THE ROLE OF FAMILY FACTORS IN CHILDHOOD DEPRESSION

DISSERTATION

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By

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ABSTRACT

Familial factors impact childhood depression in many ways. No single study has integrated the influence of genetics, family factors, and stressful events to identify the unique contribution of each to the course of childhood depression. In the current study, parent-bereaved, depressed controls and community controls from the Grief Research Study, a longitudinal study of childhood bereavement (in children ages 5-17), were examined. Children were re-categorized as initially depressed (ID: n=245) or not initially depressed (NID: n=318) based on their clinical status at the initial interview. Children and participating parents completed five interviews over 60 months.

First, survival analyses were conducted to examine the relationship between genetics, family factors, stressful events and timing of depression in the ID and NID cohorts. Second, differences in family based variables for children who became depressed following the death of a parent (bereaved depressed: BD) were compared to 1) non-bereaved depressed children (D); and 2) bereaved non-depressed (BND) children. Finally, analyses were performed to predict family variable contributions to depression chronicity.
Results indicate that BD children experience a dramatically different course of depression than depressed controls. In survival analyses, they were significantly less likely than depressed controls to experience a relapse within 25 and 60 months post-parental death. Additionally, BD children have less severe symptoms, less family history of psychopathology, fewer lifetime stressors, and less impairment on most other measures of family functioning compared to depressed controls. Within the NID group, parental psychopathology (other than mood and anxiety disorders) contributes to depression onset in the children.

When compared to BND children, BD children report more: impaired family functioning; perception of family pressure; parental affective disorder prior to the death; and family history of psychopathology in second degree relatives.

Chronicity of depression is best predicted by depression at the initial interview. Parental divorce, overall family functioning, family psychopathology, stressful life events, and family socioeconomic status also contribute to increased chronicity.

Genetics, life events and family functioning together play a role in childhood depression. These family differences may help clinicians identify those children most at risk for depression following a stressful life event such as parental death.
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INTRODUCTION

Unlike depressed adults who may suffer in relative isolation, depressed children are almost always dependent on family members. Familial factors impact childhood depression in many ways. Genetic factors account for at least 50% of the variance in the transmission of mood disorders (Birmaher et al., 1996; Kovacs, 1997). Most evidence suggests affective disorders show specificity in families (Kovacs, 1997; Wickramaratne & Weissman, 1998). However, being the child of a depressed parent is associated with sequelae beyond the genetic burden of affective disorder. Children in families with a depressed parent, typically the mother, experience more symptoms of psychopathology, decreased psychosocial functioning, and more negative images of themselves than community control children (Beardslee, Versage, & Gladstone, 1998; Downey & Coyne, 1990; Weissman, Fendrich, Warner, & Wickramaratne, 1992). The child’s difficulties are further compounded when the family environment or the marriage becomes impaired due to the maternal depression (Beardslee et al., 1998; Billings & Moos, 1983).

Family-based stressful life events also contribute to childhood depression. Stressful events may have a cumulative effect of childhood depression (Forehand, Biggar, & Kotchick, 1998). Parental death, divorce, and daily hassles are all associated with
depression in some children. Clearly, not all children experience depression as a result of stressful family events. Kovacs (1997) theorizes that the stress of a life event interacts with individual factors to contribute to childhood depression. While some life events are age-normative for children, parental death and divorce are not normative. These events plus family-based daily hassles are typically not perceived to be under the child’s control.

Unfortunately, life-events research has not firmly established which individual attributes make a child most at-risk after a family-based stressor. One would expect that children with familial loading for genetic disorder and previous depressive symptoms would be most at-risk when confronted with a stressful family event.

In addition to being associated with symptom onset, the home environment of depressed children also plays a role in the risk of relapse. Expressed Emotion (EE), the negative attitudes family members have toward their mentally ill relative, has been shown to be a good predictor of relapse in adults with mood disorders (Coiro & Gottesman, 1996; Hooley, Orley, & Teasdale, 1986; Priebe, Wildgrube, & Muller-Oerlinghausen, 1989). Family interactions have, unfortunately, been studied only to a limited degree in depressed children. Overall, families with a child with a mood disorder, have been shown to exhibit a great deal of dysfunction (Fristad & Clayton, 1991). Retrospective reports of home environments indicate that adults who experienced childhood dysthymia report poorer familial relationships than adults who did not experience childhood depression (Lizardi et al., 1995). Unfortunately, family interaction factors typically have been examined in isolation. Due to this, the contribution of family interaction patterns relative to the contribution of family genetics and stressful life events to the course of
childhood depression, and the way in which these factors interact with one another remains unknown. In their 1992 review of family interactions in mood-disordered youth, McCauley and Meyers concluded “family interactions appear to play a significant role in the timing, intensity and persistence of depressive disorder in adults. How family environment affects the presentation and course of depression in young people is not as yet clear.” Seven years later, the picture remains quite the same.

In this dissertation, I provide a brief description of childhood depression and review current longitudinal studies of childhood depression. I then review existing research on genetic factors in depression, the role of life stressors in depression and the impact of home environments on depressed children. I conclude by proposing a longitudinal examination of the role of family factors: a) in relapse for depressed children and b) in occurrence of depression in previously not depressed children.

Description of Childhood Depression

In the past 20 years, information has accumulated about the clinical presentation, course, epidemiology and family correlates of childhood depression (Kovacs, 1997). It is now known that children and adults experience similar features of MDD (Kovacs, 1995). Children provide a unique opportunity to study the longitudinal course of mood disorders as children with MDD are more likely to develop bipolar illness, and are likely to experience future episodes of affective illness (Kovacs, 1995; Kovacs, 1997).

Incidence/ Prevalence

At any given time, 0.4-8.3% of adolescents and 0.4-2.5% of children are experiencing a major depressive episode while 1.6-8% of adolescents and 0.6-1.7% of
children are experiencing dysthymic disorder (DD) (Birmaher et al., 1996). Lifetime prevalence of depression in adolescence is 15-20%, comparable to that in the adult population (Kovacs, 1997).

Suicide is the cause of 12% of teenage deaths, a fact which contributes to heightened concern for children with depressive disorders (Birmaher et al., 1996). Between 25% and 34% of depressed children and adolescents have attempted suicide by age 17 (Kovacs, 1997). Thus, childhood depression affects a considerable number of children and is a serious risk factor for premature death.

Course

The mean length of MDD in its natural course is 7-9 months. Most (90%) episodes remit by one and a half to two years after onset (Birmaher et al., 1996; Kovacs, Obrosky, Gatsonis, & Richards, 1997b). However, the probability of recurrence in children and adolescents is about 50% by 2 years and 70% by 5 years (Birmaher et al., 1996; Emslie et al., 1997). Childhood depression often persists into adulthood with adulthood recurrence rates at 60-70% compared to 27% of nondepressed psychiatric controls (Birmaher et al., 1996; Harrington, Fudge, Rutter, Pickles, & Hill, 1989; Rao et al., 1995). Childhood depression is associated with an increased risk for adulthood mood disorders but not necessarily other non-depressive psychiatric disorders. This suggests that there is considerable specificity in the lifetime course of affective disorders (Rende et al., 1997).

Several examinations of the longitudinal course of childhood depression have been conducted (Goodyer, Herbert, Secher, & Pearson, 1997; Harrington et al., 1989;
Kovacs et al., 1997b; Sanford et al., 1995). In a naturalistic follow-up study of 112 clinically referred 8-13 year olds with first episode MDD or DD, using Cox’s regression analysis and survival-analytic techniques, the duration of MDD was predicted only by underlying Dysthymic Disorder (DD) (Kovacs et al., 1997b). Double depression (MDD superimposed onto DD) is associated with the worst course (Kovacs et al., 1997b). Treatment did not contribute significantly to MDD episode duration regardless of presence or type of treatment (Kovacs et al., 1997b). In another naturalistic study of the short-term course of childhood MDD, 50% of children still met criteria, and 27% had recovered and subsequently relapsed by a 36 week follow-up (Goodyer et al., 1997). Recovery in this study was operationally defined as having fewer than two clinically significant symptoms for any diagnosis for at least eight consecutive weeks. In this study, comorbid obsessive-compulsive disorder, increased severity of depressive symptoms and age (with older children experiencing more depression) predicted depression at follow-up. Individuals who experienced persistent depression had longer illness duration before seeking treatment.

Other authors have found 66% of adolescents with MDD experience remission at one year follow-up (Sanford et al., 1995). Persisters were older, more likely to have substance use or anxiety disorders, less involved with fathers, and less responsive to maternal discipline (Sanford et al., 1995). Although few authors seem to have examined the effect of family factors in larger naturalistic studies of relapse in childhood depression, Sanford provides some evidence that the quality of relationships with parents plays a role in predicting which children relapse. Longitudinal research points to the role
of duration of mood symptoms, the effect of comorbid conditions, and family and
individual factors in determining the course of childhood depressive disorders. Some
researchers have hypothesized that neural changes brought about by the depressive
episode also play a role in predicting the course of depression (Post, 1992).

Post’s (1992) “kindling hypothesis” states that the first episode of depression is
accompanied by changes in biology which sensitize individuals to future stressors,
making the possibility of recurrence extremely likely. The kindling model posits that a
depressive episode is accompanied by long lasting changes in biological processes and
altered responsivity to stressors (Post, 1992). Post hypothesizes that these changes
include alterations in gene expression, including changes in neuropeptides and the micro-
structure of individual neurons. Thus, the first depressive episode might sensitize
patients to future affective illness under reduced stress, even small stressors. Due to this,
future multiple episodes of depression become more and more likely with each episode
experienced, making childhood depression associated with increased risk for affective
disorders in adult life because of the kindling reaction begun early in life (Harrington et
al., 1989; Kovacs et al., 1997b).

Sequelea

In addition to the problems associated with comorbidity and symptomatology,
childhood depression is associated with a range of negative developmental sequelae. It
has been suggested that childhood MDD interferes with acquisition of age-appropriate
social skills (Goodyer et al., 1997). Depressed children spend significant amounts of
time alone and are thus, “removed from the normal matrix of socialization” (Kovacs,
1997). The combination of deliberate isolation, self-deprecation and limited social skills may leave the child unable to create or sustain friendships during the depressive episode. These behaviors continue to isolate the child even after the depression subsides. The child’s withdrawal from friends and desire to spend time alone may also disrupt the child’s attachment to his/her parent, leading to relationship difficulties between the child and the rest of the family even after the episode is resolved (Kovacs, 1997).

In addition to developmental sequelae, childhood depression is associated with other psychosocial problems. Even after recovery, typically defined as an asymptomatic period for two or more months (AACAP, 1998), an MDD episode is associated with risk of increased suicidality, homicidal ideation, tobacco use, use of alcohol and other drugs, subclinical depressive symptoms, negative attributions, stressful life events such as early pregnancies and increased physical problems (Birmaher et al., 1996; Rhode, Lewinsohn, & Seeley, 1994). An episode of major depression seems to change adolescents by leaving “psychosocial scars” which are more prevalent than those seen in formerly depressed adults (Rhode et al., 1994). However, children with non-recurrent MDD experience outcomes similar to those of normal controls (Birmaher et al., 1996; Rao et al., 1995). While little research has focused on the effect of childhood MDD on the child’s family, some authors have found similar scars in the child’s relationship with parents and peers (Puig-Antich et al., 1985). In an examination of 21 children interviewed at least four months after complete recovery and one month after medication withdrawal, significant deficits remained in the child’s relationship with his/her mother,
father and siblings (Puig-Antich et al., 1985). Childhood depression is a prevalent
disorder that typically follows a pernicious course, often lasting into, or relapsing during,
adulthood. Depression in childhood seems to create a kindling effect that genetically and
psychosocially sensitizes the child to further episodes and leads to negative
developmental sequelae.

 Genetics in the Transmission of Childhood Mood Disorders

 Genetics play an important role in the transmission of childhood mood disorders. Considerable research has established genetics as a risk factor for childhood depression (Birmaher et al., 1996; Downey & Coyne, 1990; Weissman et al., 1992), but questions regarding specificity of transmission of depression remain. Being the child of a depressed parent is associated with increased risk for depression, yet what environmental variables lead to depression in individuals with a genetic predisposition, and why some children are not affected is not yet established.

 A review of nine studies of children of depressed parents suggest that, compared to control children, children of depressed parents were more likely than control children to receive a psychiatric diagnosis (including conduct disorder, attention deficit disorder, substance abuse and multiple diagnoses) (Downey & Coyne, 1990). Yet, mood disorders are the only diagnostic category in which children of depressed parents consistently receive significantly more diagnoses across studies (Downey & Coyne, 1990). Children of mood-disordered parents are three times more likely than controls to receive a mood disorder diagnosis, and children of unipolar-depressed parents are six times more likely than controls to receive a MDD diagnosis (Downey & Coyne, 1990).
By the age of 20, a child of a depressed parent has a 40% chance of experiencing a depressive episode (Beardslee et al., 1998). In a four year longitudinal follow-up, approximately one quarter of children of depressed parents received a depression diagnosis while 10% of controls received a mood disorder diagnosis (Beardslee, Keller, Lavori, Staley, & Sacks, 1993).

Offspring, Twin and Adoption Studies

Twin and adoption studies demonstrate that genetic factors account for at least 50% of the variance in the transmission of mood disorders (Birmaher et al., 1996). Children of depressed mothers who are adopted-out to families with no family history of psychopathology display high rates of affective illness (Birmaher et al., 1996). Mothers of depressed children display a 56%-73% lifetime prevalence of depressive illness (Kovacs, 1997). Parental factors associated with high risk in the child include parental depression with early onset and recurrence, and mood disorder in both parents (Birmaher et al., 1996). Lifetime risk of depression in children of depressed parents has been shown to range from 15-50% (Birmaher et al., 1996; Weissman et al., 1992). A two year longitudinal study of 174 offspring at high and low risk for depression showed that children of depressed parents were far more likely to attempt suicide and experience major depressive or anxiety episodes than children whose parent did not have an affective disorder (Weissman et al., 1992).

Specificity of Transmission

Affective disorders do seem to have some specificity in families. In first-degree relatives of depressed children, rates of lifetime depressive disorder range from 34% to
54% (Kovacs, 1997). These rates represent a five-fold lifetime risk of depressive disorder and a two-fold increase of recurrent depression when compared to the rates assessed in other psychiatric control groups (Kovacs, Devlin, Pollock, Richards, & Mukerji, 1997a). A recent study examined the association between parental MDD and depression in the offspring by the child’s developmental phase (Wickramaratne & Weissman, 1998).

Wickramaratne and colleagues (1998) found a 13-fold increase in childhood-onset MDD and a 7-fold increase in adult-onset MDD in offspring of parents with early-onset MDD. These authors hypothesize that childhood-onset MDD is a genetically homogeneous subtype of depression. The child-onset subtype has high familial loading that is not caused solely by the environment created by the depressed parent. Adolescent-onset MDD, however, may be etiologically heterogeneous, in that no strong association was found between parental MDD and adolescent-onset depression. During adolescence, risk for depression rises, especially for girls, regardless of parental history of depression (Wickramaratne & Weissman, 1998).

While most evidence points to the conclusion that mood disorders have a great deal of familial specificity, some evidence shows otherwise. In an examination of 164 sibling pairs (age 6-23) at high and 68 pairs at low risk for major depressive disorder by virtue of parental diagnosis, sibling risk in the high risk cohort was substantially greater than sibling risk in the low-risk cohort for anxiety disorder but not depression (Rende, Wickramaratne, Warner, & Weissman, 1995). What remains to be established is the role genetic risk plays in combination with other family risk factors.
In addition to the genetic burden associated with being the child of a person who suffers from a mood disorder, significant impairment seems to result from living with a parent experiencing depression. Beyond the personal effects depression has on the individual, parental depression is often associated with increased family and marital turmoil (Downey & Coyne, 1990). This creates a family environment with high levels of chronic stress. In addition, the child’s interactions with their parent, the adult upon whom they usually would rely the most, are compromised due to the parent’s limited capacity for handling relationships and daily activities.

The effect of living with a depressed parent was initially studied because children of depressed parents were used as a control group in high risk studies of the offspring of parents with schizophrenia (Downey & Coyne, 1990). While results varied by study and by the developmental stage of the children, they generally indicated that infants, toddlers and school-age children of affectively ill parents and parents with schizophrenia showed similar deficits when compared with matched controls (Downey & Coyne, 1990). These deficits include higher clinical ratings and rates of diagnoses, lower ratings of competence and higher levels of behavioral problems based on teacher and peer ratings.

When compared directly to community controls, children of depressed parents experience more: internalizing and externalizing symptoms; mental health contacts; functional impairment; social and academic deficits; and physical health complaints (Downey & Coyne, 1990). In a 2 year longitudinal follow up, children of depressed parents were more likely than children of nondepressed parents to experience additional “family risk factors” including: affectionless control, parental divorce, poor marital
adjustment, and low family cohesion (Weissman et al., 1992). Children of depressed parents also seem to have more negative self-concepts and attributional styles than children of control parents and display more guilt and interpersonal difficulties than children of control parents (Beardslee et al., 1998; Downey & Coyne, 1990). In fact, children of parents with remitted depression continue to show higher rates of problems than children of community controls, indicating that the child’s problems persist as the parent moves in and out of acute episodes (Billings & Moos, 1983). The chronic impairment and family stress which often accompany parental depression have serious implications for the child’s development above and beyond the impact of acute episodes (Downey & Coyne, 1990). A 10-year follow-up of 220 children (age 6-23 years at initial assessment) from 91 families found that children from families in which a parent had been depressed functioned at lower levels at home, in work, within marriages and families than children who were not the offspring of a depressed parent (Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997).

In order to explore whether deficits seen in children of depressed mothers are general and due to an impairment of maternal functioning or are more specific to maternal depression, comparison groups of medically ill mothers have been used. In one comparison, children of women with unipolar depression displayed more significant deficits in functioning (as measured by the CBCL) than children of women with bipolar disorder, medical illness, or no psychiatric or physical illness (Anderson & Hammen, 1993). These differences were apparent across a two-year follow-up even when differences in SES were statistically controlled (Anderson & Hammen, 1993). Unipolar
depression is hypothesized to be associated with more impairment for offspring because women with unipolar depression, when compared to women with bipolar disorder, tend to experience more depressive episodes overall which increases their child's exposure to the stressors related to depressive episodes (Anderson & Hammen, 1993).

As depression is associated with reduced behavior and speech, increased hostility irritability, and dysphoria, depressed persons are unlikely to foster positive relationships with significant others (Downey & Coyne, 1990). Mothers with a history of depression show more affectively charged negative statements than children of mothers without a history of mood disorders (Goodman, Adamson, Riniti, & Cole, 1994). Reviews indicate that depressed parents view the role of parent less positively than controls, experience negative emotions about the demands of parenthood, feel increased rejection and hostility towards their child, and perceive themselves to be less competent than other parents (Downey & Coyne, 1990). Observational studies confirm that depressed mothers are less active, less positive, more irritable and more distant than nondepressed mothers (Downey & Coyne, 1990). Thus, depressed parents are more likely to be either unavailable and unresponsive or critical and rejecting. Infants of depressed mothers direct their behavior towards their parents less frequently, smile less and are more irritable than children of controls. This behavior generalizes even to strangers (Downey & Coyne, 1990).

Regardless of the basis for these differences, these children then become more difficult to parent.

The way in which a depressed parent's behavior exerts an effect on the child has not been fully explained. Initially, the differences reported between children of depressed
mothers and children of non-depressed mothers were thought to be a result of depressed women reporting overly negative views of their children’s behavior (Anderson & Hammen, 1993; Downey & Coyne, 1990). Recent research indicates that this is not the case; depressed women are able to describe accurately their children’s behavior (Anderson & Hammen, 1993). One hypothesis for how the mother’s illness affects the child is that the mother’s affective illness creates a stressful environment for the child at the same time the child’s own deficits interfere with their further development of competencies (Anderson & Hammen, 1993). Children of depressed parents who experience depression themselves seem to be in-episode proximal to their parent’s depressive episode (Hammen, Burge, & Adrian, 1991). These findings support the view that both the parent and child contribute to each other’s current and future difficulties (Downey & Coyne, 1990) and suggest a bi-directional interaction, in which genetics and environment interact to affect both children and their parents.

In families in which a parent was depressed but marital and family stress were low, only 10% of children experienced behavioral difficulties as compared 25% in all children of depressed parents and 3% in control families (Billings & Moos, 1983). In addition, marital stress, parenting problems, and severity of parental depression are all associated with increased rates of depression in offspring (Beardslee et al., 1998). Many women with unipolar depression themselves marry men with diagnosable disorders who contribute to the child’s genetic burden and leave the marriage leading to economic difficulties and further emotional difficulties for the mother and her offspring (Hammen, 1992). These findings lend support to a theory in which parental depression places a
child at special risk for depression when the child is in a high stress environment (Downey & Coyne, 1990). Thus, it appears that risk factors for childhood depression all interact.

**Family Based Stressful Life-Events**

Stressful life events are often present prior to the onset of depression. Of depressed adults, 60-70% have experienced one or more major stressful life events in the year prior to MDD onset (Birmaher et al., 1996). Although research has demonstrated a relationship between stressful life events and adjustment, the evidence for a causal role of unique events in psychopathology is weak (Compas, 1987). Individuals who are most likely to become depressed following stressful events are those with a history of major depression (Hammen, 1992).

Major life events rarely account for more than 15% of the variance in psychiatric symptoms in children and adolescents (Compas, 1987). The lack of variance accounted for by major life events is, in part, age-related. Perhaps in childhood and adolescence, more major life events are normative and age-linked (e.g., changes related to school such as the end of a school year and graduation) than in adulthood (Compas, 1987). However, if these events are experienced out of the usual time frame, they might be associated with increased distress including depression or anxiety (Compas, 1987). One way to conceptualize the relationship between stressful life events and depression is to suggest that stressful events may “trigger” depression in children but these triggers must interact with individual or “person-specific vulnerabilities in ‘causing’ depression” (Kovacs, 1997).
Life-events might become stressful when they are perceived as under the control of the individual. When 39 depressed adolescents were compared to community controls with no history of psychiatric disorder, groups did not differ in the number of overall negative life events they marked on a life-event checklist (Williamson, Birmaher, Anderson, Al-Shabbout, & Ryan, 1995). However, depressed adolescents reported significantly more dependent life events (i.e., school suspension, unwed pregnancy, increased arguments with parents) than the community controls. While Williamson and colleagues were not able to make causal statements based on this cross-sectional study, it seems clear that adolescent depression is associated with increases in dependent life events.

It is also possible that even if stressful events do not initially trigger depression, they may become risk factors for subsequent emergence of clinical depression (Kovacs, 1997). Stressful events may have a cumulative effect on childhood internalizing disorder. Forehand et al. (1998) examined the relationship between number of family “risk factors” and short and long-term internalizing symptoms in a community sample of 285 children who were assessed at baseline (age eleven to fifteen) and six years later. These authors specifically examined parental divorce, interparental conflict, maternal physical health and affective disorder, and conflict in the mother-adolescent dyad (Forehand et al., 1998). Over half the mothers reported two or more family risk factors and 20% reported three or more. The number of family risk factors reported was associated with longitudinal report of depressive symptoms for the adolescent but not with concurrent difficulties in adjustment. An increase from three to four risk factors was associated with a significant
increase in depressive symptoms by young adulthood. Thus, Forehand, et al. suggest that the consequences of experiencing multiple family stressors might not be evident until several years later. While this study did not use a clinical sample and relied on self-report of depressive symptomatology, it does provide some evidence that stressful events exert a cumulative effect. It also suggests that children who have experienced a number of stressful life events need to be followed longitudinally in order to assess the results of these events. Of note, the Forehand study defined maternal-adolescent conflict as a stressful event in itself, an issue which speaks to the interaction of family-based stressful life events and family climate. With this in mind, we turn to examinations of some specific family stressors that have been studied in depth, parental death, divorce, and daily hassles.

**Parental Death**

Current evidence indicates that, contrary to Freud’s assumptions, “evidencing an objective loss is neither sufficient nor necessary to cause” a depressive disorder. “In particular, there is scant evidence of a direct causal relationship between major loss and depression in the younger years” (Kovacs, 1997). However, the death of a parent is a traumatic event for children. In a cross-cultural study of almost 2,000 children in six countries, children rated loss of a parent as the most stressful of 20 events. Loss of a parent was broadly defined in this study to include loss from not only death, but events such as divorce and abandonment. This was categorized by the children as more
upsetting than events such as going blind, being held back a year in school, and parental fights (Yamamoto et al., 1996). In America, approximately four percent of children lose a parent to death before they reach the age of 15 (Census, 1985).

Parental death is associated with depression in some children. Following the death of a parent from causes other than suicide or homicide, one-third of 38 parent-bereaved children met DSM-III-R criteria for a major depressive disorder (Weller, Weller, Fristad, & Bowes, 1991). In this study, greater numbers of depressive symptoms were associated with having a mother as the surviving parent, a previous history of psychiatric disturbance in the child, a family history of depression, and being from higher socioeconomic status.

However, it is notable that most children do not develop a diagnosable depressive disorder after such a profound loss. If depression was a “normal” response to major loss, we would expect a much higher rate of depression after parental death (Kovacs, 1997). Despite experiencing depression, parent-bereaved children from stable families experienced few differences in psychosocial functioning such as school performance and peer relationships when compared to non-bereaved community controls (Fristad, Jedel, Weller, & Weller, 1993). Based on kindling theory, children who experience the death of a parent and subsequently experience depression may be more at risk than children who do not experience depression after the death. Many studies have shown that adults who experienced parental death during childhood were at risk for later-life mood disorder
Children have been shown to experience guilt and dysphoria five years after parental death and are more likely to seek psychological treatment five years after parental death than at any earlier time (Cain & Fast, 1966; Rutter, 1966).

**Divorce**

Children from divorced and remarried families are more likely to exhibit internalizing disorders than children from never-divorced families. Children from divorced families are also more likely to experience academic problems, problems with relationships, problems with self-esteem and more externalizing behaviors (Hetherington, Bridges, & Insabella, 1998). However, the effect size of these results is small and these elevations in children’s emotional distress seem to be short-lived for most children, dissipating within two years after the divorce (Aseltine, 1996; Hetherington et al., 1998). For some children, especially in families characterized by chronic parental conflict, divorce may be associated with more positive adjustment over the long-term (Kelly, 1998, April). However, for other children, divorce may expose the child to more conflict than he or she experienced during the marriage, may lead to rejection from the non-custodial parent, and may place the child at risk for depression and other mental health problems (Kelly, 1998, April).

The effect of the divorce on the child depends on the quality of the family relationship, individual characteristics of the child’s temperament, the stress of the life transitions associated with the divorce, and individual characteristics of the parents (Hetherington et al., 1998). Many authors argue that divorce is problematic for children
because it is accompanied by other stressors such as family economic hardship or persistent family conflict which are more problematic to the child than the divorce itself (Aseltine, 1996).

In an examination of self-report data from over 800 high school students included as part of a prospective study of stress in the high school years, Aseltine (1996) found support for the hypothesis that the divorce is stressful not because of the event itself but because of the stressors associated with living through a divorce. Teens living with a single parent showed significantly more depressive symptoms than teens living in intact and remarried families (Aseltine, 1996). While these aggregate associations were weak, teens in these single-parent families reported more negative events, financial problems and unsupportive and conflictual relationships with parents. Correlations between these events and depressed mood were moderate. Family-conflict, parent-child relationship problems, family financial problems, personal and family stresses were all associated with high depressive symptomatology while parental warmth was associated with lower levels of depressive symptomatology. However, when exposure to these “secondary” problems resulting from divorce were controlled, no differences were found in levels of depressed mood between teens from single-parent or intact families. Thus, it does not seem to be divorce itself which leads to depression but many of the stressors associated with divorce (Aseltine, 1996). Family financial difficulties resulting from divorce are common and the impact of this on the teen is profound (Aseltine, 1996).

Aseltine (1996) also found a great deal of variability in the effects of these mediator variables on teens. Family-conflict and parent-child relationship problems
seemed less problematic for teens in single-parent families while financial problems were much more stressful to teens in single-parent families. In single-parent families, parental warmth seemed to be more of a protective measure than for teens in intact families. As divorce is often associated with diminished parenting and increased disorganization of family life, opportunities for the teen to become involved in individually stressful events (e.g., trouble with the law, pregnancy) are heightened (Aseltine, 1996).

Finally, some authors have found that frequent marital conflict and the child’s level of symptomatology and adjustment at the time of the parent’s separation are the most important factors in predicting the child’s post-divorce adjustment (Kelly, 1998, April). A child who has been exposed to the chronic daily strain of parental conflict, who feels very sad and guilty about the conflict, and, as a result of the divorce, faces economic hardship and an absentee depressed single-parent may be at highest risk for the development of depression.

**Daily Hassles**

Children with more disrupted lives including multiple moves, parental job changes, and disorganized home lives appear to be more at risk for problem behaviors (Masten, 1989). Prospective studies show that chronic strains and daily stressors are more highly associated with psychological and behavioral difficulties during adolescence than major life events (Compas, 1987). This is a bi-directional relationship, as depressed children are more likely to have experienced this chronic stress and being depressed can cause some of these stressors such as being out of school or work or experiencing increased familial conflict. In addition, individuals from low SES families are more often
faced with chronic stress and daily hassles. Low SES, then, is a specific stressor for depression as it predicts the recurrence of depressive episodes (Rao et al., 1995).

Children have much less control over their life circumstances than adults and might be differentially affected by chronic family stresses over which they have no control. Due to these findings, both major life events and daily hassles should be taken into account in any comprehensive study of family factors in childhood depression.

Home Environment

Little research has focused directly on the role the home environment plays in childhood depression. When a caregiver has critical or hostile attitudes about an individual with a mental illness, this can greatly impact the course of the illness.

EE and Childhood Mood Disorder

Expressed Emotion (EE) refers to a family member’s report of emotional aspects of family communication. In a high EE family, one or more key relatives display attitudes which are either critical and hostile, or emotionally overinvolved. Emotional attitudes about a specific individual, in particular, attitudes of criticism and/or emotional overinvolvement, have been shown to predict clinical deterioration or relapse in various psychiatric disorders. Since the initial studies of EE in families of people with schizophrenia, high EE has been associated with relapse rates in relatives of individuals with depression, alcoholism, bipolar disorder, and even obesity (Kuipers, 1992). Overall, a review of the literature on EE in relatives of depressed patients indicated that depressed adults with high-EE relatives are about 13 times more likely to relapse than those with low-EE relatives (Cairo & Gottesman, 1996).
Few studies have directly examined the effect of familial EE on children, especially those suffering from mood disorders. Children might be even more directly influenced by attitudes of family members than adults because children do not have the option to be independent of these family members (Asarnow, Goldstein, Tompson, & Guthrie, 1993). Depressed children, because they often withdraw from peers and social activities outside the home and are more dependent on parents and other family members, can be particularly influenced by the affective climate in their home (Asarnow et al., 1993).

Asarnow and colleagues (1993) examined the association between 26 children’s one year post-hospitalization outcome and the level of EE in their homes when they returned from the hospital. Results indicate that none of the 11 children in the high EE group, based on maternal EE measures, were categorized as recovered at one year follow-up. By contrast, 53% of the 15 children in the low EE group were categorized as recovered in that same time period (Asarnow et al., 1993). This study shows that EE is highly predictive of one year outcome for children with mood disorders, replicating findings with depressed adults. Asarnow (1993) also examined the predictive power of parental EE when controlling for sociodemographic factors of age, gender, SES and single versus dual-parent families and “clinical factors” including the presence of comorbid diagnoses, presence of treatment post-hospitalization, and chronicity of the mood disorder. Sociodemographic and clinical factor variables were entered into a hierarchical regression model prior to EE scores. EE continued to predict outcome when variance accounted for by each of these variables was controlled (Asarnow et al., 1993).
While the generalizability of Asarnow and colleagues results are somewhat limited by the small sample size, the findings suggest that family EE is a powerful predictor of outcome in children who have been hospitalized for mood disorders.

Another study conducted by Asarnow and colleagues assessed expressed emotion level in families of 86 children (ages 6-14) with depressive disorders, children with schizophrenia spectrum disorders and community controls (Asarnow, Tompson, Hamilton, Goldstein, & Guthrie, 1994). Results from this study indicated that EE levels are higher in families of depressed children than children with schizophrenia spectrum disorders and normal controls. The outcome was independent of mediating variables such as SES and severity of illness. This suggests EE, particularly high critical EE, is a specific risk factor for depression (at least in contrast to children with schizophrenia spectrum disorders) and not just a non-specific correlate of child psychopathology (Asarnow et al., 1994).

Expressed emotion has been widely studied in adults and seems to be a good predictor of relapse. The construct is less widely studied in children, especially children with mood disorders. Children in high EE families are more likely to relapse than children in low EE families. High EE appears to be a specific risk for childhood depression and comorbid conduct disorders.

Other Family Interactions

The manner in which family members relate to the identified patient and communicate with each other can have a profound impact on the course of the patient's illness. Overall, families with a mentally ill child experience a high level of dysfunction
and conflict. Family dysfunction, as measured by the Family Assessment Device (FAD), a self-report assessing areas such as family problem solving, communication, roles, affective responsiveness and involvement, was reported in 89% of 100 consecutive outpatient children (Fristad & Clayton, 1991). Children with mood disorders showed fewer indications of family dysfunction than children with behavior or anxiety disorders (Fristad & Clayton, 1991). However, one difference which has been found is that parents of depressed children exhibit more conflict over child-rearing practices than parents of children with other psychiatric problems both during the child’s acute depressive episode and after recovery (Puig-Antich et al., 1985). While some studies have found differences in the family environments of depressed and non-depressed children, research in this area is sparse.

Retrospective Reports of Family Environment

Another way the relationship between childhood mood disorders and family relationships have been explored is through retrospective reports of adults with child-onset depression. An example of retrospective reporting can be seen in Lizardi’s (1995) examination of retrospective reports of the childhood home environment of adults with childhood-onset dysthymia. This study indicated that patients with early-onset dysthymia report poorer relationships with both parents, lower levels of parental care, and higher parental overprotection than adults who had not experienced childhood-onset dysthymia (Lizardi et al., 1995). The use of retrospective report in patients who have experience prolonged dysthymia without a childhood-onset major depression control group limits the applicability and generalizability of this work to the current examination
of the home environment of depressed children. Nevertheless, this study does suggest that childhood mood disorder is associated with problematic family relationships. However, retrospective reporting is problematic. When symptoms last longer than a year, children and their parents are not able to reliably retrospectively report even the year of symptom onset (Angold, Erkanli, Costello, & Rutter, 1996). As retrospective reporting does not give an accurate representation of the timing of the child’s depressive episode, retrospective reporting probably does not give an accurate picture of the home environment while the child experienced depression.

Interactions between Genetics, Life Events, and Family Variables

Individuals at high genetic risk are also more sensitive to the effects of stressful environment than individuals at low genetic risk (Birmaher et al., 1996). Therefore, a child genetically at risk for depression who experiences high rates of parental criticism and stressful life-events might be more at risk than a child not genetically at risk who experiences the same set of circumstances. By the same measure, a child who experiences lower parental criticism than her twin-sister who is irritably depressed will be less at risk for depression despite her genetic loading for affective disorder. Unfortunately, research relating the role of genetics to the role of family factors in the course of childhood depression is quite limited.

Reiss and colleagues’ study (1995) shows that family climate is strongly associated with adolescent depressive symptoms. In a study of 708 families with two same-sexed adolescent siblings, 37% of the variance in depressive symptomatology could be accounted for by conflictual and negative parenting behavior directed at the
adolescent. The conflict/negativity construct accounted for 31% of the variance in fathers’ and 41% of the variance in mothers’ report of children’s depressive symptoms. Warmth/support, on the contrary, was associated with positive outcome in Reiss’ sample of adolescents. Adolescents from families high in conflict and negativity show higher rates adolescent depressive symptoms than adolescents from families with low levels of conflict (Reiss et al., 1995). When the harsh, aggressive and inconsistent parenting was directed towards a sibling, psychopathology was less evident in the proband (Reiss et al., 1995). This indicates that genetic data would benefit from direct measurement of environmental data, as environmental variables specific for each sibling in a family have the biggest impact on normal and pathological development (Reiss et al., 1995). Genetics are influential in determining risk for affective disorder. However, genetics and environmental factors act in conjunction at every level, with genetics providing the biological vulnerability and stressors providing diatheses for the expression of genetic phenotype.

Maternal psychopathology also plays a role in determining family relationships. In a study of 79 adolescents with a lifetime diagnosis of major depression compared to adolescents who had never experienced depression, only the previously-depressed adolescents with mothers who had experienced a depressive episode reported significantly poorer family functioning in variables such as family cohesion, parent-child conflict, maternal involvement and parental regard for the child (Shiner & Marmorstein, 1998). Most adolescents in the study with a history of depression were not currently depressed but still reported more of these differences in their family environment than
adolescents who had never experienced depression. While these results should be interpreted with some caution because they come from an epidemiological study of twins, they demonstrate the interplay between genetic and family environment factors in adolescent depression, a rarely examined area.

Children of chronically depressed parents are typically exposed to high levels of stressful life events and chronic stress but do not have a responsive and available parent to help them cope (Hammen, 1992). These children might also have maladaptive internal coping mechanisms which leave them vulnerable to depression (Hammen, 1992). Maternal history of depression and recent life events have been shown to be related to depression onset in a sample of 82 non-referred adolescent girls (Goodyer, Cooper, Vize, & Ashby, 1993). Despite these few exceptions, studies of life-events generally do not take into account genetic and family influences. The evidence suggests genetic liability, family psychopathology, and family climate lead to conditions which are associated with child depressive symptomatology. Unfortunately, research which can help to answer how these influences interact with each other is limited.

Summary

Childhood depression is a prevalent condition which is associated with substantial developmental sequelae. Genetic transmission plays an important role in determining which children will experience depression but these factors account for only half the variance (Birmaher et al., 1996; Downey & Coyne, 1990; Weissman et al., 1992). Likewise, stressful life events such as death, divorce or chronic daily hassles often proceed onset of a depressive episode but research has not found that these play a causal
role in the onset or recurrence of childhood depression (Compas, 1987). High expressed emotion has been shown to be risk factors for relapse of childhood depression (Asarnow et al., 1993; Asarnow et al., 1994) but the evidence for the role played by other family factors is limited.

No study has examined the combined role of family variables on the course of childhood depression. Community samples are much more widely used than clinical samples in stressful life event research and family stressor research. While it is much easier to examine large samples of non-clinical families, family relationships in more severely depressed children may be very different from those seen in community samples. In addition, most research in these areas is cross-sectional. The lack of longitudinal data is notable in that little comment can be made about the course of depression with such data. Another significant problem is that family based risk factors are typically examined individually, with minimal integration of factors.

The current study integrates the influence of genetics, family factors, and stressful events so that the unique contribution of each to the course of childhood depression can be determined. Data will come from the Grief Research Study, a longitudinal study of childhood bereavement which has followed three cohorts of children, parental-bereaved, clinically depressed and community control, over a five year period. These three cohorts will be divided into two roughly equal groups, depressed (i.e., the clinically depressed group and the bereaved children depressed at their initial interview) and nondepressed (i.e., the community control group and the bereaved children who were not depressed at
their initial interview). Analyses will be conducted for the depressed and nondepressed children separately, to determine if the same variables play a role in development of a new episode of depression as do for the recurrence of depression.

As previous studies indicate that genetics, stressful family events and home environment each contribute to depression or relapse, I hypothesize the following will be related to timing of relapse: 1) high familial loading for affective disorder; 2) high familial loading for overall psychopathology; 3) concurrent parental depressive severity; 4) having experienced parental death; 5) having experienced parental divorce; 6) overall level of stressful events experienced; and 7) a family environment characterized by high levels of criticism and low levels of shared activities.
CHAPTER 2

METHOD

Procedure

Children and their surviving parents were evaluated separately approximately one month after the death of one of their parents (spouse). All participants gave informed assent/consent prior to the interview. Face-to-face interviews were conducted simultaneously by different interviewers in the family’s home or in study offices. Community control children were paid $10 at the 1, 6, and 13 month interviews and $20 at the 25 month interview. All participants were paid $40 at the 60 month interview. Initial interviews took 2-4 hours while follow-up interviews took approximately 1-3 hours. Interviewers were highly trained staff, graduate students and undergraduates who obtained interrater reliability measures of greater than .80 before interviews were conducted independently. The initial interview assessed symptoms and events which occurred since the death. It also included assessment of premorbid functioning in the month prior to the death and over the child’s lifetime. Families were recontacted and interviewed again 6, 13, 25 and 60 months after the death.
Participants

Participants were recruited as part of the Grief Research Study, a longitudinal study of childhood bereavement. They included 368 children bereaved from parental death, 111 depressed, and 129 community control children. Participants completed one or more interviews at 1, 6, 13, 25, and 60 months post-parental death. Bereaved families were recruited through daily examinations of obituaries from local papers, as well as from contact with local funeral homes. Families then were contacted by phone and asked to participate. Approximately one-third of eligible families chose to do so. Despite special efforts to recruit non-Caucasian families, 98% of participant families were Caucasian. No child had experienced sibling death. All had experienced the death of only one parent. Children and their deceased parent had regular contact over the two years pre-death.

Depressed participants were recruited after chart-reviews of inpatients and outpatients in the Division of Child and Adolescent Psychiatry. Parents were contacted by phone and asked to participate. All children in this group received a diagnosis of MDD, Dysthymia or Bipolar Disorder, Depressed prior to study entry. As the relationship between familiar stressors and relapse of depression might be associated with a different course for those children diagnosed with Bipolar disorder, those children were eliminated from the analyses for the current project. No child had ever experienced the death of an immediate family member and none had experienced the death of a close relative in the two years prior to study entry.
Community control participants were recruited through contact with local church
groups and schools. No control participant had ever experienced a death of an immediate
family member, and none had experienced a death of a close relative in the past two
years. No community control or their family member had received mental health
treatment in the two years prior to study entry.

Measures

Child depression measure. The Diagnostic Interview for Depression in Children
and Adolescents-(DIDCA) is a structured interview to assess depressive symptoms in
children and adolescents (Weller & Weller, 1979). The DIDCA has excellent sensitivity
and specificity (Fristad, Weller, & Weller, 1995). The DIDCA uses various questions, in
language familiar to children, to ask about each of the nine symptoms of depression. If
one or more questions within a diagnostic section is endorsed, the symptom is considered
to be present. An algorithm was constructed to calculate depressive diagnoses based on
endorsement of one or more cardinal symptoms and five total symptoms of depression.

Family psychopathology measures. The Hamilton Rating Scale for Depression
(HRSD) is a severity rating scale used to assess depressive symptomatology in adults
(Hamilton, 1967). The Ham-D was administered at each interview to the surviving parent
to assess the severity of his or her depressive symptoms during the two weeks prior to the
interview.

The Psychiatric Diagnostic Interview (PDI) is a structured diagnostic interview to
assess 13 psychiatric diagnoses in adults (Othmer, Penick, Powell, Read, & Othmer,
1989). Reliability and validity studies are acceptable (Othmer et al., 1989). In this study,
the PDI was used to ascertain presence of psychiatric diagnoses in the surviving (informant) parents at five time periods: 1 month pre-death, 1 month post-death, 13 months, 25 months and 60 months.

The Family History-Research Diagnostic Criteria (FH-RDC) Interview is a semi-structured interview designed to assess symptoms of psychopathology (20 disorders) in first-degree relatives of the informant (Endicott et al., 1978). The FH-RDC was administered to the surviving parent at the initial, 13 month, 25 month, and 60 month interviews and was also administered retrospectively at the one month interview to assess psychiatric symptomatology in the deceased parent pre-death.

Life events measures. The Diagnostic Interview for Children and Adolescents-Revised (DICA-R) is a structured interview designed to assess presence or absence of DSM-III-R symptomatology and psychosocial stressors in children (Reich and Welner, 1988). In the current study, a scale was created from the psychosocial stressors section. The scale sums the 11 psychosocial stressors assessed on the DICA (i.e., familial arguing, divorce/separation of relatives, child abuse, abuse of other family member, financial problems, death of a close relative or friend, death by accident or murder of a someone the child knows well, serious illness in the family, problems with the law, anxiety about someone in the home getting hurt, substance abuse in the family). This interview covers eight time periods: 1 month pre-death, 1 month post-death, events at any time since last interview (SLI) at 6, 13, 25 and 60 months, and in the past 2 weeks (current) for 6, 13, and 25 month interviews.
The Coddington Life Events Scale asks the parent to report which of 36 stressors have occurred in the child/adolescent's life (Coddington, 1983). This instrument was used to indicate the types of stress the child and adolescent had experienced since the last interview at 6, 13, 25 and 60 months (SLi).

Family functioning measure. The Home Environment Interview- Abbreviated Version- Child and Parent Forms is a semi-structured interview designed to gain information regarding the quality and quantity of familial and nonfamilial interactions prior to and following the death (Robbins, 1983). In the initial interview, yes/no questions are asked about several types of parental criticism, and the child’s opinion of positive and negative aspects of their parent’s behavior. The interview also includes questions about the types of activities in which the child and his/her parent(s) participate. Five categories of activity are coded: going places together; participating in activities at home; doing schoolwork or homework together; talking and arguing; and “other” activities such as a hobby at which the child and parent work together. These items were compiled into a scale ranging from 0-5 (0=no activities; 5=all activities). The parent version also includes a question which asks if the parent believe he/she and his/her spouse do “a lot” with the child and questions about activities the entire family does together including: going places; dong household chores or spending time together at home; talking as a family; attending worship services; and doing other types of activities as a family. These items were compiled into a scale with a range of 0-5 (0=no activities; 5=all activities). At the initial interview, pre-death information on family functioning was obtained.
Data Analysis

Time to relapse was examined using a survival analytic technique that can accommodate censored observations, i.e., those individuals who drop out of the study before relapse has occurred. Using this procedure allows for use of the entire cohort to determine distributions of time to relapse. This technique is fully described in Kovacs et al., (1997, p.779). The relationship of family covariates and time to relapse will be examined using Cox’s proportional hazard model, also known as Cox’s regression (Cox, 1972). As some covariates change across time, the model allows for individual differences in each covariate across time. For each covariate, the model yields an estimate of the regression coefficient and its standard error. Positive coefficients indicate that higher values of the covariate are associated with an increased hazard rate and shorter time to relapse. A forward stepwise-selection was used with a criterion of \( p < .05 \) for the improvements in chi-square, to retain a covariate in the model. Assumptions for the proportional hazards were checked for all significant covariates.
CHAPTER 3

RESULTS

Participants

Five depressed participants were deleted from analyses as these children were diagnosed with bipolar disorder—depressed and evidence suggests different genetic factors may influence childhood-onset bipolar disorder and depression. Thirty-five bereaved children were also excluded from analyses because they did not complete an initial interview and so could not be classified based on presence/absence of depression in the first month post-parental death.

The remaining participants were divided into two groups based on the presence/absence of a depressive episode at the initial interview: 1) 245 children were categorized as Initially Depressed (ID). The ID cohort included 106 children from the depressed cohort (43%), 137 (56%) from the bereaved cohort (this represents 42% of the complete bereaved cohort), and 2 from the community cohort (1%). 2) 318 children were categorized as Not Initially Depressed (NID). The NID cohort included 127 community controls (40%) and 191 bereaved children (60%).
ID children were more likely than NID children to: be older; come from families in which a divorce or separation had taken place; come from a family with lower socioeconomic status; and have a younger informant parent (see Table 1). No other demographic variables differentiated ID from NID participants.

Variable Selection

Stressful events variables.

1. Coddington. Initially, the Coddington Life Events Scale (Coddington, 1983) was to be used to measure types of stress the child and adolescent had experienced. Unfortunately, this instrument was administered as part of a packet of self-report instruments the participating parent was instructed to complete and mail back. As no monetary incentives were available until the 5 year interview, approximately half of the Coddington scores are missing at each time period. The decision was made to not include Coddington data in the survival analyses as their limited numbers would decrease the overall sample size available for analysis, thereby greatly limiting power.

2. DICA stressors scale. A scale summing parent and child report of the 11 psychosocial stressors assessed on the DICA was constructed. This yielded a potential range of 0-44 at the initial time period to account for “lifetime” (pre-death) stressors [11 stressors X 2 informants x 2 time periods(lifetime, past month)] and 0-22 at subsequent time periods (11 stressors X 2 informants x 1 time period).

Parental psychopathology variables.

1. Family History of Psychopathology: FH-RDC. The FH-RDC (Endicott et al., 1978) was used to examine lifetime history of psychopathology in the deceased parent
and family history of psychopathology in second degree relatives and siblings over the age of 18. The number of second degree relatives with symptoms and/or diagnosis for each of 20 disorders are recorded. As it was unknown whether the total number or percent of relatives endorsing symptoms/diagnosis for each disorder should be examined, two scales were created and compared. The Family History Second Degree Sum (FH2SUM) variable sums endorsements of each diagnostic category if any second degree relative has endorsements in that category. The FH2SUM gives full weight to each diagnosis endorsed and half weight to each disorder for which symptoms only are endorsed [e.g., (any relatives with diagnosis of depression) + .5 x (any relatives with symptoms of depression) for each disorder]. This yields a possible range of 0 (no relative with symptoms/diagnosis of any disorder) to 20 (at least one relative with a diagnosis of each disorder).

The Family History Second Degree Percentage (FH2PER) variable sums the percentage of all second degree relatives with diagnoses of each disorder and adds to this the half weight percentage of all second degree relatives with symptoms of each disorder [e.g., (# relatives with diagnosis of depression/# second degree relatives) + .5 x (# relatives with symptoms of depression/# second degree relatives)]. This yields a potential range of 0 (no endorsements for any second degree relative) to 20 (all second degree relatives have diagnoses of each disorder).

Results from these two scales were compared between ID and NID as it was thought that the scale which indicated the largest between group difference would be the most sensitive measure of family psychopathology. The two scales were very similar in
their ability to differentiate groups (see Table 2). As the FH2SUM scale is more easily interpreted [scores indicate the cumulative number of disorders in which any extended family member has symptoms (half-weighted) and diagnoses], it was used in all subsequent analyses.

2. Psychopathology in the surviving/informant parent: PDI. As it was thought that different types of parental psychopathology would differentially impact onset/relapse of childhood depression, a variable was created to sum mood and anxiety endorsements (mania, depression, OCD, panic disorder) by the surviving parent (PDI-Mood and Anxiety) while another variable summed behavior and other disorder symptoms (alcohol abuse, drug abuse, antisocial personality, schizophrenia, organic brain syndrome, hysteria, anorexia, phobia, and mental retardation; PDI-Behavior and Other Disorders). If a parent endorsed symptoms but no diagnosis for a disorder they received a 1 for that disorder. If they endorsed symptoms equivalent to a diagnosis, they were given a 2 for that disorder. For each scale, component disorders were summed. On the BDI-Behavior and Other Disorders scale, phobia was down-weighted by half so that phobia endorsements did not disproportionately impact outcome. The PDI-Mood and Anxiety scale had a potential range of 0 (no endorsements) to 8 (diagnosis of all four disorders) and the PDI-Behavior and Other Disorders scale had a potential range of 0-17. PDI indices were compared between ID and NID groups at all time periods to ensure the PDI could be used to successfully differentiate groups (see Table 3).
3. Depression symptom severity in the surviving/informant parent: HRSD. The HRSD total score (potential range 0-71) was used as the measure of level of parental depressive symptomatology at each time period.

**Family environment variables.**

1. Family Functioning Scale. This scale is comprised of ten positive (i.e., showing interest in the child’s friends and activities, making a big deal out of birthdays, making a big deal out of holidays, attending sporting events or performances, appearing to enjoy their children, showing affection, being available to comfort child, being consistent, being easy to talk to, parent doing “a lot” with the child) and 11 negative items (i.e., complaining a great deal about having too many children; frequently finding fault with children; telling child they do not love him/her; being away from home a great deal; breaking promises; pressuring the child to much to: do well in school; do well in sports; be artistic or musical; only spend time with people he/she knows or approves of; be religious; and be polite and obedient) asked about each parent. Family functioning (famfun) is calculated by subtracting negative items from positive items. This yields a scale with a potential range of -22 to 20 \[((10 \text{ positive items} \times 2 \text{ parents} \times 1 \text{ informant}) - (11 \text{ negative items} \times 2 \text{ parents} \times 1 \text{ informant}))\]. Items included on this scale were only asked at the initial interview.

2. Family Activities Scale. A family activities scale (famwith) was created at each time period. This scale included: the child’s endorsement of engaging in five categories of activities with each parent (2 parents \times 5 items); the parent’s endorsement of five activity categories the child participates in with each parent (2 parents \times 5 items); the
parent’s endorsement of participation in family activities (5 items); and the parent’s endorsement of whether they or the other parent (if applicable) does “a lot” with the child (2 parents x 1 item). This yielded a scale with a potential range of 0-27 at each time period.

**Initial Model**

As proposed, the first group of analyses examined the relationship of family covariates and time to relapse using Cox’s proportional hazard model with a forward stepwise-selection procedure. Relapse is defined the first subsequent time period in which a depressive episode (as calculated by the DIDCA algorithm) occurs. The five non-time dependent variables (i.e., marital status, parental death, family functioning at Time 1, family history-second degree relatives, family history-deceased or non-informant parent) and five time dependent variables (i.e., HRSD, PDI-Mood and Anxiety, PDI- Behavior and Other Disorders, stress, and family activities) were entered into the Cox model in a stepwise procedure. ID and NID groups were examined separately.

**ID Group.** Of the 244 children used in this analysis, 133 experienced depression at subsequent time intervals and 111 (45.5%) were censored (i.e., they survived without an episode of depression until the endpoint or dropped out of the study before experiencing depression). Only one variable, parental death, emerged as significantly altering time to further depression. Bereaved children experienced further depression at a slower rate than non-bereaved depressed children (regression coefficient = -.90, SE= 0.18, $\chi^2$=24.31 df=1, p<.001; hazard ratio=.41; see Figure 1).
To ensure accuracy of the procedure and to provide some tests of the model's assumptions that cannot be completed using the SAS statistical package, consultation was sought from the Statistics Department. These consultants used the statistical package S+, which provides numerical tests of the assumptions of proportionality, as opposed to the less exact visual tests used in SAS. This allows for construction of more precise models that meet model assumptions without increasing the number of independent variables added in a time-dependent fashion. S+ also provides an $R^2$ measure which is different from the typical $R^2$. The $R^2$ obtained from S+ provides a measure of the model's effect size (Nagelkerke, 1991). Results from the statistical consultants confirmed that the proportional assumption obtained via the SAS analysis was satisfied and the effect size of the model was adequate ($R^2=.33$). As S+ calculates values differently than SAS, slightly different values were found (regression coefficient = -1.30, SE= 0.25).

**NID group.** Of the 318 children included in the analysis, 60 experienced an episode of depression and 258 (81.1%) were censored. One variable, PDI-Behavior and Other Disorders, significantly influenced time to further depression. Children whose parents experienced more behavior and other disorders were more likely to experience a depressive episode than those children whose parents did not experience these difficulties (regression coefficient = .37, SE= 0.14, $\chi^2=6.9$ df=1, p<.01; hazard ratio=1.45; see Figure 2).

The statistical consultant suggested that the NID group’s model’s assumptions are better met when the independent variables are not all time-dependent, so analyses were rerun using the PDI indices and the HRSD scores as non-time dependent variables while
stress and family activities variables were examined as time-dependent. However, because using time dependent variables allow for participants with missing data to be used and non-time dependent variables do not, the sample size was greatly reduced. Of the 164 children used in this analysis, 38 experienced an episode of depression and 126 (77%) were censored. HRSD score at 60 months (regression coefficient = .09, SE= 0.02, $\chi^2$=20.40 df=1, p<.001; hazard ratio=1.11) and having experienced death of a parent (regression coefficient = .79, SE= 0.33, $\chi^2$=5.88 df=1, p<.05; hazard ratio=2.22) were the two variables which predicted time to onset of a depressive episode. This model resulted in an adequate $R^2$ ($R^2=.30$), especially given the decreased likelihood of depression in this sample.

Adjustment to the Model

As many participants were censored due to an incomplete 60 month interview, data were reanalyzed using depression in the two years post-entry as the dependent variable in order to increase power. This did not yield significantly different results. For the ID group, having experienced death of a parent continued to be the only predictor of further depression (i.e., non-bereaved children relapsed earlier; regression coefficient=-.90, SE= 0.18, $\chi^2$=21.3 df=1, p<.001; hazard ratio=.41). For the NID group, having experienced parental death was also the only predictor. In this case, children who had experienced parental death were more likely to have depression onset (regression coefficient =1.29, SE= 0.42, $\chi^2$=9.5 df=1, p<.01; hazard ratio=3.65) than non-bereaved children.
Further Analyses

Timing of relapse/onset of depression appears to be influenced by variables related to the relative composition of the bereaved, depressed and community cohorts. Due to power constraints, the depressed cohort cannot be examined alone. Therefore, the decision was made to examine differences in course of illness, demographic variables and family variables within the ID cohort to determine what differences exist between children depressed directly in response to a major stressor (parental death: BD) and children depressed for a variety of other reasons (D). Next, the same demographic and family variables were compared between those bereaved children who experienced depression in the first month post-death (BD) and those who did not (BND).

Comparison of bereaved-depressed (BD) and depressed (D) children. First, to determine if the course of previous depression differs for BD versus D children, two course of illness variables were compared. Each was significant. Depressed controls experienced more severe symptoms [CDRS-R at Time 1: BD=31.9±10.3 vs D=45.9±16.6, t(148)=7.39, p<.001] and more chronic or episodic depression [percent with prior episode in “lifetime”: BD= 8.5% vs D= 84%,χ²(1)=140.52, p<.001] than bereaved depressed children.

Eight demographic variables were compared between groups. Two significant differences emerged. Bereaved-depressed and depressed children did not differ in terms of their gender, age, age of their living parent, SES, sibling position and number of siblings in the family. Depressed children, however, were more likely than bereaved-depressed children to come from homes in which a divorce or separation had occurred.
[54.7% vs. 6.0%; \( \chi^2(1) = 70.53, p < .001 \)], and were more likely to have a mother as an informant parent [92% vs 73%, \( \chi^2(1) = 15.00, p < .001 \)]. Of note, divorce and having a mother as the informant were not independent in the ID cohort. In divorced ID families, 91% of informant parents were female, while only 78% of informants were mothers in nondivorced ID families [\( \chi^2(1) = 5.58, p < .05 \)].

Finally, 11 family functioning variables were examined. On most measures (9/11, 82%), bereaved-depressed children had better family functioning than depressed children. In one case, parental depressive symptom severity, bereaved children had worse family functioning. Only one family functioning variable, family pressure, did not differ between families of BD and D children (see Table 4).

As many family home environment variables were created to sum the child’s involvement with both parents pre-death (or study entry), and the majority of children in the depressed cohort came from families who had experienced a divorce or separation, family variables were recalculated to determine if between-group differences would persist if the child’s relationship with the informant parent only was assessed. Even with this recalculation, depressed children reported poorer family functioning on the same 9 out of 11 family variables.

**Comparison of bereaved-depressed (BD) to bereaved-nondepressed (BND).** Of the 8 demographic variables compared, only 1 differed between groups. Bereaved-depressed and bereaved non-depressed children did not differ in terms of their gender, age, age of their living parent, sex of living parent, parental marital status, sibling
position and number of siblings in the family. Bereaved depressed children did, however, come from lower socioeconomic status homes than BND children [BD=3.2±1.1 vs BND =2.8±1.1, t (326)=3.1, p<.005].

When the 11 family functioning variables were compared, 4 /11 (36%) variables--family functioning scale, perception of parental pressure, PDI-Mood and Anxiety, and family history of psychopathology in second degree relatives-- were more impaired in bereaved-depressed children’s families than in families of bereaved non-depressed children (see Table 5).

While interviews of depressed and community cohorts did not differentiate lifetime (i.e., the period of time before the death) and current (i.e., in the month prior to the interview) parental functioning, these specific time periods were assessed for bereaved participants. Thus, to more precisely examine differences in parental psychopathology within the bereaved cohort, current PDI Mood and Anxiety and PDI Behavior and Other indices were also compared. No differences emerged in these two comparisons indicating that spousal death was associated with increased symptomatology.

To further explore differences in lifetime rates of mood and anxiety disorders between parents of BD children and parents of BND children, rates of endorsement for specific mood disorders were compared. This resulted in four analyses [2 disorders (Bipolar disorder, depression) x 2 time periods (lifetime, post-death)] with three
significant findings. BD children were more likely than BND children to have a parent with lifetime symptoms/diagnosis of bipolar disorder and depression and post-death symptoms/diagnosis of bipolar disorder (see Table 6).

As anticipating parental death might contribute to the family differences found between BD and BN children, a chi-square test was conducted to determine if either of these groups was disproportionately comprised of anticipated-bereaved or unanticipated bereaved children. The percentage of anticipated deaths were equivalent in the two groups [BD= 42% vs BN= 44%; $\chi^2(1)=0.21$, NS].

**Predicting the number of depressed time periods.** As the survival analyses were not conclusive, and as large family differences emerged between depressed and bereaved depressed children, the research question was further revised to determine how family factors impact the course of depression. To investigate this, the number of time periods that the child and/or parent endorsed a depressive episode for the child after the initial interview was summed (range: 0-8 time periods). ID children experienced significantly more subsequent intervals with depression than NID children [ID=2.2±2.1 vs NID=0.4±1.0; $t(279)=11.45; p<.001$]. Within the ID group, depressed children experienced more time intervals with depression than bereaved-depressed children [3.1±2.0 vs 1.5±1.9; $t(208)=5.82, p<.001$]. No differences were detected between NID children from the bereaved or community cohorts [0.5±1.1 vs. 0.3±0.8; $t(296)=1.84; NS$].

Finally, multiple regression analyses were performed to determine if family factors and the presence/absence of depression at the initial interview could predict chronicity of depression. A stepwise selection process was used to determine the best
predictors from 12 family variables. These included the 10 variables from the survival analyses (i.e., marital status; parental death; family functioning at Time 1; family history-second degree relatives; family history-deceased or non-informant parent; family activities at Time 1; HRSD Time 1; PDI Mood and Anxiety index at Time 1; PDI Behavior and Other disorders at Time 1; stress at time 1) and two additional variables [initial depressed status (ID vs NID) and socioeconomic status] as previous analyses indicated these variables robustly differentiated groups. Six variables made significant contributions to the prediction of depression chronicity (See Table 7).
CHAPTER 4

CONCLUSION

The current study represents the first attempt to integrate the influence of genetics, family factors, and stressful events on the longitudinal course of childhood depression using both clinical and community samples. First, survival analyses were conducted to determine the relationship between genetic, family factors and stressful events and timing of depression relapse. Two subsets of children from the Grief Research Study were examined separately as it was hypothesized that new onset and relapse of depression would be associated with different survival hazards. Initially depressed children (ID) included depressed controls recruited from the affiliated outpatient and inpatient clinics and parent-bereaved children who experienced an episode of depression in the month post-parental death. The Not Initially Depressed (NID) cohort included those parent-bereaved children who did not experience depression in the month post-parental death and community controls.

Survival Analyses

ID group. Within the ID group, the only variable which emerged as a predictor of relapse was parental death. Having experienced the death of a parent was associated with longer survival without subsequent depression both at 25 months and 60 months.
post-death. The finding is striking as it suggests that major differences exist between children who become depressed following a major stressor (parental death) compared to depressed children from outpatient/inpatient settings. Depressed clinic patients, who have sought out clinical attention, are more impaired in terms of their course of illness and family functioning than children who meet criteria for depression in the month post-parental death but have not necessarily sought clinical attention. While parental death has been implicated as a risk for affective disorder (Kovacs, 1997; Weller et al., 1991), this finding suggests that, even for those parent-bereaved children who experience depression in the month post-death, the overall experience of depression in the face of this major stressor is less severe than in those seeking clinical attention.

**NID group.** Within the NID group, the only variable which emerged as a predictor of timing of relapse over 60 months was parental Behavior and Other disorders. Increased parental impairment was associated with shorter survival without onset of depression. However, scores on the PDI-Behavior and Other scale were very restricted, with most parents reporting no symptoms or diagnoses in these categories. Thus, having a parent with externalizing pathology was rare in this sample. When parents did experience Behavior and Other disorder symptomatology, this was predictive of subsequent depression in their child. The presence of Behavior and Other disorders might have intensified stress in the home environment and modified variables such as hostility and criticism which were not able to be assessed in this study after the baseline interview. By
way of contrast, mood symptomatology, which is generally expected following the death
of a spouse, was relatively frequent in bereaved spouses, and did not play a role in the
course of the child's depression.

When outcome during the first 25 months was examined, having experienced
parental death emerges as a significant predictor. While relatively few NID children
became depressed over this time period, having experienced parental death does make
these children more susceptible than community children.

Methodologic limitations. Several limitations occurred in the first set of analyses.
First, although survival analysis is designed to handle right censored observations (i.e.,
data from those participants who drop out of the study and do not return), current
statistical computer packages are not able to make adjustments for interval censored
observations (i.e., data from participants who skipped an interview in the middle of the
sequence). Unfortunately, interval censoring was common in this study. This interval
censoring was associated with decreased power that made further survival analyses (e.g.,
to examine the depressed cohort alone) impossible in the current sample.

Second, relapse, per se, was not precisely measured in this study. Available data
did not allow for the distinction between remission followed by relapse versus chronic
mood disturbance. Rather, presence/absence of depression at each assessed time interval
was ascertained. As a majority of the depressed cohort experienced depression at one or
more subsequent interviews (e.g., over half appear depressed at their six month
interview) comment cannot be made as to the prediction of chronic or recurrent
depression in this study. Due to these limitations, a third set of analyses (discussed later) was conducted to determine which variables are associated with overall time-in-episode for depressed, BD and BND children.

**Differences in Family Variables**

Demographic differences and family variables were compared between BD and D children to determine how family variables differ between children depressed directly in response to a major stressor (BD) and children depressed for a variety of other reasons (D), as well as between BD and BND families to determine how families of bereaved children who become depressed differ those who do not.

**Bereaved-depressed vs depressed.** BD children were compared to D children in terms of the chronicity and severity of their illness, demographic factors, and measures of family functioning. Although both BD and D children were depressed at study entry, children in the depressed cohort were experiencing more severe depression and were much more likely to have experienced depression in the past. While it is unknown whether the depression at Time 1 was a continuation of the past depression or separate episodes, depressed children probably had spent more time in-episode than BD children.

When demographic differences were examined between BD and D cohorts, two differences emerged. Depressed children were more likely to have a mother as informant parent and they were more likely to come from homes in with a divorce or separation had taken place. These two demographic differences were related, as children from homes in which a divorce had taken place were much more likely to have a maternal informant parent.
Bereaved-depressed children were substantially different from depressed children on most (9/11) family-based measures. In one instance, severity of parental depressive symptoms, BD children evidenced more impairment than D children. This is not surprising, as bereaved parents had just lost a spouse and were actively grieving. One variable, the child’s perception of being excessively pressured by the parent to behave well, did not differ between groups. This family pressure variable will be considered in more detail in a later section.

**Bereaved-depressed vs bereaved non-depressed.** Demographic and family variables were also compared between BD and BND children. These two groups differed on only one demographic variable. BD children came from families of lower socioeconomic status. It is unknown whether this lower socioeconomic status plays a causal role in the development of these children’s depression or if it is associated with other family variables that increased the children’s vulnerability to depression following the loss.

When family functioning, family history of psychopathology, and lifetime stressors were compared between BD and BND children, only four differences emerged. BD children reported more impaired overall family functioning. This might suggest that negative family interactions play a causal role in the development of depression in these bereaved children. Alternately, depressed children may report more negative family interactions than actually exist, as their perception is negatively colored by their
depressive symptom severity. Finally, the child’s depressive symptoms of irritability and/or social withdrawal might have altered family interactions, making them less positive.

Parents of BD children exhibited higher rates of internalizing disorder pre-death including bipolar disorder and depression. Post-death, the groups looked identical on depression, due to the expected high rate of depression in parents who had just experienced the death of a spouse. However, parents of BD children continued to experience higher rates of bipolar disorder than parents of BND children. As one could argue that anticipatory grieving might account for the pre-death differences, I next examined the relative percentage of BD and BND children who had experienced unanticipated versus anticipated parental death. No differences were found. This indicates that parents of BD children exhibited more lifetime psychopathology for reasons other than anticipatory grieving.

Families of BD children also had more psychiatric impairment in second degree relatives than did families of BND children. The increased rate of psychiatric illness in second degree relatives of BD children indicates they have more genetic risk for affective illness than BND children.

The final family variable on which BD were more impaired than BND children was children’s perception of receiving too much pressure from parents. This variable appears to be a surrogate for the child’s perception of family “Expressed Emotion” (i.e., negative attitudes such as overinvolvement and criticism that family members have toward another family member). While few authors have examined children’s perception
of family expressed emotion, it does appear that children’s perceptions of family climate is somewhat related to their symptoms (Cerel & Fristad, in submission). Interestingly, family pressure was the only family based variable that did not differ between BD and D children. Family pressure might directly result from being in the middle of a depressive episode and then might merely indicate the child’s unhappiness with his/her family. Alternatively, the child’s perception of family pressure might play a causal role in the development or continuation of their depression. As perception of family pressure is relatively simple to assess, it may prove useful in future studies assessing family climate and family-based interventions.

Predicting Chronicity of Depression

Finally, multiple regression analyses were performed to predict family variable contributions to the chronicity of depression, as measured by the number of time intervals from the one month to the 60 month interview that the child was in-episode.

Past episodes. The majority of variance in depression chronicity was predicted by depression at the initial interview. This is consistent with research in which previous episodes of depression have reliably played a major role predicting future depression (Frost, Reinherz, Pakiz-Camras, Giaconia, & Lefkowitz, 1999; Lewinsohn, Hoberman, & Rosenbaum, 1988). While the etiology of the past episodes of depression is unknown in this sample, one can assume that family variables played a role in the development of those prior episodes. As having a past episode of depression emerged as the strongest predictor of subsequent depression, this finding can greatly assist clinicians to identify those children most at-risk for future depression.
Divorce. Having experienced parental divorce prior to study entry also contributed to the chronicity of depression. This is consistent with prior research which cites the influence of living through a divorce and living with a single parent as associated with depressive symptomatology (Aseltine, 1996; Hetherington et al., 1998).

Family functioning. The composite family functioning variable at Time 1 made a small contribution to the chronicity of children’s depression. This supports the hypothesis that positive and negative family interactions play a role in childhood depression. As family pressure is included in family functioning, it lends further support to the predictive power of a child’s perception of family climate. The presence of this variable as a contributior, however small, to chronicity of childhood depression is promising for intervention efforts. Unlike stressful events and family history of psychopathology, clinicians can impact family climate. Psychoeducation has been shown to addresses these family issues and shows potential to reduce family expressed emotion (Fristad, Gavazzi, Centolella, & Soldano, 1996).

Family history of psychopathology. Psychopathology in second degree relatives contributed to the chronicity prediction. The extent of parental symptomatology was not precisely measured in this study due to issues already discussed. However, the ability of second degree relative symptomatology to play a small predictive role indicates that genetic factors contribute to determining which children have a prolonged course of illness.

Stress. The quantity of stressful life events a child had experienced prior to study entry made a small contribution to the prediction of depression over the 60 months post
study entry. The measure of stressful life events used in this study was very broad, encompassing events ranging from frequent family arguments to death of a family member (other than the already deceased parent in the bereaved cohort). Consistent with other research demonstrating that individuals who are most likely to become depressed following stressful events are those with a history of major depression (Hammen, 1992), the presence of life stressors as a predictor of chronicity of depression in this study indicates that for children, stress has a small role in the course of depressive illness.

Family socioeconomic status also emerged as playing a small predictive role in determining chronicity of depression. Socioeconomic status might also be conceptualized as a measure of daily hassles. Due to the small amount of variance in the prediction of depression chronicity contributed by family SES, future studies might wish to assess daily hassles or other more sensitive measures of family stress.

Limitations

Several limitations of the current study need to be addressed. First, the study was conducted in a moderate-sized Midwestern city in which cultural and the study sample was predominately Caucasian. For this reason, results might not generalize to a more ethnically and/or demographically diverse population. Second, as the data from which this examination were drawn were not specifically designed to measure the impact of family variables, some measurement problems exist. For example, family functioning prior to depression-onset could not be examined. Questions about family functioning relied primarily on the child’s perspective. Within the ID group, the child was actively depressed when family functioning was assessed which might have negatively affected
their answers. More complete measures of family functioning were only available at the initial interview. Although Expressed Emotion is one of the only family factors previously associated with depression relapse in children (Asarnow et al., 1993), no direct measure of EE was available in the current study. However, the variable examining children’s perception of family pressure appears to play a similar role as more traditional EE assessment (Friedmann, 1993; Magana et al., 1986; Vaughn & Leff, 1976). Further research should address briefer measurements of both child and parent perception of EE.

Measures of family history of psychopathology were broad. No continuous measure of lifetime parental depressive symptoms was available. For extended family history measures, scores did not differ whether the family member had only one symptom or was only one symptom short of meeting diagnostic criteria (both were counted as positive for symptoms). Bereaved parents were first interviewed after the death of a spouse, at a time when depressive symptoms were almost universally present. Finally, the measurement of stress was broad and daily hassles, which have been implicated as a contributor to childhood depression, could not be examined.

Conclusions

The present study adds to our knowledge about families of depressed children. No other study combines genetics, stressors, and family environmental data to examine continuation of childhood depression or onset of depression, especially in sample of children who have experienced major stressor such as parental death and those who have experienced chronic depression. The present study demonstrates that genetics, stressful events and family environment each play a role in determining the course of childhood depression.
depression. In general, families of children who experience acute depression after a very stressful event such as death of a parent appear very different from those of children who experience depression for a variety of reasons beyond death of a loved one. When parent-bereaved children who are depressed in the first month post-parental death are compared to parent-bereaved children who do not experience depression post-parental death, BD children have more family history of psychopathology, worse overall family functioning and the children perceive more parental pressure. These family differences may help clinicians identify those children most at risk for depression following a stressful event such as parental death. They also suggest the need for clinical intervention designed to reduce pressure and/or hostility in the family environment.
REFERENCES


<table>
<thead>
<tr>
<th></th>
<th>Initially Depressed</th>
<th>Not Initially Depressed</th>
<th>Test statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group Composition</td>
<td>137 Bereaved</td>
<td>191 Bereaved</td>
<td>$\chi^2(2)=230.4$ **</td>
</tr>
<tr>
<td></td>
<td>106 Depressed</td>
<td>0 Depressed</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2 Community</td>
<td>127 Community</td>
<td></td>
</tr>
<tr>
<td>Child’s gender</td>
<td>54% male</td>
<td>49% male</td>
<td>NS</td>
</tr>
<tr>
<td>Informant parent’s gender</td>
<td>81% female</td>
<td>77% female</td>
<td>NS</td>
</tr>
<tr>
<td>Child’s age</td>
<td>11.8±3.1 (5-18)</td>
<td>11.0±3.1 (5-18)</td>
<td>$t(522)=2.74$ **</td>
</tr>
<tr>
<td>Marital status</td>
<td>73% married</td>
<td>96% married</td>
<td>$\chi^2(1)=58.22$ **</td>
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<tr>
<td>SES (Hollingshead)</td>
<td>3.1±1.1 (1-5)</td>
<td>2.5±1.1 (1-5)</td>
<td>$t(555)=6.66$ **</td>
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<td>Siblings in family</td>
<td>2.8±1.2 (0-8)</td>
<td>2.7±1.1 (0-7)</td>
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<td>Sibling number</td>
<td>1.8±1.1 (0-8)</td>
<td>1.7±1.0 (0-7)</td>
<td>NS</td>
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<tr>
<td>Age of informant parent</td>
<td>39.5±6.3 (23-55)</td>
<td>40.6±5.4 (23-58)</td>
<td>$t(479)=2.19$ *</td>
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</table>

* p<.05  ** p<.001

Table 1: Demographic differences between children who were depressed or not depressed at initial interview
<table>
<thead>
<tr>
<th>Scale</th>
<th>Initially Depressed</th>
<th>Not Initially Depressed</th>
<th>Test statistic</th>
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<tbody>
<tr>
<td>FH2SUM</td>
<td>4.1±3.0 (0-15)</td>
<td>2.1±1.7 (0-10.5)</td>
<td>t(351)=9.10 **</td>
</tr>
<tr>
<td>FH2PER</td>
<td>1.4±1.2 (0-5.4)</td>
<td>0.5±0.5 (0-2.62)</td>
<td>t(298)=10.62 **</td>
</tr>
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</table>

** p<.001

Table 2: Differences in the family history second degree sum (FH2SUM) and the family history second degree percentage (FH2PER) variables by initially depressed status
<table>
<thead>
<tr>
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<th>Initially Depressed</th>
<th>Not Initially Depressed</th>
<th>Test statistic</th>
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<tbody>
<tr>
<td><strong>PDI-Mood and Anxiety</strong>(^a)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>1 month</td>
<td>1.4±1.6 (0-7)</td>
<td>0.6±1.0 (0-6)</td>
<td>t(375)=7.20 ***</td>
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<tr>
<td>13 months</td>
<td>1.0±1.3 (0-6)</td>
<td>0.5±0.9 (0-4)</td>
<td>t(287)=4.77 ***</td>
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<tr>
<td>25 months</td>
<td>1.0±1.4 (0-6)</td>
<td>0.5±0.9 (0-5)</td>
<td>t(251)=4.14 ***</td>
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<tr>
<td>60 months</td>
<td>0.9±1.1 (0-4)</td>
<td>0.5±0.8 (0-3)</td>
<td>t(170)=2.95 **</td>
</tr>
<tr>
<td><strong>PDI-Behavior and Other</strong>(^b)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 month</td>
<td>0.9±1.6 (0-17)</td>
<td>0.4±0.7 (0-4)</td>
<td>t(303)=4.75 ***</td>
</tr>
<tr>
<td>13 months</td>
<td>0.3±0.7 (0-5)</td>
<td>0.1±0.3 (0-2)</td>
<td>t(225)=2.99 *</td>
</tr>
<tr>
<td>25 months</td>
<td>0.3±0.6 (0-4)</td>
<td>0.1±0.4 (0-2)</td>
<td>t(237)=2.56 **</td>
</tr>
<tr>
<td>60 months</td>
<td>0.1±0.3 (0-1)</td>
<td>0.1±0.3 (0-2)</td>
<td>t(178)=2.12 *</td>
</tr>
</tbody>
</table>

\(^a\) PDI Mood and Anxiety Disorders sums symptoms (1) and diagnoses (2) of mania, depression, OCD, panic disorder; \(^b\) PDI Behavior and Other Disorders sums symptoms (1) and diagnoses (2) of alcohol abuse, drug abuse, antisocial personality, schizophrenia, organic brain syndrome, hysteria, anorexia, phobia, and mental retardation

\(*p<.05, **p<.01, ***p<.001\)

Table 3: Differences in psychiatric diagnostic interview (PDI) mood and anxiety and behavior and other disorders between initially depressed and not initially depressed children at 1, 6, 13, 25, and 60 months post-study entry.
<table>
<thead>
<tr>
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<th>Bereaved-Depressed</th>
<th>Depressed</th>
<th>Test statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=137)</td>
<td>(n=106)</td>
<td></td>
</tr>
<tr>
<td>Lifetime stress</td>
<td>3.9±3.1</td>
<td>7.9±4.7</td>
<td>t(170)=7.48 **</td>
</tr>
<tr>
<td>Family Functioning$^1$</td>
<td>13.2±5.1</td>
<td>8.6±5.2</td>
<td>t(225)=6.68 **</td>
</tr>
<tr>
<td>Family Activities$^1$</td>
<td>18.8±4.0</td>
<td>15.1±5.0</td>
<td>t(194)=6.21 **</td>
</tr>
<tr>
<td>Hamilton total</td>
<td>11.1±8.1</td>
<td>8.4±8.9</td>
<td>t(231)=2.46 *</td>
</tr>
<tr>
<td>Second degree relatives</td>
<td>2.8±2.8</td>
<td>5.8±2.4</td>
<td>t(235)=8.91 **</td>
</tr>
<tr>
<td>Family History-deceased/ non-informant</td>
<td>1.2±1.7</td>
<td>2.1±2.3</td>
<td>t(180)=3.38 **</td>
</tr>
<tr>
<td>PDI- Mood and Anxiety</td>
<td>0.8±1.1</td>
<td>2.2±1.7</td>
<td>t(169)=7.22 **</td>
</tr>
<tr>
<td>PDI- Behavior and Other Disorders</td>
<td>0.5±0.9</td>
<td>1.4±2.2</td>
<td>t(130)=4.19 **</td>
</tr>
<tr>
<td>Pressure</td>
<td>2.0±2.5</td>
<td>2.1±2.2</td>
<td>t(228)=0.27 NS</td>
</tr>
<tr>
<td>Negative Interactions</td>
<td>1.0±1.3</td>
<td>1.7±1.6</td>
<td>t(173)=3.96 **</td>
</tr>
<tr>
<td>Positive Interactions$^1$</td>
<td>14.5±3.3</td>
<td>11.5±3.8</td>
<td>t(229)=6.38 **</td>
</tr>
</tbody>
</table>

$^1$ higher scores indicate more adaptive functioning

* p<.05, ** p<.001

Table 4: Differences in family functioning variables between bereaved-depressed and depressed children
<table>
<thead>
<tr>
<th></th>
<th>Bereaved-Depressed</th>
<th>Bereaved-Nondepressed</th>
<th>Test statistic</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=137)</td>
<td>(n=191)</td>
<td></td>
</tr>
<tr>
<td>Lifetime stress</td>
<td>3.9±3.1</td>
<td>3.3±3.2</td>
<td>NS</td>
</tr>
<tr>
<td>Family Functioning(^1)</td>
<td>13.2±5.1</td>
<td>14.3±4.6</td>
<td>t(315)=2.01 *</td>
</tr>
<tr>
<td>Family Activities(^1)</td>
<td>18.8±4.0</td>
<td>18.9±4.4</td>
<td>NS</td>
</tr>
<tr>
<td>Hamilton total</td>
<td>11.1±8.1</td>
<td>10.2±11.1</td>
<td>NS</td>
</tr>
<tr>
<td>Second degree relatives</td>
<td>2.8±2.8</td>
<td>2.2±1.6</td>
<td>t(198)=2.00 *</td>
</tr>
<tr>
<td>Family History-deceased</td>
<td>1.2±1.7</td>
<td>1.1±1.8</td>
<td>NS</td>
</tr>
<tr>
<td>PDI- Mood and Anxiety</td>
<td>0.8±1.1</td>
<td>0.6±1.1</td>
<td>t(257)=2.17 *</td>
</tr>
<tr>
<td>Other Disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pressure</td>
<td>2.0±2.5</td>
<td>1.3±2.0</td>
<td>t(249)=2.81 *</td>
</tr>
<tr>
<td>Negative Interactions</td>
<td>1.0±1.3</td>
<td>0.8±1.3</td>
<td>NS</td>
</tr>
<tr>
<td>Positive Interactions(^1)</td>
<td>14.5±3.3</td>
<td>14.7±3.0</td>
<td>NS</td>
</tr>
</tbody>
</table>

\(^1\)higher scores indicate more adaptive functioning

* p<.05

Table 5: Differences in family functioning variables between bereaved-depressed and bereaved non-depressed children
<table>
<thead>
<tr>
<th></th>
<th>Bereaved-Depressed</th>
<th>Bereaved-Nondepressed</th>
<th>Chi-Square$^1$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Symptom</td>
<td>Diagnosis</td>
<td>Symptom</td>
</tr>
<tr>
<td>Bipolar Disorder</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lifetime</td>
<td>3.8%</td>
<td>3.0%</td>
<td>0.5%</td>
</tr>
<tr>
<td>Post-Death</td>
<td>3.0%</td>
<td>3.0%</td>
<td>0.3%</td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lifetime</td>
<td>13.5%</td>
<td>24.8%</td>
<td>12.4%</td>
</tr>
<tr>
<td>Post-Death</td>
<td>18.9%</td>
<td>25.8%</td>
<td>25.3%</td>
</tr>
</tbody>
</table>

$^1$Chi-square compares individuals with symptoms and/or diagnosis vs. none

*p<.01; ** p<.001

Table 6: Differences in rates of lifetime and post-death bipolar disorder and depression between parents of bereaved-depressed and bereaved non-depressed children
<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SEB</th>
<th>β</th>
<th>Cumulative R²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presence of Depression at Time 1</td>
<td>1.17</td>
<td>0.16</td>
<td>0.32</td>
<td>.24</td>
</tr>
<tr>
<td>Parental Marital Status</td>
<td>0.89</td>
<td>0.24</td>
<td>0.17</td>
<td>.30</td>
</tr>
<tr>
<td>Family Functioning Scale</td>
<td>-0.04</td>
<td>0.01</td>
<td>-0.13</td>
<td>.32</td>
</tr>
<tr>
<td>Family History Second Degree</td>
<td>0.08</td>
<td>0.03</td>
<td>0.12</td>
<td>.34</td>
</tr>
<tr>
<td>Stress</td>
<td>0.05</td>
<td>0.02</td>
<td>0.11</td>
<td>.34</td>
</tr>
<tr>
<td>Hollingshead rating</td>
<td>-0.13</td>
<td>0.06</td>
<td>-0.08</td>
<td>.35</td>
</tr>
</tbody>
</table>

1 0=married 1=divorced, separated or not married prior to parental death/study entry;

Note: R²= 0.35, adjusted R²=0.34, N=462, p<.001

Table 7: Family variables contributing to the prediction of number of assessment periods in which the child is depressed
Figure 1: Survival without a subsequent depressive episode in initially depressed (ID) children based on presence/absence of parental death
Figure 2: Survival without onset of depressive episode in those not initially depressed (NID) children based on parental behavior and other disorder (PDIBEH) scores.