolescent suffered from depression. After the initial screening question, the comparison sample was directed through

the computer program via branching rules to the attribution and expressed emotion sections, skipping the depression items.

Depressive symptomatology. Depressive symptomatology was assessed among the clinical sample with the items from the Children's Depression Inventory – Parent Version (CDI:P; Kovacs, 2002). This is a 17-item self-report measure designed to be administered to parents to evaluate the presence and severity of depressive symptoms among 7-17 year olds (see Appendix E). Norms are available for the age group from 13 to 17 years. The scale contains items such as the child crying or looking tearful, appearing lonely, looking tired or fatigued, not liking himself or herself, being cranky or irritable, having trouble sleeping at night, not enjoying being with people, being uncooperative, having to push himself or herself to do schoolwork, and not spending time with friends. Higher scores on the CDI:P indicate greater levels of depression. The scale has demonstrated excellent internal consistency ($\alpha = .87$) and discriminant validity (Kovacs).

Controllability. Next, respondents were asked to rate the amount of controllability they believed the adolescent had over 20 behaviors that have been related to depression in previous research. The items were chosen by the researcher based on descriptors from the major depressive episode section of the DSM-IV-TR (American Psychiatric Association, 2000), as well as on additional items considered to be associated features among depressed adolescents, such as academic performance (Kazdin & Marciano, 1998). The items were rated on a 5-point scale anchored by “no control = 1” and “complete control = 5.” In addition, items could be excluded by answering “not observed/don't know.” A scale was constructed by computing the mean of the controllability items.

Causality. Next, the participants were asked to rate the degree to which they agreed with seven statements concerning the etiology of depression (e.g., Clinical depression is due to genetic/biological factors, chance/bad luck, family of origin conflict, etc.) using a 5-point scale anchored by “1 = strongly agree” and “5 = strongly disagree.” These items were also chosen by the researcher based on a review of the literature concerning depression attributions.

Expressed Emotion. The Level of Expressed Emotion Scale (LEE; Cole & Kazarian, 1988) was administered to assess the level of EE perceived by the respondent (see Appendix F). The LEE was designed to be presented to individuals with a mental disorder and has primarily been used in adult populations. The LEE is a 60-item self-report questionnaire that explores four dimensions: intrusiveness, emotional response, attitude toward illness, and tolerance/expectations. The LEE has demonstrated strong internal reliability with α above .92 for adult and adolescent, psychiatric and nonpsychiatric samples (Cole, 1992). The original scale used a true/false format. For the purposes of this study, a four-point Likert-type scale was utilized instead, anchored by “1 = untrue” and by “4 = true.” Half the items were reverse scored as described in the appendix. Reliabilities above .90 were also obtained with a revised version of the LEE that used this Likert format (Gerlsma, Van der Lubbe, & Van Nieuwenhuizen, 1992). Predictive validity and convergent validity with the Camberwell Family Interview (CFI) has been demonstrated (Donat, 1996; Gerlsma & Hale, 1997; Kazarian et al., 1990). In addition, results of relatives’ responses on the LEE correlated with measures of EE as measured by the CFI (Kazarian et al.).

Results

Preliminary Analyses

Depression. Data preparation included scoring the section of the questionnaire consisting of CDI:P items separately to create a variable for severity of depression. The 17 items in this section were added together to create a summed score ($M = 42.39$, $SD = 6.00$). A minimum of 15 items were used to compute this score. Missing items were not prorated in order to avoid artificially inflating scores if a particular symptom was not observed in an individual. This resulted in 6 clinical cases missing data for this scale. An a priori cut-off value of 19 was chosen based on recommendations from the CDI manual. Cases with scores at or above this point were considered to have a high level of symptomatology consistent with clinical depression. All of the scores were above this cutoff with most being at least twice this much, negating the need for further analyses separating currently symptomatic cases from those that were asymptomatic. The CDI:P scores were used in analyses as a measure of severity of symptomatology.

Causal attributions. The mean ratings for the clinical and comparison group were computed on the seven causal attributions (biogenetic, chance, family conflict, other conflict, higher power/evil, environment, intrapersonal). The results are listed in Table 2. Not surprisingly, the participants agreed most strongly that biogenetics and environmental difficulties (stress, illness, etc.) are the causes of depression. This was followed by intrapersonal reasons (personality, poor choices, insufficient effort, etc.), family conflict, and conflict with others. The lowest rated explanations for both groups were chance and higher power/evil.

Table 2

Mean Ratings for Causal Attributions by Group

	Clinical <i>M (SD)</i>	Comparison <i>M (SD)</i>
Biogenetic	4.25 (0.98)	4.20 (1.00)
Chance	2.29 (1.11)	2.04 (1.11)
Family conflict	3.68 (0.89)	3.96 (0.78)
Conflict w/ others	3.59 (1.04)	3.84 (0.87)
Higher power/evil	1.93 (1.22)	2.16 (1.22)
Environment	4.21 (0.67)	4.24 (0.59)
Intrapersonal	3.92 (1.03)	3.96 (0.86)

Note. Ratings were made on 5-point scales (1 = strongly disagree, 5 = strongly agree).

Controllability. A principal components factor analysis was conducted on the 20 items in the controllability attributions section. A two-factor solution was rotated using a Varimax rotation procedure because of the a priori assumption that the subscales, although correlated, would have some independence and represent distinct constructs. Incidentally, both orthogonal and oblique rotations were performed, which had essentially identical results. Only items loading above .4 were included in the solutions. The factor loadings are presented in Appendix G.

The analysis did not support the formation of two factors consisting of internalizing and externalizing behaviors. In fact, most items loaded heavily on one factor that seemed to measure beliefs concerning major depressive symptoms. The second factor consisted of three items related to school and an item referring to frequent crying. Due to the low number of items on the component with academic variables, this factor was not considered further. A composite for controllability was created by computing the mean of the 16 remaining items ($M = 3.47$, $SD = .81$). If a participant endorsed some items as “not observed, don’t know,”

those items were not used to compute the mean. A minimum of ten items was required for this composite. This resulted in 25 participants with missing data. A reliability analysis revealed an alpha of .95 for the controllability scale.

Expressed emotion. Similarly, the 60 items in the LEE section of the questionnaire were averaged to create a measure of expressed emotion. A minimum of 30 items was used to compute this score, which resulted in four cases missing data for this scale. An alpha of .93 was obtained with a reliability analysis. A principal components factor analysis was also conducted for the LEE to determine if any underlying factor structures emerged that should be included in the analyses. Two-, three-, and four-factor solutions were rotated using a Varimax rotation procedure. Only items loading above .4 were included in the solutions. Again, oblique rotations were performed due to the high intercorrelations between items but produced results similar to the orthogonal rotations.

The four-factor solution yielded too few items on several factors and was difficult to interpret, so it was not considered further. The three-factor solution appeared to roughly approximate three of the four *a priori* subscales described by one of the scale's authors (Cole, 1992). The *a priori* subscale attitude toward illness was not represented in the factor analysis in this current study. The remaining three factors represent tolerance/expectations, intrusiveness, and emotional responsivity. A two-factor solution was attempted because of the small number of items loading on the emotional responsivity factor. However, the two-factor solution retained much the same structure as the three-factor solution, with the emotional responsivity factor simply dropping off the solution.

Subscales were created to reflect each of these factors: tolerance/expectations consisted of 37 items and had an alpha of .94; intrusiveness had 10 items with an alpha of

.77; and emotional responsivity had 5 items with an alpha of .70. One *a priori* subscale, attitudes toward illness, did not emerge in the factor analyses of the expressed emotion items. Most of the items from the attitudes toward illness subscale loaded on the tolerance/expectations factor in the analyses for this study. In all of the analyses, many more items loaded on the tolerance/expectations factor than on the other factors, including items from the other *a priori* subscales, suggesting that tolerance/expectations may be the main construct that the Level of Expressed Emotion Scale is measuring. The three-factor solution is presented in Appendix H.

After preliminary analyses were conducted, relationships between causal and global controllability attributions and measures of EE were analyzed with bivariate correlations. T-tests were conducted to analyze differences between clinical and comparison families' beliefs concerning causality and controllability of symptoms and levels of EE. In order to support hypotheses that predicted no difference between EE and causal beliefs in the clinical and comparison samples, equivalency analyses were used to provide evidence that the groups were equivalent. Finally, multiple regression was utilized to identify individual characteristics, such as age, chronicity, and severity, that predict controllability attributions.

Analysis of Differences between Groups

As predicted, independent samples t tests for differences in causal attributions between the clinical and comparison groups resulted in no significant difference between the groups. In addition, no significant difference was found between the groups on controllability beliefs. The clinical and comparison families also exhibited no differences in patterns of behavior regarding expressed emotion within the families. T tests for differences in ratings between the two groups had nonsignificant results at the .05 level.

Previous researchers have noted that failing to reject the null hypothesis in standard difference testing does not prove equivalence (Cribbie, Gruman, & Arpin-Cribbie, 2004; Epstein, Klinkenberg, Wiley, & McKinley, 2001; Rogers, Howard, & Vessey, 1993; Stegner, Bostrom, & Greenfield, 1996). Therefore, equivalency analyses were used to provide evidence that the groups in this study were equivalent on these attribution ratings using a procedure described by Rogers et al. (1993). This involved conducting two one-sided t-tests on the comparison group using an a priori equivalence criterion equal to $\pm 20\%$ of the clinical group mean. Although no standard for equivalency criteria exists yet, Stegner et al. suggested a 20% criterion that evolved in the pharmaceutical industry from which these methods were derived. Thus, any difference between group means that falls within this equivalence interval is considered too small to be clinically or practically important (Rogers et al.). In addition, a confidence interval around the difference between means was constructed, which allows for identifying the degree of equivalence (Stegner et al.). The results for traditional and equivalence testing are shown in Table 3. Although difference tests suggested that parental beliefs across groups are not different, equivalency analyses showed that attributions for chance and higher power were not statistically equivalent.

Table 3

Mean Differences in Causal and Controllability Attributions by Group

	<u>Clinical</u>		<u>Comparison</u>		<u>Difference</u>		<u>Traditional</u>		<u>Equivalence</u>			<u>90% CI</u>	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SE</i>	<i>t</i>	<i>p</i>	EC	<i>t</i>	<i>p</i>	LCL	UCL
B	4.25	0.98	4.20	1.00	.04	.17	0.25	.80	±.85	4.55	.001*	-.24	.33
C	2.29	1.11	2.04	1.11	.25	.19	1.27	.21	±.46	-1.26	.11	-.07	.57
FC	3.68	0.88	3.96	0.78	-.28	.15	-1.86	.06	±.74	2.63	.005*	-.53	-.03
CO	3.59	1.04	3.84	0.87	.25	.17	-1.43	.15	±.72	2.39	.009*	-.53	.04
HP	1.93	1.22	2.16	1.22	-.23	.21	-1.10	.28	±.39	.64	.26	-.58	.12
E	4.21	0.67	4.24	0.59	-.03	.11	-0.25	.80	±.84	6.31	.001*	-.21	.16
IP	3.92	1.03	3.96	0.86	-.04	.17	-0.24	.81	±.78	3.82	.001*	-.32	.24
Cont	2.64	0.76	2.60	0.80	.04	.14	0.29	.78	±.53	3.41	.001*	-.20	.29
EE	1.99	0.37	1.91	0.38	.20	.08	1.30	.20	±.40	-5.16	.001*	-.02	.19

Note. CI, confidence interval; EC, equivalence criterion set at 20% of the clinical mean; LCL, lower confidence limit; UCL, upper confidence limit. For the equivalence analyses, the greater of the two *p* values is reported for each variable. B = biogenetic; C = chance; FC = family conflict; CO = conflict others; HP = higher power; E = environment; IP = intrapersonal; Cont = controllability; EE = expressed emotion.

**p* < .05 for equivalency, per each one-tailed test.

The confidence interval approach to equivalence testing can be used to provide a visual representation of similarities and differences between group means by converting the upper and lower confidence limits to a percentage of the clinical group mean. Figure 1 graphically represents the results of traditional difference testing and the confidence interval analysis for equivalence testing. If the 90% CI falls within the equivalence interval ($\pm 20\%$), one may conclude equivalence. Further, if the 95% CI excludes zero, the traditional hypothesis test of no difference may be rejected. Therefore, one can see that the two groups have no significant differences for each of the variables, but equivalence testing reveals that chance and higher power are also not equivalent. As mentioned above, the CIs expressed as a

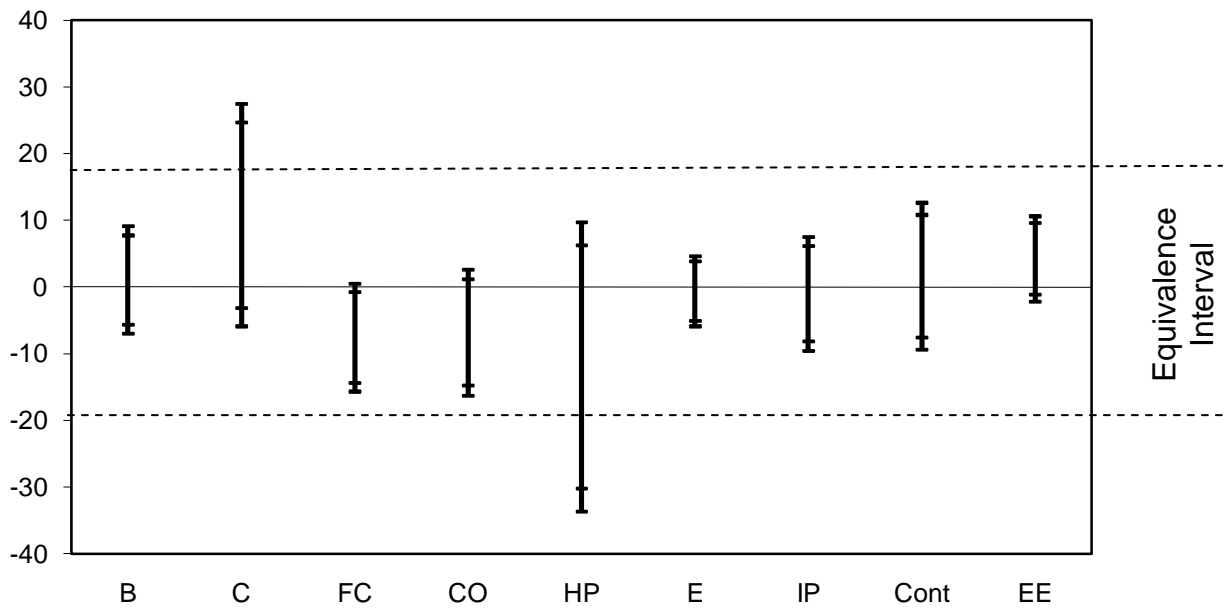


Figure 1. The 90% and 95% confidence intervals around the difference between group means expressed as a percentage of clinical group means. Inner tick marks represent 90% CI (equivalence test); outer tick marks represent 95% CI (difference test). B = biogenetic; C = chance; FC = family conflict; CO = conflict others; HP = higher power; E = environment; IP = intrapersonal; Cont = controllability; EE = expressed emotion.

percentage provide an estimate of the degree of equivalence. For example, this analysis demonstrates equivalence for the EE ratings within 9.6% of the mean implied by the clinical group, while equivalence for family conflict attributions is demonstrated within 14.4% of the mean for the clinical group.

T-tests were also conducted to test for differences between the two groups on individual controllability items to identify if any symptoms are more salient to the parents of depressed adolescents. Interestingly, the items that showed significant differences between groups were the exact items that were excluded by factor analysis, specifically the items reflecting academic concerns and frequent crying. The results are shown in Appendix I. In order to confirm that these symptoms were not reflective of the controllability construct that this study intended to measure, correlations were run for a scale created from these four items, and the EE ratings. The results were nonsignificant for both the clinical and comparison samples, as well as for the sample as a whole, $p > .05$.

Due to the large number of parents who reported psychiatric diagnoses in the comparison group (almost 40%), further analyses were conducted to test for differences between those reporting depression and a nonclinical group (parents who reported no psychiatric diagnosis). Independent samples t tests for differences in controllability attributions between the clinical and nonclinical groups resulted in no significant difference between the groups. In addition, no significant difference was found between the groups on ratings of expressed emotion within the families. T tests for differences in ratings between the two groups had nonsignificant results at the .05 level.

Equivalency analyses were then conducted to fulfill the requirement for evidence of equivalence. The one-sided t-tests on the nonclinical group using an a priori equivalence

criterion equal to $\pm 20\%$ of the clinical group mean demonstrated equivalence for ratings of EE, $t(59) = 3.88$, $p < .001$, but not for ratings of controllability beliefs, $p = .09$. Parents whose teens had no diagnosed psychiatric disorder had higher controllability beliefs than parents whose teens had been diagnosed with depression.

Relationship between Causal and Controllability Attributions

As predicted, bivariate correlations indicated that the relationship between causal attributions and controllability attributions is inconsistent. Analyzing the entire sample, correlations for causal ratings and global controllability attributions found only two of the seven causal attributions were related to controllability attributions. The belief that chance was responsible for depression and the belief that intrapersonal problems were responsible for depression were each related to how much control participants believed individuals had over all the symptoms of depression. Subjects who rated these beliefs higher as a cause of depression were more likely to believe that individuals have control over their symptoms. Correlations for all causal beliefs and global controllability attributions are listed in Table 4.

Table 4

Correlations between Causal and Controllability Attributions for the Whole Sample

	Biogenetic	Chance	Family of origin conflict	Conflict with others	Higher power/evil	Environment	Intrapersonal
Controllability	-.16	.19*	.07	-.01	.11	-.04	.21*
<i>n</i>	(126)	(128)	(124)	(126)	(127)	(129)	(129)

Note. Participants were allowed to answer “not observed/don’t know” for the controllability attributions and these were excluded from analyses.

* $p < .05$.

When only those in the clinical sample were included in the analyses, similar results were found. Chance and intrapersonal etiologies were associated with global controllability ratings. There were no significant correlations when analyzing the comparison sample.

Correlations for causal and global controllability ratings when only the clinical sample was analyzed are listed in Table 5.

Table 5

Correlations between Causal and Controllability Attributions for the Clinical Sample

	Biogenetic	Chance	Family of origin conflict	Conflict with others	Higher power/evil	Environment	Intrapersonal
Controllability	-.10	.25*	.11	-.02	.05	-.10	.30**
<i>n</i>	(87)	(88)	(87)	(86)	(87)	(89)	(89)

Note. Participants were allowed to answer “not observed/don’t know” for the controllability attributions and these were excluded from analyses.

* $p < .05$. ** $p < .01$.

Attributions and EE

Causal beliefs. Various causality attributions were related to EE measures in the clinical or comparison families, with fewer correlations among the comparison families. In the clinical sample, parents who more strongly believed that biogenetic or environmental factors caused depression were less likely to report expressed emotion, including hostility and overinvolvement. A belief in chance, family conflict, or higher power as a cause for depression was also related to reported levels of EE. Parents who endorsed such beliefs were more likely to report higher levels of EE. Among the parents in the comparison group, only a belief in intrapersonal causation was related to levels of EE. Those who were more likely to believe that the adolescent was personally responsible for causing the depression were also more likely to report high levels of expressed emotion. Results are reported in Table 6.

Table 6

Correlations between Causal Attributions and EE for the Clinical and Comparison Groups

	Clinical	Comparison
Biogenetic	-.27**	-.05
Chance	.30**	.03
Family conflict	.22*	.03
Conflict w/ others	.16	-.10
Higher power/evil	.36**	-.17
Environment	-.26**	-.01
Intrapersonal	.04	.34*

Note. N for each set of variables in the clinical group ranged from 94-97 and ranged from 47-50 in the comparison group.

* $p < .05$. ** $p < .01$.

In order to compare results from this study with earlier EE work that suggests that expressed emotion consists of several factors, correlations were also calculated between attributions and the EE factors obtained through the data reduction methods described above. Results from these analyses were consistent with earlier findings. Controllability attributions were significantly correlated with the tolerance/expectations factor, $r(129) = 0.27, p < .01$, but not with intrusiveness or emotional responsivity. Thus, the more control parents believed their child or any child has over various symptoms of depression, the more likely they are to report higher expectations and intolerance for adolescent behaviors. When the correlations were run separately for the clinical and comparison samples, the relationship between tolerance/expectations and controllability was significant only for the clinical group, $r(89) = 0.28, p < .01$.

For causal attributions, the clinical group had correlations clustered most heavily on the tolerance/expectations factor. Belief in biogenetic causation is related to tolerance/expectations and intrusiveness but in opposite directions. Parents with biogenetic beliefs reported lower expectations and intolerance but more intrusiveness. Beliefs in chance, a higher power, or environmental causation were only related to reported tolerance/expectations. Parents who endorsed belief in environmental factors as a cause for depression were less likely to report high expectations or intolerance, while parents who believed that depression is caused by chance or a higher power were more likely to report high expectations and intolerance. Finally, parents who believed that conflict with family members or others caused depression were more likely to report higher levels of intrusiveness. Results are shown in Table 7.

Table 7

Correlations between Causal Attributions and EE Subscales for the Clinical Group

	Tolerance/Expectations	Intrusiveness	Emotional Responsivity
Biogenetic	-.38**	.22*	-.03
Chance	.33*	.08	.03
Family conflict	.19	.26*	-.03
Conflict w/ others	.13	.22*	.00
Higher power/evil	.35**	.17	-.03
Environment	-.35**	.18	-.04
Intrapersonal	.01	.18	-.17

Note. N for each set of variables ranged from 94-97.

* $p < .05$. ** $p < .01$.

Controllability. As predicted, bivariate correlations indicated that there is a positive relationship between controllability attributions and EE. Analyzing the entire sample, the

correlation for controllability attributions suggested that as the amount of control a parent expects their teen to have over depressive symptoms increases, the amount of expressed emotion within the family increases correspondingly, $r(129) = 0.27, p < .01$. This relationship also existed when analyzing only the clinical group, $r(89) = 0.28, p < .01$, but not with only the comparison group, $r(40) = 0.25, p > .05$. Correlations for the individual controllability items are reported in Appendix J.

In order to determine the relative contribution of parental attributions to prediction of levels of EE in the home, a regression analysis was performed. A stepwise procedure was employed so that only those variables that contributed incrementally beyond variables already in the model would be retained. The dependent variable was EE and the independent variables were controllability beliefs, biogenetic, chance, family conflict, other conflict, environment, higher power, and intrapersonal causal beliefs. Higher power and environmental causal beliefs and controllability beliefs were the only significant predictors of EE (see Table 8). The remaining variables did not add significantly to the model.

Table 8

Stepwise Regression Analysis for Variables Predicting EE (N = 87)

Variable	<i>B</i>	<i>SE B</i>	β
Step 1			
Higher power	.11	.03	.37**
Step 2			
Higher power	.10	.03	.34**
Environment	-.13	.05	-.24*
Step 3			
Higher power	.10	.03	.33**
Environment	-.12	.05	-.23*
Control	.10	.05	.21*

Note. $R^2 = .14$ for Step 1; $\Delta R^2 = .16$ for Step 2, $\Delta R^2 = .04$ for Step 3, ($ps < .05$).

* $p < .05$. ** $p < .01$.

Controllability Attributions and Individual Characteristics

The data were entered in a multiple regression analysis where the dependent variable was global controllability and the independent variables were age of adolescent, chronicity, and severity of illness. Severity and chronicity were significant predictors, indicating that each contributed unique information to the prediction of controllability attributions. Severity was a positive predictor, while chronicity was a negative predictor. In other words, as severity of symptoms increased, reported controllability beliefs also increased. Conversely, as the chronicity of the depression went up, the report of controllability beliefs decreased. Severity predicted 10% of the variance in controllability attributions, and chronicity predicted 7% of the variance, while age of adolescent did not add significantly to the model. The regression statistics appear in Table 9.

Table 9

Stepwise Regression Analysis for Variables Predicting Controllability Attributions (N = 88)

Variable	<i>B</i>	<i>SE B</i>	β
Step 1			
Severity	.04	.01	.32**
Step 2			
Severity	.04	.01	.30**
Chronicity	-.17	.07	-.26*

Note. $R^2 = .10$ for Step 1; $\Delta R^2 = .07$ for Step 2, ($ps < .05$).

* $p < .05$. ** $p < .01$.

The relationship between severity and chronicity were explored to identify factors that led to the opposite relationship of these variables to controllability beliefs. A univariate analysis of variance suggested that there were significant differences in the severity of depressive symptoms that parents reported based on the length of time that the child had had depression, $F(5,89) = 2.67$, $p < .05$. Although Tukey's post hoc testing showed only one significant difference between chronicity groups (5-10 years and over 10 years), an examination of the means reveals that there is a clear trend toward decreasing severity as chronicity increases until the depression has lasted for more than 10 years (see Figure 2).

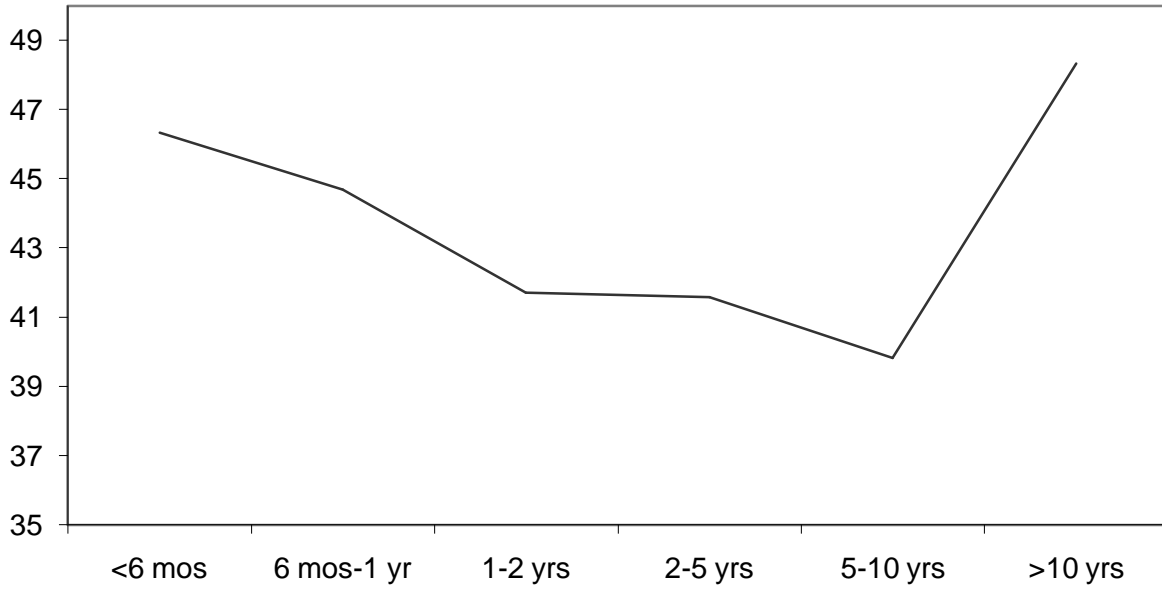


Figure 2. Mean ratings for severity of depressive symptoms by chronicity of depression.

Discussion

The main goal of this study was to explore the relationship between expressed emotion and various attributions, specifically to determine if causal attributions or attributions concerning controllability of symptoms are useful in the formation of theories of expressed emotion. Previous research has reported that levels of EE within the family predict non-recovery or relapse of mental illness. Some researchers have speculated that the beliefs family members hold concerning the illness are related to the amount of EE within the family setting. However, studies searching for such links have proved inconclusive. This study explored attributions concerning the illness from two dimensions: beliefs about the etiology of the disorder and beliefs about whether the ill family member has control over symptoms of the disorder. Most of the research in the area of expressed emotion has been conducted with an adult schizophrenic population using family interviews. Since this study used a depressed adolescent population and parent-report measures, a non-depressed sample was also recruited in order to contrast with comparison families. The findings largely support the hypotheses put forth in this study. Specifically, this research found that controllability attributions are related to measures of expressed emotion, while beliefs about causality had mixed results. It is suggested by this study that constructs regarding controllability and causal beliefs may be less related than previous researchers suspected, and controllability beliefs may be more salient to theories of EE.

Relationship between Causal and Controllability Attributions

The relationship between causal and controllability attributions may have significant consequences for the understanding of expressed emotion, attributions, and depressive outcomes. These attributions represent a basic difference between past and present

responsibility. Beliefs concerning past and present responsibility have typically been coded into the same categories (Barrowclough et al., 1994; Hooley & Licht, 1997; Licht, 2001). Adding to the confusion, it appears that some investigators have had difficulty defining attributions as internal or external (Bolton et al., 2003; Hooley & Licht).

The results of this current study suggest that beliefs concerning etiology are not consistently related to beliefs about the controllability of symptoms. In other words, some parents may believe that a child's depression is caused by genetics yet still expect the child to control symptoms of the illness. In fact, five of the seven etiologies did not show significant correlations with controllability attributions. Only two causes (chance and intrapersonal problems) were related to parents' beliefs concerning controllability of depressive symptoms. These relationships were found only in the clinical sample. No correlations between causal and controllability beliefs were found in the comparison sample.

Brickman et al.'s attributional model (1982) provides a framework from which to explore the various possible combinations of attributions. Considering the causal attributions (intrapersonal problems and chance) correlated with controllability in this study, it appears that the moral and compensatory models describe the relationships of the attributions in question. It would be expected that an individual would be held responsible for intrapersonal factors that caused a past event (causal attribution) and for a solution to current events (controllability attribution). This is the theme of the moral model. The compensatory model best fits a situation in which relatives believe in chance causation of the depression but also have current controllability attributions. The relatives do not necessarily hold the individual responsible for causing the depression (chance factors), yet the individual is expected to contribute to recovery (by controlling the symptoms). Given that such a compensatory model

is the most common belief system regarding mental illness (Kleinke & Kane, 1997), we would expect other causal beliefs that do not ascribe responsibility to the individual (biology, higher power) to be linked with controllability attributions. However, in this study, these associations were not found, suggesting that, although causal and controllability beliefs may influence each other, they are not inextricably linked. In addition, as will be discussed later, beliefs concerning chance and intrapersonal problems did not add predictive value to a regression model for expressed emotion among the clinical sample, although several other causal beliefs did add to the regression model. Regardless, the majority of the causal attributions were not found to be related to controllability beliefs.

Attributions and EE

The behaviors that family members exhibit toward a particular individual within the family system are measured as expressed emotion, while the attributions that family members hold are the beliefs about the individual's illness and behaviors. The attributions might be considered underlying cognitive belief systems that cause or affect EE. A review of the literature regarding attributions and expressed emotion confirmed that research has been consistent in supporting the idea that critical relatives are more likely to hold their ill relative responsible for their difficulties (Barrowclough & Hooley, 2003). Longitudinal studies would need to be conducted to test for causal effects. Some researchers have begun to find promising results in this area. A study of 26 relatives of patients with schizophrenia compared EE and attribution ratings and found that there were measurable changes in EE and attributions after an intervention aimed at reducing EE (Brewin, 1994). It appears that the LEE scale is conducive to future projects of this nature due to its ease of administration. The results of this current study add support for the validity of this measure.

This current study corroborated the finding of an association between controllability attributions and EE. This indicates that family members' beliefs concerning how well their teens can control depressive symptoms affects the level of expressed emotion within the family, although causation cannot be assumed. Controllability attributions were significantly related to EE, but not for the comparison group. Not surprisingly, the more parents believe their children have control over depressive symptoms, the more likely they are to be overly critical and hostile. However, it is interesting that these correlations were significant for the clinical sample but not for the comparison group. Apparently, the experience of the depressed children affects parents' beliefs regarding whether their children have control over their behaviors. This hypothesis is supported by the fact that severity of symptoms and chronicity predicted controllability in the regression analysis.

As noted above, there were few relationships between controllability and causal beliefs; however, some causal attributions were directly related to EE. For causal attributions, many of the clinical group's ratings were correlated with EE, with the exception of conflict with others and intrapersonal etiologies. Biological and environmental causation were negatively related to EE, while chance, family conflict, and higher power were positively related. Among the comparison group parents, only the intrapersonal variable was related to EE. The lack of correlation between causal beliefs and EE among comparison families is not surprising, considering that the families did not have a depressed teen and the questionnaire asked them to rate their beliefs concerning depression.

It appears that belief in biological or environmental causation may provide a protective factor against the development of critical behaviors within the family system. Presumably, parents with these types of etiological beliefs found fewer faults with their

children because the causal explanation for the behaviors in question relieved the children of parental expectations by taking the responsibility out of the hands of the child.

Conversely, chance, family conflict, and higher power explanations for depressive behaviors, which at the surface appear to relieve the children of responsibility, apparently did not alleviate parental behavioral expectations for their teens. In the case of chance and higher power beliefs, this may reflect the ethereal and intangible nature of these constructs. Parents are critical of their children due to their own inability to fix an unambiguous cause to the event. Or the critical behavior may be a result of some moral judgment that ascribes responsibility for seemingly chance events by judging them to be a sort of punishment for the individual.

One surprising finding is that intrapersonal beliefs were not related to expressed emotion in the clinical sample. While there was a relationship in the comparison sample, it was not true among the clinical families. Of all the possible etiological beliefs, it seems that this would engender the most criticism from parents. Belief that an adolescent had depression due to poor choices, manner of thinking, insufficient effort, or other things specific to the teen would be expected to result in criticism from the parents who expect the teen to change the personal factors involved. However, it may be that this category encompasses individual factors that parents do not believe the child can change. If this were the case, the results in this study would not be so surprising.

It appears that we do not have a complete understanding of what meaning these causal beliefs have for parents. We often infer blame from statements regarding these beliefs. Yet we do not know, for example, if parents feel that they are personally responsible for a genetic cause, whether they hold the individual responsible, or whether they feel there can be

no personal responsibility for such an uncontrollable event. It may be worth exploring what parents think regarding blame when they endorse specific causal beliefs.

Due to the discrepancies in expected correlations, these results need to be replicated to determine if they are simply an artifact of this study. The variance in results among causal explanations also creates some difficulty with using causal beliefs in theories of EE.

Further analyses were conducted to reveal the relative relationships between parental beliefs and EE using stepwise regression. Among causal attributions, belief that a higher power/evil caused the depression and belief in environmental causation each added to the predictive power of the equation, but in opposite directions. Controllability beliefs also added significantly to the model. The remaining etiological beliefs were excluded from the model. There may be several mediating or moderating relationships between these variables that make it difficult to understand from a correlational perspective. Path analysis models may help to untangle the associations among these constructs.

The results of the factor analysis for the LEE, although supportive of the *a priori* constructs from which the subscales were created, were quite different in makeup from the subscales reported by the authors of the LEE. This may be an artifact of the sample and methodology used in the current study. The sample for this study consisted primarily of parents of adolescents with depression, while Cole's work (1992) used adult and adolescent patients with schizophrenia. In addition, the relative version of the LEE was used instead of the patient version. Considering these major differences, it is encouraging that the constructs for the *a priori* subscales do hold together in the current study, even though the items that loaded onto the subscales may have varied. These results add support for the validity of this

easy-to-administer instrument that shows promise for use in research as well as clinical settings.

Analyses examining attribution-EE links through subscales of the LEE were not surprising. Only the tolerance/expectations subscale correlated with controllability attributions and only among the clinical group. Causal attributions showed similar patterns; correlations were clustered on the tolerance/expectations scale and much more heavily for the clinical group. Belief in biogenetic or environmental causation appears to decrease parental criticism within this sample, while belief in chance or a higher power as causal agents increased parental criticism.

The finding that tolerance/expectations was the most strongly correlated subscale suggests that future research should focus on the tolerance/expectations construct of EE. It appears from an examination of individual items that this subscale measures critical attitudes and behavior within the family system. This is the construct of EE most consistently linked in the literature to attributions and to relapse or remission of illness. The results of this current study provide further support. The fact that intrusiveness did not correlate with controllability is not surprising considering that other researchers have speculated that intrusiveness (or emotional over-involvement) measures a parenting construct that may be developmentally appropriate for this age group. In other words, parents are expected to know where their children are and what they are doing.

This raises an interesting question of whether we should continue to use the term “expressed emotion.” First, the term is misleading, implying that there is too much emotion within the household. Secondly, the research literature has consistently shown that the most salient aspect of EE is criticism. In a validity study for EE, Hooley and Richters (1991)

argued that searching for better measures of EE through correspondence with existing measures may be unproductive. Instead researchers should focus on identifying predictors of relapse in order to improve the measures. Similarly, we should be willing to discard portions of existing theories that are no longer useful in favor of more profitable avenues. It may be time to relabel EE as criticism in order to be clear concerning what is being measured.

Differences between Groups

No differences between groups were predicted for causal attributions because these attitudes seem to be shaped by culture and exposure to whatever beliefs are current among associates, medical personnel, and so on. Indeed, there were no significant differences between groups. Equivalency analyses supported these findings with the exception of causal attributions concerning chance and a higher power. Analyses for these two variables did not demonstrate statistical equivalence. Therefore, although clinical and comparison families' causal beliefs concerning chance and a higher power are not statistically different, they also are not statistically equivalent. In general, however, clinical and comparison families held similar causal beliefs regarding adolescent depression. These results suggest that causal beliefs are most likely not a result of exposure to a family member with depression. Beliefs regarding the etiology of the disorder must be formed through other avenues, such as those hypothesized above.

Further exploration of the differences between groups contradicts previous research demonstrating that psychiatric populations have higher LEE scores than nonpsychiatric populations and supports the hypothesis of equivalence in this study. In fact, there was no difference in EE ratings between the clinical and comparison groups, and equivalence was demonstrated. One factor that may explain why the current results contradict previous

research is the modification to the scoring of the LEE in this study. The distribution for the LEE is typically positively skewed, reflecting a normative pattern for this instrument (Cole, 1992). This means that the scores would be clustered towards the lower end of the scale. However, in the current study, a 4-point scale was used rather than the true/false format with which the scale was created. Respondents were freer to report varying levels of EE rather than committing to a true/false dichotomy. This resulted in a normal curve for this sample.

Another possible explanation is the use of an adolescent population in the current study. It is reasonable to assume that EE would be higher in an adolescent population than in an adult population because of the level of control parents exert over their children, which may result in similar levels of EE between clinical and comparison families. Indeed, this difference between teen and adult levels of EE was demonstrated in previous research (Cole, 1992). In an exploration of the psychometric properties of the patient version of the LEE, it was discovered that adolescents scored significantly higher than adults on this instrument. In other words, EE among families in which the psychiatric patient was an adolescent was rated higher than EE among families in which the patient was an adult. This, coupled with the ability of parents to report EE on a scale, may explain the lack of differences between the clinical and comparison groups.

This finding suggests that EE should not be viewed as a causal factor. Apparently, EE exists in many households. In fact, it could be expected among families with an adolescent due to the developmental level of the teen and the power differential between the parents and their child. As mentioned earlier, criticism is commonly used, whether effective or not, as a means of exerting behavioral control over others. This apparently becomes especially

problematic after the development of depression when criticism affects the course of the disorder.

In terms of controllability, beliefs were expected to be higher in comparison families because the severity of symptoms among depressed teens would lead these families to view the behaviors as less controllable than behaviors in comparison families. One possible explanation for the finding of no differences between groups is that the percentage of other disorders among the comparison group was high enough that families may have been rating behaviors with similar severity.

The difficulty in securing “pure” groups is commonly reported in the literature. One argument proposes that the fact that the clinical group included comorbid conditions does not diminish the value of the research because pure cases are the exception rather than the norm. However, the comparison group also reported a number of psychiatric diagnoses, as well as comorbidities, which limits the inference that we can make from comparisons.

In fact, almost 40% of the comparison group reported some sort of mental disorder. This seems much higher than expected in a comparison group and may be due to the method of recruitment. Parents might be seeking parenting help on the internet due to problem behaviors among their children. Therefore, many of the comparison group parents may have been rating behaviors at a clinical level equivalent to the parents with depressed teens, which would create a situation in which no differences would be expected.

In fact, only when parents whose adolescents did not have a psychiatric diagnosis were compared to parents whose teens had a diagnosis of depression were the ratings for controllability beliefs not equivalent. As predicted, the nonclinical group parents expected

adolescents to have more control over depressive symptoms than parents in the clinical group.

Individual controllability items were also tested for differences between the two groups, which resulted in several items showing significant differences. The difference in parents' ratings of controllability items corresponds to the findings from the factor analysis of controllability items, with the academic items and frequent crying standing alone. Comparison families expected their teen to have more control over crying and problems in academic areas. The findings regarding the academic items standing out among the individual controllability variables in the factor analysis and in difference testing between groups lends support to the idea that academic symptoms are not salient in attribution research concerning depressed adolescents. The comparison families expected adolescents to maintain more control in this area than the clinical families, which, coupled with the results from the factor analysis, suggests that these types of symptoms may not contribute substantially to parents' controllability attributions for depressed adolescents. This, in fact, seems to be the case considering that a subscale consisting of academic items was not correlated with EE among clinical families. However, it also suggests that parents of depressed adolescents may provide more latitude in this area than parents of nondepressed teens. Parents of depressed adolescents may not have as high expectations of their children in school-related endeavors as parents without a depressed teen. It is possible that the illness serves as a justification or explanation for poor performance.

Individual Characteristics and Controllability

Finally, analyses exploring the contribution of individual characteristics to ratings of controllability found that the severity and chronicity of the illness predicted parental beliefs

regarding controllability. Contrary to hypotheses, the age of the adolescent did not add significantly to the prediction. It is possible that the age of their teen has no effect on parents' controllability beliefs. On the other hand, low numbers of 13- and 14-year-olds in this sample may have precluded abilities to find effects in this study. Severity was measured by the CDI:P, while chronicity was established from a single categorical rating of the length of time the adolescent had suffered from depression. Greater severity predicted higher ratings for controllability, while longer chronicity predicted lower ratings for controllability.

In general, the findings regarding individual characteristics were not surprising. The longer a child has had depression, the more likely it seems that parents would have accepted the idea that their child has a disorder with symptoms that are difficult if not impossible to control. In contrast, parents who reported greater severity of depressive symptoms also indicated higher controllability attributions. The decline in severity over time that was evident in this study is unlikely to be due to an actual reduction in symptoms considering what is known about the course of depression. An alternative explanation is that parents adapt to the symptoms and view them as less severe over time. This hypothesis is supported by the apparent effect of chronicity on controllability beliefs. Although every effort was made to obtain an objective report of symptoms, the CDI:P is essentially parental experience of the depression. Apparently what were measured were the parents' beliefs about severity moderated by the length of time that they had dealt with their teen's depression. In effect, this has the opposite predictive function for controllability beliefs. Parents whose teen had been diagnosed with depression for a shorter period were more likely to report high severity. This resulted in the opposite predictive effect.

Limitations and Future Directions

The limitations of this particular study include the fact that the design is cross-sectional and primarily correlational in nature. Therefore, no causal assumptions can be made based on these observations. Although the constructs are linked, we do not know in what direction each acts upon the others. Studies based on a longitudinal design will be of value in clarifying some of the relationships described herein. Similarly, interventions may be developed that aim at the specific behaviors (criticism) or at the underlying belief systems. Future research may indicate which avenue is most effective.

It is also important to note the composition of the sample. Although no effort was made to limit participants to females, this sample consisted primarily of mothers of depressed adolescents. With fewer than 6% of the sample being male, no analyses for the contribution of fathers' beliefs and expressed emotion could be conducted. Do fathers' attributions compound the affects on expressed emotion in the household? Conversely, if parents hold opposing beliefs, is there some protective factor at work? These issues may be addressed in future studies from a more systemic perspective. A similar limitation arises due to the fact that the sample was not culturally diverse. We cannot assume that families of various backgrounds will have patterns similar to those in this sample.

Furthermore, there is likely a self-selection bias based on the recruitment methods of this study. Respondents must have had access to a computer and are assumed to have had a concern and interest in gaining knowledge concerning their teens' behaviors, based on the fact that they were accessing websites designed to promote such knowledge. It is possible that those not included in our sample may differ in important characteristics and, therefore, that findings from this study may not generalize to all families.

Finally, although it appears that there is a great deal of information in this study that may be useful in the conceptualization of expressed emotion, this current study utilized an understudied population with a relatively novel measure and a fresh conceptualization of parental attributions. Therefore, this study needs to be replicated to ensure that the results are not artifacts of the present study.

Conclusions

In summary, the findings support the idea that controllability beliefs may be an important construct in understanding levels of EE within families. This study adds to the existing literature by underscoring the importance of defining constructs carefully for the measurement of attributions. When beliefs concerning etiology have been combined with controllability beliefs or assumed to predict controllability beliefs, inconclusive results regarding the predictive validity of the constructs emerged. The results of this study suggest that further research should be conducted specifically with controllability beliefs. Furthermore, this study provides evidence that these constructs are useful for adolescent and depressive populations and that self-report scales for family members may be a valid and effective means for measuring these constructs. Future studies will need to confirm and generalize these results for similar and other types of populations.

It is important to note that the early researchers who created measurement instruments for the EE construct were not attempting to study etiologic factors. Rather, they were interested in factors that precipitated relapse. The results of this study -- in particular, the finding that EE levels were equivalent for the clinical and comparison groups -- support the usefulness of the EE construct under these assumptions. There are apparently other factors that contribute to the development of depression since EE appeared at the same level in

families without a depressed teen. Consequently, using EE as a measure to determine which families are likely to develop a depressed member would be inappropriate. However, the predictive validity of the EE construct for relapse of mental illness provides an avenue for understanding high EE as a risk factor for further difficulties and possibly a treatment target for families with an ill member. By targeting the belief systems of family members, clinicians could modify the criticism that is expressed toward the ill individual and, thereby, avoid relapse, increase the time of remittance, or decrease the time to remittance. This model fits well with current cognitive-behavioral treatment modalities while also including the family in the goals for treatment.

A final caution is in order when making conclusions from studies of expressed emotion. Efforts to include these theories in treatment of mental illness should have as a basis the idea that the entire family system is struggling to adapt to the difficulties caused by the disorder. Treatment that views family members as the cause of problems would most likely not be beneficial. Families already feel overwhelmed. A report commissioned by the National Alliance for the Mentally Ill provided the results of a national survey of families with a child with a serious mental illness (Vitanza et al., 1999). This report showed that families struggle with great adversity, facing risks such as family dissolution, bankruptcy, or relinquishment of their children in order for them to receive treatment. Fully 50% of the families in the study reported that they felt blamed for their child's condition by treatment providers. In order for treatment to be most effective, it would, accordingly, focus on relieving burden, strengthening the family, and helping family members adapt. With this basis, family members would then be more open to exploring alternative attributions that

tend to promote lower levels of criticism in the home, thereby creating a better functioning system.

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APPENDICES

APPENDIX A:

List of Recruitment Websites

<http://health.groups.yahoo.com/group/2ndchance/>
<http://www.clubmom.com/display/295351>
<http://www.clubmom.com/display/295351>
<http://www.dbsalliance.org/>
<http://www.healingwell.com/community/default.aspx?f=19>
<http://forums.healthyplace.com/ubbthreads/postlist.php?Cat=&Board=parentingdepression>
<http://forums.healthyplace.com/ubbthreads/postlist.php?Cat=&Board=depressionchildren>
<http://www.sjwinform.org/>
<http://www.mentalearth.com/>
<http://www.moodgarden.org/forum/>
<http://www.nami.org/Template.cfm?Section=Families&template=/Security/Login.cfm>
<http://www.nami.org/Template.cfm?Section=Families&template=/Security/Login.cfm>
<http://groups.yahoo.com/group/parentsofdepressedteens/>
<http://www.strugglingteens.org/cgi-bin/ultimatebb.cgi?category=1>
<http://groups.yahoo.com/group/timeoutforfamilies/>
<http://www.topix.net/forum/health/depression>
<http://unconditionalparentingboard.com/groupee/forums>
<http://www.wingofmadness.com/forums/index.php?s=d6f0c3c7e1b3552c18a9f39e73f0d29a&showforum=43>

<http://parentingteens.about.com/mpboards.htm>
<http://forums.adoption.com/>
http://groups.yahoo.com/group/Christian_parents_of_teens/
<http://www.conductdisorders.com/>
<http://www.mlive.com/forums/parenting/>
<http://groups.yahoo.com/group/oldfashionparentingforteens/>
<http://groups.yahoo.com/group/parent-hurts/>
<http://www.practicalparent.org.uk/page22.html>
<http://groups.yahoo.com/group/parentingateen/?yguid=184284239>
<http://groups.yahoo.com/group/parentingandpunishing/?yguid=184284239>
<http://groups.yahoo.com/group/ParentingOurTeens/>
<http://groups.yahoo.com/group/parentingtroubledteens/>
http://groups.yahoo.com/group/parents_who_need_help/
http://groups.yahoo.com/group/parents_with_teens/messages
<http://www.rentscafe.com/>
<http://talk.sheknows.com/forumdisplay.php?s=&daysprune=&f=363>
http://www.troubledteensinfo.com/option.com_simpleboard/Itemid,61/
<http://forums.healthyplace.com/ubbthreads/ubbthreads.php?Cat=&C=17>

APPENDIX B:

Recruitment Letter

I am a clinical psychology doctoral student at Eastern Michigan University. I am seeking volunteer participants to complete an anonymous web-based survey of parental attitudes concerning depression in order to better understand how relationships are affected by having a depressed teen in the home. The study includes questions about your relationship with your child, what you believe about depression, and your teen's mood. Although there are no direct benefits to you, your participation in this study will provide information that could lead to greater understanding of depression and help with designing new treatment models. This study will be published as a dissertation and the results may be used in scholarly journals.

Your participation is voluntary, and your responses are anonymous. The survey does not ask you for any information that would identify you. Data are transmitted to a secure server. There is no penalty for not volunteering or for discontinuing at any time, and there are no known risks for participants. It should take 15-20 minutes to complete.

I strongly encourage you to take part in the survey and contribute to the development of better methods of assessment that can be utilized to make treatment decisions and better methods of treatment that focus specifically on each individual's problems.

You may access the survey by clicking on the link below:

http://survey.emich.edu/jmcdowell/jmcdowell_depression.htm

All participant users who visit this Web site are prohibited from digitally copying, saving, or temporarily storing the Test and may not use the Test for any purpose other than completing the administration.

Any concerns or questions can be addressed to: jmcdowell4@emich.edu or cfreedman@emich.edu (Committee Chair). This study has been reviewed and approved by the University Human Subjects Review Committee. You may contact EMU Human Subjects Committee co-Chairs for any questions regarding the consent agreement and research protocol approval procedures (Dr. Patrick Melia and Dr. Steve Pernecky, 734-487-0379).

Sincerely,

Joan McDowell, M.S.
Psychology Department
Mark Jefferson
Eastern Michigan University
Ypsilanti MI 48197

APPENDIX C:
Human Subjects Review Approval



EASTERN MICHIGAN UNIVERSITY

January 30, 2006

Ms. Joan E. McDowell
Department of Psychology

RE: "*Parental Attributions Concerning the Causes and Controllability of Adolescent Depression.*"

The Human Subjects Institutional Review Board (IRB) of Eastern Michigan University has granted approval to your proposal: "Parental Attributions Concerning the Causes and Controllability of Adolescent Depression".

After careful review of your application, the IRB determined that the rights and welfare of the individual subjects involved in this research are carefully guarded. Additionally, the methods used to obtain informed consent are appropriate, and the individuals are not at a risk.

You are reminded of your obligation to advise the IRB of any change in the protocol that might alter your research in any manner that differs from that upon which this approval is based. Approval of this project applies for one year from the date of this letter. If your data collection continues beyond the one-year period, you must apply for a renewal.

On behalf of the Human Subjects Committee, I wish you success in conducting your research.

Sincerely,

A handwritten signature in black ink that reads "Patrick Melia".

Dr. Patrick Melia
Administrative Co-Chair
Human Subjects Committee

CC: Dr. Steve Pernecky, Faculty Co-Chair
Dr. Carol Freedman-Doan.

APPENDIX D:
Depression Attribution Questionnaire

We all get sad from time to time, and many people say they are depressed. But clinical depression is a persistent depressed mood that interferes with daily functioning. Your answers on this survey can give us a better understanding of depression in order to design appropriate treatments.

1. Do you have a teen who has been diagnosed with clinical depression, or who has been hospitalized or placed on antidepressants for depression (not for bipolar disorder or schizophrenia) by a psychologist, psychiatrist or other medical professional?
A) Yes B) No (*If respondents answer no to this question, branching rules will take respondents to question 4.*)
2. How long has this child had depression?
A) Less than 6 months
B) 6 months – 1 year
C) 1-2 years
D) 2-5 years
E) 5-10 years
F) 10+ years
3. What was the severity of this child's most recent episode?
A) Very mild B) Mild C) Moderate D) Severe E) Very severe
4. Do you have a teenage child? (*only asked if answer to question 1 is "no"*)
A) Yes B) No
5. How old is this teen?
(write in box)
6. What is this child's gender?
A) Male B) Female
7. Please select any other disorders your child has been diagnosed with:
A) ADHD
B) Conduct Disorder/Oppositional Defiant Disorder
C) Anxiety Disorder (including phobia, panic disorder, PTSD, OCD)
D) Other, please specify_____
8. What is your age?
(write in box)
9. What is your race or ethnic group?
A) White, non-Hispanic B) Black C) Latino/Hispanic D) Asian E) Other
10. What is your gender?
A) Male B) Female

Teens have a variety of behaviors over which they appear to have varying degrees of control. How much control do you think your child should have over each of the behaviors listed below?
My child has _____ control over:

- A – Not observed/don't know**
- B – None**
- C – A little**
- D – Moderate**
- E – A lot**
- F – Complete**

11. Frequent crying.
12. Irritability, complaining.
13. Lack of interest in activities that were previously enjoyed.
14. Being unpopular with peers.
15. Failing to complete school assignments.
16. Conflict with parents.
17. Insomnia or sleeping all the time.
18. Being so tired, that he or she does not accomplish normal activities or won't get out of bed.
19. Poor grades.
20. Talk of worthlessness, hopelessness, or inappropriate guilt.
21. Inability to concentrate or make decisions.
22. Talking of death or suicide.
23. Low self-esteem.
24. Delusions or hallucinations (hears or sees things that others don't).
25. Complaining of body aches and pains.
26. Social withdrawal from others.
27. Tardy or absent from school.
28. Increasing/decreasing appetite or weight change.
29. Having difficulty making friends.
30. Beliefs about failure ("it's all my fault, I can't do it").

There are many theories that attempt to explain what causes depression. Please think about the following items and rate the extent to which you agree with each statement using the following scale. Clinical depression is due to:

- A – Strongly Disagree**
- B – Disagree**
- C – Neutral/undecided**
- D – Agree**
- E – Strongly Agree**

31. genetic/biological factors.
32. chance/bad luck.
33. family conflicts.
34. conflicts with others (peers, romantic partners, etc.).
35. a higher power/evil working in the world.
36. environmental difficulties (e.g. stress, illness, death of a loved one, trauma, etc.)
37. intrapersonal problems- something in particular about the individual (e.g. personality, poor choices, manner of thinking, insufficient effort, unfulfilled desires).

Thank you for your participation!

APPENDIX E:

Child Depression Inventory – Parent Version

(Kovacs, 2002)

Published and copyrighted psychological instrument has been omitted.

APPENDIX F:

Level of Expressed Emotion Scale – Relative Version

The following are a number of statements that describe the way in which you may act towards someone. Please read each statement and indicate whether you have been acting in these ways towards your teen over the past three months.

1. I say I understand if sometimes he/she doesn't want to talk.	31. I try to make him/her feel better when he/she is upset or ill.
2. I calm him/her down when he/she is upset.	32. I am realistic about what he/she can and cannot do.
3. I say he/she lacks self-control.	33. I am always nosing into his/her business.
4. I am tolerant with him/her even when he/she doesn't meet my expectations.	34. I hear him/her out.
5. I don't butt into his/her conversations.	35. I say it is not OK to seek professional help.
6. I don't make him/her nervous.	36. I get angry with him/her when things don't go right.
7. I say he/she just wants attention when he/she says he/she is not well.	37. I always have to know everything about him/her.
8. I make him/her feel guilty for not meeting my expectations.	38. I make him/her feel relaxed when I am around.
9. I am not overprotective with him/her.	39. I accuse him/her of exaggerating when he/she says he/she is unwell.
10. I lose control of my temper.	40. I will take it easy with him/her, even if things aren't going right.
11. I am sympathetic towards him/her when he/she is not feeling well.	41. I insist on knowing where he/she is going.
12. I can see his/her point of view.	42. I get angry with him/her for no reason.
13. I am always interfering.	43. When he/she is upset, I am a considerate person.
14. I don't panic when things start going	44. I support him/her when he/she needs it.

wrong.	
15. I encourage him/her to seek outside help when he/she is not feeling well.	45. I butt into his/her private matters.
16. I don't feel that he/she is causing me a lot of trouble.	46. I can cope well with stress.
17. I don't insist on doing things with him/her.	47. I am willing to gain more information to understand his/her condition, when he/she is not feeling well.
18. I can't think straight when things go wrong.	48. I am understanding if he/she makes a mistake.
19. I don't help him/her when he/she is upset or feeling unwell.	49. I don't pry into his/her life.
20. I put him/her down if he/she doesn't live up to my expectations.	50. I am patient with him/her when he/she is not well.
21. I don't insist on being with him/her all the time.	51. I don't blame him/her when he/she is feeling unwell.
22. I blame him/her for things not going well.	52. I expect too much from him/her.
23. I make him/her feel valuable as a person.	53. I don't ask a lot of personal questions.
24. I can't stand it when he/she is upset.	54. I make matters worse when things aren't going well.
25. I leave him/her feeling overwhelmed.	55. I often accuse him/her of making things up when he/she is not feeling well.
26. I don't know how to handle his/her feelings when he/she is not feeling well.	56. I "fly off the handle" when he/she doesn't do something well.
27. I say he/she causes his/her troubles to occur in order to get back at me.	57. I get upset when he/she doesn't check in with me.
28. I understand his/her limitations.	58. I get irritated when things don't go right.
29. I often check up on him/her to see what he/she is doing.	59. I try to reassure him/her when he/she is not feeling well.
30. I am able to be in control in stressful situations.	60. I expect the same level of effort from him/her, even if he/she doesn't feel well.

LEE Subscales

INTRUSIVENESS	1, 5, 9, 13, 17, 21, 25, 29, 33, 37, 41, 45, 49, 53, 57
EMOTIONAL RESP	2, 6, 10, 14, 18, 22, 26, 30, 34, 38, 42, 46, 50, 54, 58
ATTITUDE TOWARD	3, 7, 11, 15, 19, 23, 27, 31, 35, 39, 43, 47, 51, 55, 59
TOLERANCE/EXPECT	4, 8, 12, 16, 20, 24, 28, 32, 36, 40, 44, 48, 52, 56, 60

Reverse score item #s 1, 2, 4, 5, 6, 9, 11, 12, 14, 15, 16, 17, 21, 23, 28, 30, 31, 32, 34, 38, 40, 43, 44, 46, 47, 48, 49, 50, 51, 53, 59.

APPENDIX G:
Factor Analysis of Controllability Items

	Component 1	Component 2
Crying		.65
Irritability	--	--
No interest	.67	.51
Unpopular	.61	
School assignments		.85
Conflict w/ parents	.61	
Insomnia/sleeping	.85	
Tired	.81	
Poor grades		.81
Hopelessness	.76	
Concentration	.79	
Suicidal	.70	
Self-esteem	.85	
Delusions	.65	
Aches	.84	
Withdrawal	.74	.52
School attendance		.81
Appetite/weight	.76	
Difficulty making friends	.61	
Failure beliefs	.70	

Note. Only loadings >.40 are included.

APPENDIX H:
Factor Analysis of Expressed Emotion Items

	Tolerance/ Expectations	Intrusiveness	Emotional Responsivity
Say I understand if doesn't want to talk	.49		
Calm him/her when upset	.58		
Say lacks self-control	--	--	--
Tolerant when doesn't meet expectations	.44		
Don't butt into conversations	--	--	--
Don't make nervous	--	--	--
Say just wants attention when not well	-.43		
Make feel guilty not meeting expectations	-.60		
Not overprotective		-.45	
Lose temper	-.46		
Sympathetic when unwell	.59		
Can see point of view	.59		
Interfering		.53	
Don't panic when things go wrong			-.70
Encourage to seek help when unwell	.55		
Don't feel causing me trouble	.46		
Don't insist on doing things with him/her		-.47	
Can't think straight when things go wrong			.67
Don't help when he/she's upset or unwell	-.71		
Put down if not up to expectations	-.77		
Don't insist on being with him/her all time	--	--	--
Blame when things don't go well	-.62		
Make feel valuable as a person	.73		
Can't stand it when he/she is upset			.51
Leave him/her feeling overwhelmed	-.50		

	Tolerance/ Expectations	Intrusiveness	Emotional Responsivity
Don't know how to handle his/her feelings	-.48		.45
Say causes troubles to get back at me	-.65		
Understand limitations	.41		
Check up to see what he/she's doing		.46	
In control in stressful situations			-.60
Make feel better when upset or ill	.68		
Realistic about what he/she can do	.52		
Nosing into business		.76	
Hear him/her out	.67		
Say it's not OK to seek professional help	--	--	--
Get angry when things don't go right	-.55		
Have to know everything about him/her		.64	
Make feel relaxed	.60		
Accuse of exaggerating when unwell	--	-	--
Take it easy when things aren't going right	.47		
Insist on knowing where going		.55	
Angry for no reason	-.60		
Considerate when he/she's upset	.77		
Support when needs it	.84		
Butt into private matters		.78	
Cope well with stress			-.75
Willing to gain info to understand	.69		
Understanding if makes mistake	.74		
Don't pry into life		-.59	
Patient	.79		
Don't blame when unwell	.78		
Expect too much	-.48		
Don't ask personal questions	--	--	--

	Tolerance/ Expectations	Intrusiveness	Emotional Responsivity
Make matters worse	-.59		
Accuse of making things up when unwell	-.45		
“Fly off handle” when doesn’t do well	-.50		
Upset when doesn’t check in		.44	
Irritated when things don’t go right	-.55		.43
Reassure when unwell	.82		
Expect same effort, even if unwell	--	--	--

Note. Only loadings >.40 are included.

APPENDIX I:
Mean Scores for Individual Controllability Items by Group

	<i>t</i>	Clinical <i>M (SD)</i>	Comparison <i>M (SD)</i>
Crying	-2.92**	2.59 (0.87)	3.12 (0.84)
Irritability		2.99 (0.81)	3.20 (0.79)
No interest		2.70 (0.97)	2.84 (1.24)
Unpopular		2.44 (1.02)	2.36 (1.06)
School assignments	-3.89***	3.54 (1.04)	4.23 (0.93)
Conflict w/ parents		3.16 (0.84)	3.12 (0.90)
Insomnia/sleeping		2.55 (1.15)	2.35 (1.17)
Tired		2.63 (1.09)	2.74 (1.25)
Poor grades	-3.78***	3.32 (0.99)	3.94 (0.78)
Hopelessness		2.63 (1.12)	2.37 (1.15)
Concentration		2.52 (1.04)	2.31 (1.05)
Suicidal		2.60 (1.35)	2.62 (1.39)
Self-esteem		2.38 (1.04)	2.45 (1.02)
Delusions		1.81 (1.17)	1.59 (1.08)
Aches		2.48 (1.21)	2.41 (1.04)
Withdrawal		2.66 (1.00)	2.70 (1.02)
School attendance	-3.14**	3.39 (1.16)	4.08 (1.09)
Appetite/weight		2.56 (1.15)	2.60 (1.13)
Difficulty making friends		2.59 (0.98)	2.61 (0.95)
Failure beliefs		2.94 (1.15)	2.70 (1.07)

Note. Ratings were made on 5-point scales (1 = no control over symptom, 5 = complete control over symptom).

Differences between groups are indicated by *.

** $p < .01$. *** $p < .001$.

APPENDIX J:
Correlations between Individual Controllability Items and EE by Group

	Whole Sample	Clinical	Comparison
Crying	--	--	--
Irritability	--	--	--
No interest	.25**	.21*	.34*
Unpopular	--	--	--
School assignments	--	--	--
Conflict w/ parents	.23**	--	.37**
Insomnia/sleeping	.23**	.26*	--
Tired	.24**	.29**	--
Poor grades	--	--	--
Hopelessness	.21*	.26*	--
Concentration	.24**	--	.33*
Suicidal	.23*	.27*	--
Self-esteem	.23**	--	--
Delusions	--	--	--
Aches	--	--	--
Withdrawal	--	--	--
School attendance	--	--	--
Appetite/weight	--	--	--
Difficulty making friends	--	.24*	--
Failure beliefs	.22*	.29**	--

Note. Ratings were made on 5-point scales (1 = no control over symptom, 5 = complete control over symptom).

Participants were allowed to answer “not observed/don’t know” for the controllability attributions and these were excluded from analyses. N for each set of variables in the whole sample ranged from 81-143, from 54-95 in the clinical group, and from 27-49 in the comparison group.

-- nonsignificant results

* $p < .05$. ** $p < .01$. *** $p < .001$.

